Clinical pediatrics / by John Lovett Morse.

H. Fallows

CLINICAL PEDIATRICS

By

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ILLUSTRATED

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To the men who have been my assistants, Doctors Henry I. Bowditch, Fritz B. Talbot, Richard M. Smith, Edwin T. Wyman and Lewis W. Hill, in recognition of their unfailing loyalty, enthusiasm and helpfulness. I am indebted to them for much inspiration at times when the game hardly seemed worth the candle and the study and practice of Pediatrics flat, stale and unprofitable.

PREFACE

THIS book was written primarily for my own amusement, not with the idea of enlightening the world or of adding anything to the sum of human knowledge. I hope, nevertheless, that it may be interesting and instructive to both students and general practitioners. In it I have endeavored to summarize what I have found it necessary to know of the physiology and anatomy of early life, of gross pathology and of bacteriology to make a proper physical examination, to appreciate the etiology of and the pathologic changes in the diseases of infancy and childhood and to serve as a basis for an intelligent diagnosis. I have laid especial stress on methods of physical examination and on what can be learned from it, because I feel that in daily practice the physician must depend very largely on his own powers of observation for a diagnosis and because I know that it is not only impossible, but also unnecessary, for him to carry out complicated laboratory procedures except in occasional instances. I have not attempted to give detailed descriptions of microscopic pathologic changes, which I should have been compelled to copy from some other book and which I would not recognize if I saw them. I have also not attempted to describe diseases with which I have had no practical experience. In consequence, there are many omissions. I realize, moreover, that the space given to the various diseases is not commensurate with their relative importance. The explanation is that I have written most about those diseases concerning which I felt I knew most or was most interested in, and least about those with which I was less familiar or in which I was not especially interested. In discussing differential diagnosis I have tried to emphasize the facts that, in general, the diagnosis rests on one or two special symptoms or signs and that symptoms or signs common to several conditions are of no importance. I have described only those methods of treatment which I have found useful and which seem to me to be rational. Finally, I have endeavored to show the futility and unreasonableness of much of the treatment in common use.

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BOSTON, MASS.

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SECTION XIX

CLINICAL PEDIATRICS

SECTION I

CONGENITAL MALFORMATIONS

MENINGOCELE, ENCEPHALOCELE AND HYDRENCEPHALOCELE

In these conditions there is a protrusion of some portion of the cranial contents through an opening in the skull. The hernia almost always occurs through one of the normal openings of the skull, at the junction of some of the cranial bones or in a suture. The most common locations are in the median line in the occipital region or a little to one side of the median line at the root of the nose. If the tumor is made up of the meninges and cerebrospinal fluid, it is called a meningocele; if it contains brain tissue, it is an encephalocele; if it contains a portion of a distended lateral ventricle inside of the brain tissue, it is an hydrencephalocele. These tumors are presumably due to an intrauterine increase in the intracerebral pressure. The cause of this increase in pressure may or may not be still active after birth. The tumor may be covered only with the meninges, which may or may not be ulcerated, or with skin. If it is covered with skin, there is usually no hair on it. It is always present at birth. It is likely to increase in size, if it is a meningocele, not so likely to, if it is an encephalocele or an hydrencephalocele. It varies in size from that of a walnut to that of the patient's head.

A meningocele has a smooth surface, is symmetrical, is pedunculated, fluctuates and is translucent. It seldom pulsates. It can always be partly and sometimes completely reduced. Pressure on it causes bulging of the anterior fontanelle and symptoms of cerebral irritation, such as crying, twitching, rigidity and convulsions. An encephalocele is usually small. It is ordinarily smooth and symmetrical. It is not pedunculated, does not fluctuate and is not translucent. It usually pulsates. It may or may not be reducible, pressure causes bulging of the anterior fontanelle. Pressure on it always produces symptoms of cerebral irritation. An hydrencephalocele is large and irregular in shape. It is pedunculated and semi-translucent. Fluctuation is more or less distinct. It usually does not pulsate. It is not reducible. Pressure on it does not usually cause any bulging of the anterior fontanelle or symptoms of cerebral irritation.

Diagnosis.—Confusion sometimes arises between these tumors and *caput succedaneum* and *cephalhematoma*. *Caput succedaneum* is an edema of the scalp and is present at birth. It is situated at the location of the presenting part, is largest at the base, is covered with skin and hair, pits on pressure and is not fluctuant nor translucent. Pressure on it does not cause bulging of the fontanelle nor symptoms of cerebral irritation. It gradually diminishes in size and is gone in a few days. A *cephalhematoma* is not present at birth, but develops soon after. It is

due to a hemorrhage between the periosteum and one of the cranial bones. The periosteum being attached to the edge of the bone, the swelling is always limited to one bone, usually one of the parietals. It is covered with skin and hair, is largest at the base, is fluctuant, but not translucent. Pressure on it does not cause bulging of the anterior fontanelle or symptoms of cerebral irritation. After a few days a boney ridge is usually palpable at the edge of the tumor. The tumor begins to diminish in size in a few days or a week and is gone in a few weeks. If the characteristics of these tumors are borne in mind there should be no difficulty in differentiating between them. A *fatty tumor* or a *nevus* may sometimes be present at birth and occupy one of the positions where meningoceles and allied tumors are usually located. If the characteristics of these tumors are kept in mind and it is remembered that the meningoceles and

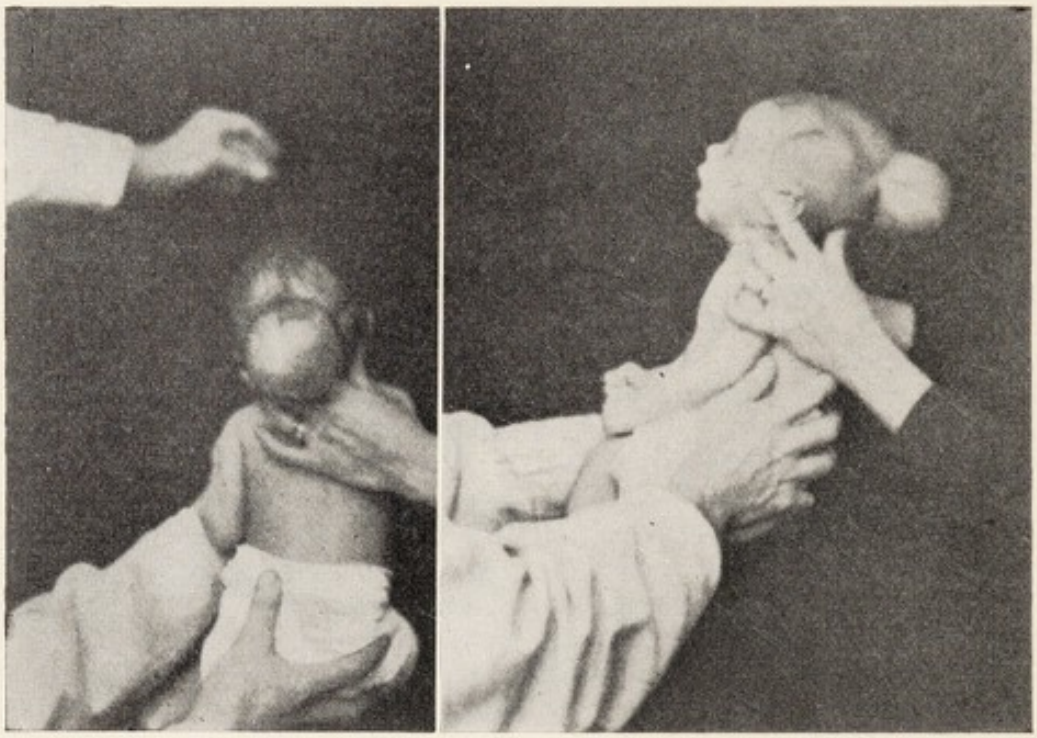

FIG. 1.—Meningocele.

allied tumors originate inside the skull and protrude out from it, while fatty tumors and nevi originate outside of it, there will be no difficulty in distinguishing between them.

Prognosis.—If these tumors are covered only by membrane, ulceration is almost certain to take place in a short time with, sooner or later, rupture of the sac. Death may then occur at once from loss of cerebrospinal fluid or, if not, is almost certain to in a short time from meningeal infection. In very rare instances, if the tumor is a meningocele, healing, with recovery, may occur. When these tumors are covered with skin, there is, of course, no danger of rupture. Sometimes a meningocele will gradually diminish in size and recovery take place. If the tumors are encephaloceles or hydrencephaloceles, there is no possibility of any diminution in their size or of recovery. In fact, both are liable to grow larger. Pressure or a blow on the tumor may at any time increase the symptoms or even cause death.

Treatment.—There is no medical treatment for these tumors. External applications and pressure can do nothing but harm. Whatever is

done must be surgical. It is usually a comparatively simple matter to tie off a meningocele, although there is always the possibility that death may occur during the operation from loss of cerebrospinal fluid or soon after it from infection. Moreover, if the operation is successful, hydrocephalus is likely to develop, if the original cause of the hernia is still active.

In certain instances, when an encephalocele is small, it is possible to remove it. If the portion of brain removed is a part of one of the so-called "silent areas" of the brain, no ill effects may result from the removal. When an encephalocele is large, the chances of a successful operation are small, but the outlook for the future is so poor without an operation that it is wise to operate and take what little chance there is. An hydrencephalocele is, of course, inoperable. Finally, meningoceles should be operated upon; encephaloceles, unless very large, should be operated upon; hydrencephaloceles must be let alone.

MALFORMATIONS OF THE CRANIUM

There are a number of other congenital malformations of the cranium, none of which is of much clinical importance. **Anencephalia**, a condition in which, on account of a failure of development, more or less of the brain and cranium are lacking, is merely a pathologic curiosity, as it is incompatible with life. Most babies with this malformation are born dead. The others live but a few hours or days. The causes of **congenital hydrocephalus** are the same as when it develops later. The diagnosis, prognosis and treatment are also the same, and consequently deserve no special consideration. **Asymmetry of the cranium and face** not due to injury, edema or hemorrhage, is not very uncommon. The tendency is always toward improvement, and in most instances the malformation is not noticeable in adult life. There is no treatment for it.

PROTRUSION OF THE EARS

Protrusion of the ears, a trivial malformation , but one which troubles mothers much, is not uncommon. Although the protrusion often seems to increase, especially in middle childhood, the comparative infrequency of protruding ears in adults seems to show that the natural tendency is to spontaneous disappearance of the deformity. There is no doubt, however, that if the baby is allowed to lie with the ear turned forward, the deformity increases. Care should be taken, therefore, to see that the ear is turned backward when the baby lies on it. It is also probable that keeping the ears flat on the head with a cap or some simple apparatus does tend to improve the condition. If the deformity is extreme, it can be readily overcome by removing a semilunar piece of skin from the back of the ear and another from the side of the head and stitching the ear back.

CERVICAL FISTULÆ AND CYSTS

The clinical evidences of a cervical fistula are a dimple in the neck, with or without a watery, somewhat glairy, discharge. The opening is usually along the anterior border of the sterno-cleido-mastoid muscle, but may be in the median line. If lateral, it is more often on the right than on the left and, whether lateral or median, is usually below the level of the hyoid bone. The discharge resembles saliva, contains columnar and squamous epithelial cells, often irritates the skin and sometimes forms crusts over the opening. The lateral fistulae are due to failure of closure of

the branchial clefts; the median may be due either to imperfect closure of the sinus praecervicalis or to persistence of the thyro-glossal duct of His. These fistulae are lined with epithelium of various sorts. They may end blindly or may connect with the pharynx.

If the external opening of a cervical fistula becomes closed or if there is a fistulous tract without an external opening, the secretions of the tract may be retained and form a cyst. The tumor may or may not be present at birth. It increases in size after birth, may be either lateral or median, and is covered with skin. It is almost always unilocular and is fluctuant or semi-fluctuant.

Treatment.—The treatment is entirely operative. Unless the fistulous tract is entirely removed, recurrence is almost certain. The operation is almost always more serious and difficult than is anticipated, because of the length and situation of the fistulous tract. Unless the discharge is considerable or the tumor deforming, it is probably wise, therefore, not to interfere.

CYSTIC HYGROMA OF THE NECK

A cystic hygroma is a lymphangioma of the neck. It is usually situated on the side of the neck. It is present at birth and may be large

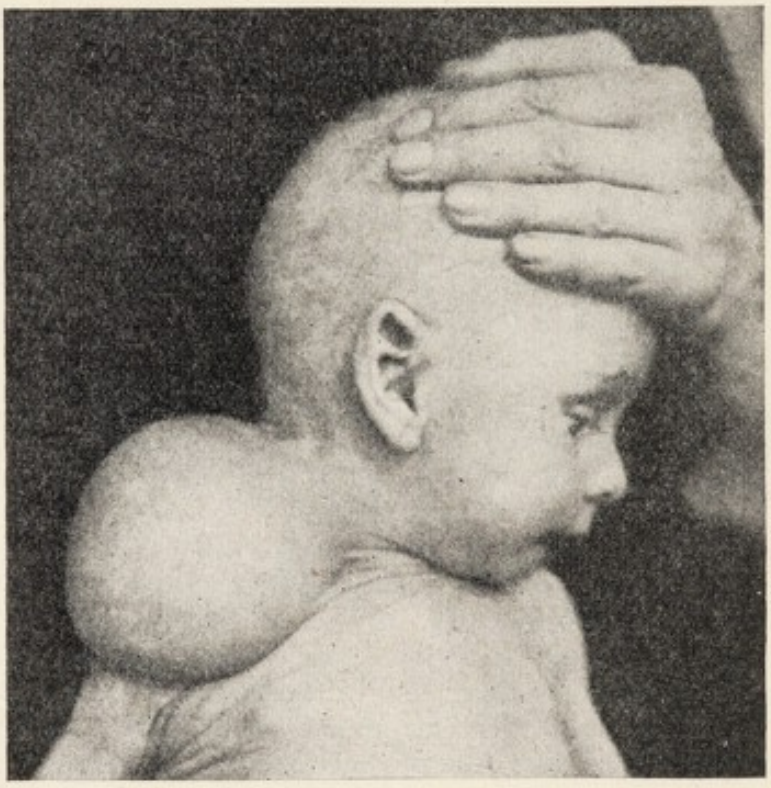

FIG. 2.—Cystic hygroma of the neck.

enough to interfere with delivery. Its tendency is to increase in size, both externally and internally. Increase in size internally may cause interference with respiration and deglutition. The tumor is multilocular and elastic, sometimes semifluctuant, but never fluctuant. It is covered with normal skin.

Treatment.—These tumors should be removed as soon as the condition of the infant will warrant it, because of the tendency to increase in size internally, which makes their removal more difficult. The tumor

must be entirely removed, because, if it is not, it will recur. The operation is always a very difficult and dangerous one.

HARELIP

Etiology.—Harelip is due to the failure of the maxillary processes to unite with the nasofrontal process. Hence the fissure is always to one side of the median line. The processes may fail to unite on either one or both sides. Harelip may be, therefore, either single or double. The fissure may be very slight at the edge of the lip or deep, extending into the nose. It is often associated with *cleft palate,* but either condition may occur alone.

Symptomatology.—Except for the deformity, harelip causes no symptoms, unless it interferes with nursing. It may then cause considerable disturbance of the nutrition from an insufficient supply of food.

Treatment.—Harelip should be operated upon when the baby is three or four weeks old, if its general condition warrants it. If it does not, as soon after this time as it does. If the deformity interferes with nursing, the baby must be given breast milk or modified milk with a Breck feeder, spoon, or dropper, or, if necessary, be fed through a tube.

CLEFT PALATE

Etiology.—Cleft palate is due to the failure of the ridges which grow out from the inner surfaces of the two portions of the upper jaw to form the roof of the mouth to unite in the median line. The failure to unite may occur at any point or be complete. As union begins anteriorly and extends backward, it is uncommon to find defects in the hard palate without involvement of the soft. The slightest degree of cleft palate is the *bifid uvula.*

Symptomatology.—Cleft palate is a severe condition in that it almost always interferes with or prevents nursing from either the breast or bottle and hence causes serious disturbance of the nutrition, which may even be fatal. There is very likely to be an infection of the nasal passages as the result of irritation from the food which gets into them. This infection is liable to extend into the ears and nasal adnexa as well as to the mouth. Thrush is also a not uncommon accompaniment. Disturbances of the digestion and infections of the lungs often develop secondarily and not infrequently prove fatal.

Treatment.—If the baby is unable to suck, as it usually is, it must be fed in some other way. There is a nipple with a soft rubber flange on each side which sometimes works very well, the wings closing the defect in the palate. Some babies can take their food from a Breck feeder, others have to be fed with a spoon or a dropper. They often take their food better, if the head is turned to one side or tipped back. If there is much difficulty in feeding, it is advisable to use a stomach tube, as this method of feeding not only insures a sufficient supply of food but saves the baby's strength and diminishes the dangers of infection. If possible, the mother's milk should be expressed and given to the baby, as it is even more important for these than for normal babies to get breast milk. It is important to keep the mouth and nose clean. This can be done with water, boracic acid solution or some mild alkaline wash. It must be remembered, however, that it is very easy to do more harm than good by too vigorous attempts at cleanliness. If thrush or other infections of the mouth and nose develop, they are to be treated by the usual methods. The best time for operation is at about eighteen months.

RANULA

A ranula is a soft, semifluctuant, spherical or elongated, bluish swelling in the floor of the mouth beside the frenum of the tongue. It may be single or double. It may be congenital or develop at any time during childhood or adult life. The tumor is not tender and causes inconvenience only because of its size. It is a cyst due to the retention of the secretion of a mucous gland as the result of the blocking of its duct. The contents are a thick, glairy, colorless fluid.

The best treatment is the complete removal of the sac. It is useless to aspirate or incise the tumor, as the fluid invariably re-collects.

CONGENITAL HYPERTROPHY OF THE TONGUE

Congenital hypertrophy of the tongue, or *macroglossia,* is usually bilateral, but may be unilateral. The hypertrophy is usually due to dilatation of the lymph vessels, but sometimes to that of the blood vessels. It is occasionally due to an overgrowth of connective tissue. It is never a sign of *cretinism* or *Mongolian idiocy,* as the enlargement of the tongue in cretinism is not marked at birth and that in Mongolian idiocy does not appear until childhood.

When the enlargement is lymphangiomatous or angiomatous, the treatment must be operative and carried out as soon as the infant is old enough to bear the operation, because the tendency of these growths is to increase rapidly in size. When it is due to an overgrowth of connective tissue, it is wise to delay the operation for a time, unless the tongue is very large, because there is a chance that the tongue may become smaller or that it may not increase in size and that the child may "grow to the tongue."

TONGUE-TIE

This name is applied to the condition in which the frenum of the tongue is short and prevents the normal protrusion of the tongue. It is evident that there may be all degrees of this malformation. If the shortening of the frenum is extreme, it may interfere with sucking and talking. Such interference is in my experience very unusual and certainly does not occur, if the tongue can be protruded beyond the gums. If there is difficulty in sucking, the cause is much more often to be found in the nose or nasopharynx. If there is delay in talking, the child is usually either backward or feeble-minded. If the speech is indistinct, the cause is generally to be found in the nervous system, not in the tongue.

Treatment.—The treatment is, of course, to cut the frenum. This is seldom necessary, however, and there is no doubt that many more frenums have been cut unnecessarily than for good cause. Fortunately it seems to do but little harm to cut the frenum, even when it is already long enough. In performing this operation the lower part of the tongue is pushed up and guarded with the perforated flange of a director. The frenum is then cut as deeply as desired with scissors. The cut should be made near the gum in order to avoid cutting the ranine arteries, which run close to the tongue.

CONGENITAL MALFORMATIONS OF THE CHEST

The chest may be malformed in many ways, but most of these malformations are very uncommon and of little clinical importance. A few of them, however, are worthy of mention.

Funnel Chest.—In funnel chest there is a depression of the sternum with an increase in the lateral diameter of the chest. This malformation is congenital, but is usually not noticed until the baby is one or two years old. It is often hereditary and familial. It causes no symptoms and can not be remedied. A similiar condition may develop in connection with rickets, when there is interference with the entrance of air into the lungs as the result of adenoids or some other obstruction to respiration. In such instances there are other signs of rickets and usually the characteristic facies of adenoids.

Cervical Ribs.—A cervical rib seldom causes any symptoms and is usually overlooked. It may, however, sometimes be seen and felt as a prominence in the neck and be mistaken for an enlarged gland or some form of new growth. It may also, by pressing on an artery, cause pallor and coldness of the arm or, by pressure on nerve trunks, cause pain, numbness or tingling in the arm. Such symptoms should suggest the possibility that there may be a cervical rib. The diagnosis can easily be made with the aid of the Roentgen ray. The treatment is, of course, surgical.

Sprengel's Deformity, or **Congenital Elevation** of the **Scapula,** is not very uncommon, but is usually overlooked or misinterpreted. In this condition one scapula is higher than the other and rotated outward. The scapula is usually malformed and there are often associated malformations of the ribs. The treatment is surgical.

SPINA BIFIDA

Spina bifida is a condition in which there is a lack of closure of one or more of the vertebral arches. This defect may occur anywhere in

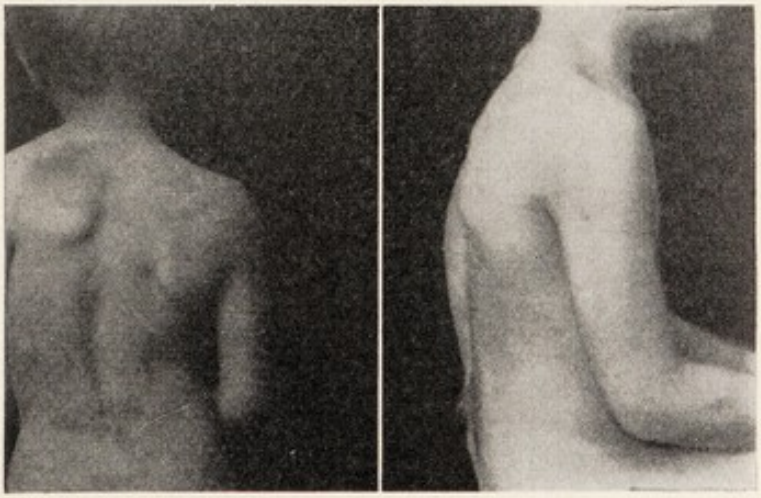

FIG. 3.—Sprengel's deformity. Spina bifida occulta.

the vertebral column, but is most common in the lower dorsal and lumbar regions. It occurs next most frequently in the neck. I have seen one instance in which the defect was in the upper dorsal region and the hernia through it protruded into the right chest, giving the physical signs of fluid in the upper chest.

Spina Bifida Occulta.—When there is no hernia through the defect in the arches and it is covered with skin, the condition is usually spoken of as *spina bifida occulta.* In such instances, however, there is quite likely to be a mole or a growth of hair in the area over the defect. In fact, a large mole or a localized growth of hair over the spine should always

suggest the possibility of this defect. There are usually no other symptoms of this abnormality, but I have seen both *enuresis* and *incontinence of feces*, presumably as the result of some involvement of the nerve fibres. This abnormality should, therefore, always be thought of as a possible cause in intractable cases of incontinence of either urine or feces, and sought for by physical examination and the Roentgen ray. Unfortunately, there is nothing to be done, if it is discovered.

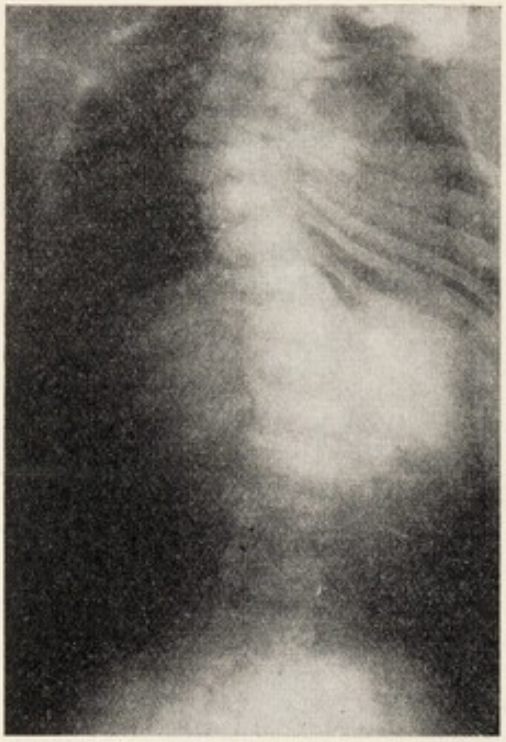

FIG. 4.—Spina bifida occulta. Sprengel's deformity.

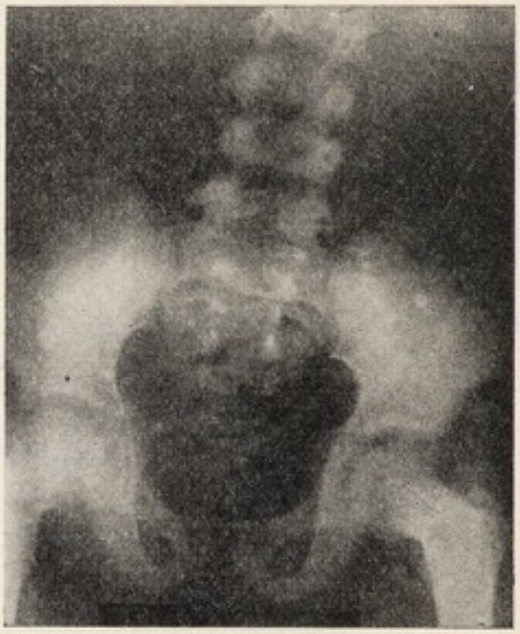

FIG. 5.—Spina bifida occulta.

SPINAL MENINGOCELE, MENINGO-MYELOCELE AND SYRINGO-MYELOCELE

There may be a hernia of the contents of the vertebral canal through the spina bifida. It is to these herniæ that the term "spina bifida" is usually and improperly applied. If the sac contains only cerebrospinal fluid, it is a *spinal meningocele;* if it contains parts or the whole of the spinal cord, it is a *meningo-myelocele;* if, as sometimes happens, the coverings of the sac are made up of the meninges and cord spread out about the distended central canal of the cord, it is a *syringo-myelocele.* The meningo-myelocele is much the most common form. The hernia may be covered with normal skin, by the membranes of the cord, or by a combination of the two. If the membranes make up the covering, they are almost certain to become ulcerated in a short time. Except in spinal meningoceles, there are likely to be deformities and spasm or paralysis of the lower extremities, as the result of the involvement of the cord or nerve fibres in the sac. Paralysis of the perineum and of the sphincters of the anus is also common. Pressure on these tumors often causes bulging of the anterior fontanelle and symptoms of cerebral and spinal irritation.

Diagnosis.—It is possible that a large *nevus* or a *fatty tumor,* which happened to be located over the back bone, might be mistaken for one of these tumors. If the difference in origin and contents of the tumors are borne in mind, there should be, however, no difficulty in distinguishing

between them. A roentgenogram of the spine will settle the question at once and conclusively.

Prognosis.—There is no danger of rupture of the sac when these tumors are covered with skin. A blow or a fall on the sac may, however, cause great discomfort and possibly prove fatal. There is, moreover, no probability of improvement in any paralysis or spasm which may be

FIG. 6.—Cervical meningocele.

present. In some instances, when the skin extends well on to the sac, it may gradually grow in and replace the membranous coverings. In general, however, when the sac is made up wholly or in part of membrane, ulceration occurs in spite of the greatest care and, sooner or later, rupture. When rupture occurs, the infant almost always dies, either at once or after some hours or days from the loss of cerebrospinal fluid, or from a

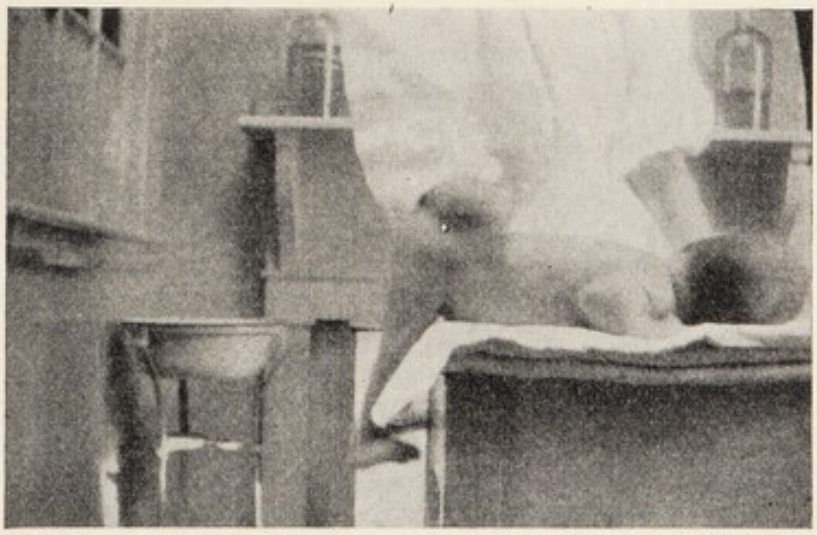

FIG. 7.—Lumbar meningo-myelocele. Dislocation of hips.

secondary ascending meningitis. In very rare instances, however, the sac becomes puckered down after it ruptures, heals and recovery occurs. In such cases, also, there is not likely to be any improvement in the spasm or paralysis and secondary hydrocephalus may later develop. There is a reasonable chance that, in spite of the dangers from loss of cerebrospinal fluid and infection, the sac may be successfully removed in spinal meningocele and healing take place. There is great danger, however,

that the increased cerebrospinal pressure, which originally prevented the closing of the vertebral column may still be present and cause a secondary hydrocephalus. There is some chance of a successful operation on a meningo-myelocele, but there is very little chance of relieving any existing paralysis or spasm and great probability of increasing them by further injuring the cord or nerves. If the perineum and the sphincters of the anus are paralyzed, an operation almost never does any good. Nothing can be done, of course, for a syringo-myelocele.

FIG. 8.—Spontaneous cure of "spina bifida." Secondary hydrocephalus.

Treatment.—A spina bifida occulta should be let alone. When the hernia through a spina bifida is covered with skin it should not be operated upon, but should be covered with a shield of thick leather or metal to protect it from injury. This may seem unnecessary, but I knew one boy with this condition who was tortured by his playmates hitting and kicking him there. If the sac is covered with membrane and there is no ulceration, it is advisable to delay operation until the baby is a

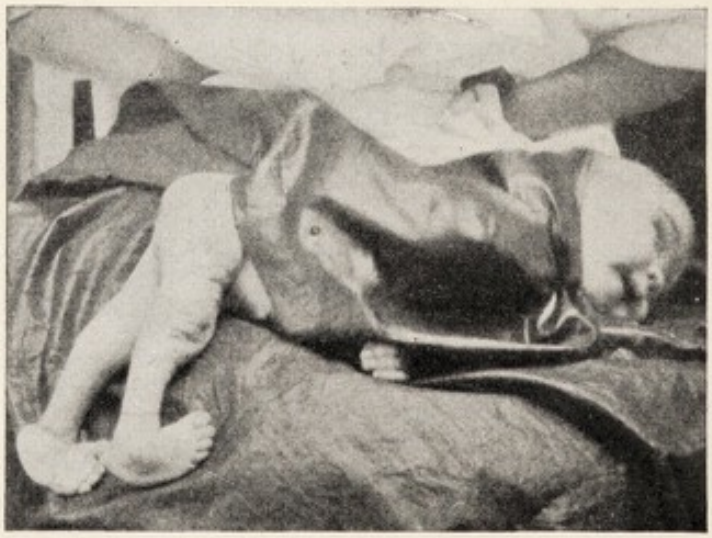

FIG. 9.—Spastic paralysis after successful operation for "spina bifida."

few weeks or months old, being constantly on the lookout for the slightest sign of ulceration. The sac should be covered with a piece of linen or cotton, never gauze, to which sweet oil, vaseline or boracic acid ointment has been applied, and protected from injury and, as far as possible, contamination with urine and feces. The advisability of operation is very doubtful, however, if there is paralysis of the perineum or marked paralysis or spasm of legs, as these will not be relieved by the operation and may

perhaps be increased. The probability of the development of hydrocephalus, if the operation is successful, must always be borne in mind and explained to the parents. If there is ulceration, an operation should be done at once, because of the dangers of rupture of the sac and meningitis, if it is delayed.

DIAPHRAGMATIC HERNIA

Diaphragmatic hernia is a very uncommon condition. The defect in the diaphragm is more often on the left and may be either large or small. The symptoms are due primarily to the mechanical interference with

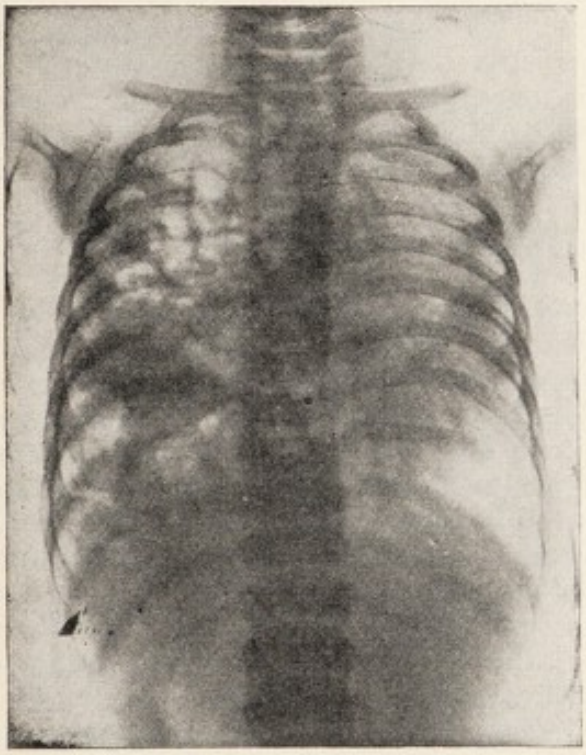

FIG. 10.—Left diaphragmatic hernia.

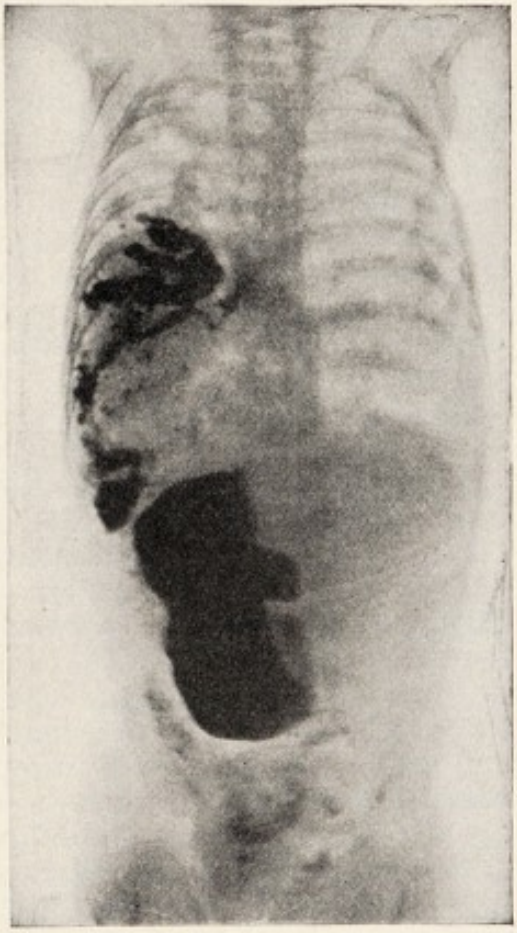

FIG. 11.—Left diaphragmatic hernia, after opaque meal.

respiration from the presence of the intestines in the thoracic cavity. If the hernia is large, there may also be interference with the action of the heart. If the hernia is small, there may be interference with the function of the intestines or even strangulation. It is evident how much the symptoms may vary in different cases and at different times in the same case, according to the size of the hernia. They are usually present at or soon after birth, but may not appear for some time or may not be noticed for years. They may be either constant or intermittent. Whenever a new-born or young infant is cyanotic or shows other signs of interference with respiration, or has attacks of dyspnea or apnea, diaphragmatic hernia should be thought of as one of the possible causes. While a possible cause of constipation and intestinal obstruction, it hardly need be very seriously considered.

The **physical signs** are, of necessity, most confusing. In addition to cyanosis, rapid pulse and respiration, the heart is likely to be more or

less displaced. The respiratory and voice sounds are likely to be diminished on the affected side and may be bronchial in places, if the lung is compressed. The percussion note may be normal, if the hernia is small, while, if the hernia is large, it may be dull, flat or tympanitic, according to whether the bowel is filled with feces or gas. Gurgling may be heard in the chest. If so, a strong suspicion of diaphragmatic hernia is justified. In many instances, however, a positive diagnosis is not justifiable on the physical signs. A diaphragmatic hernia should always be suspected, however, when the physical signs are incompatible with any of the usual pulmonary diseases. The diagnosis can be easily and certainly made with the aid of the Roentgen ray.

Prognosis.—Persons with a diaphragmatic hernia have been known to reach adult life, having had few or no symptoms. In general, however, death occurs in the first few days or weeks of life or, at any rate, before the close of infancy.

Treatment.—The chance of closing the defect in the diaphragm by operation is very small, but there is a chance. It is better, moreover, than the chance for life, if no attempt at repair is made. It seems wiser to operate in these cases, therefore, and to give the baby what little chance there is, unless the defect is shown by the roentgenogram to be a very large one.

CONGENITAL LARYNGEAL STRIDOR

In congenital laryngeal stridor a crowing sound is made at the beginning of inspiration. It is noticed at or within a few days after birth. The intensity of the sound varies from one that is scarcely audible to one which may be heard on another floor through several closed doors. Except in the mildest cases, it is heard with every inspiration, but is louder when respiration is forced than when it is quiet. It is audible during sleep. In severe cases and in mild cases, when the respiration is forced, there is more or less marked retraction of the supraclavicular and suprasternal spaces and of the epigastrium, but almost never of the intercostal spaces. In most instances, however, there is no retraction. There are no other symptoms; the infant is not uncomfortable, there is no cyanosis or cough, and no interference with nursing. The cry is clear. The crowing sound remains of about the same intensity for several months, then gradually diminishes and finally ceases, in most instances when the baby is about a year old. It may, however, persist for some months or a year longer.

Etiology.—Congenital laryngeal stridor is due to a congenital narrowing or infolding of the epiglottis with consequent laxness of the aryepiglottidean folds or to a congenital elongation of these folds. The opening of the larynx is consequently narrowed during inspiration, but there is no interference with expiration. The occurrence of the crowing sound in inspiration only and the clearness of the cry are thus explained. Recovery takes place with the growth of the parts and the consequent establishment of normal conditions.

Diagnosis.—The symptomatology of this malformation is so characteristic that it should always be recognized at once and never be confused with other conditions. It is, however, often so confused, either from ignorance of its existence or from lack of care in observation. It is most often confused with *laryngismus stridulus*, one of the manifestations of the spasmophilic diathesis. Laryngismus stridulus is never present at birth. The crowing sound is always intermittent, occurring in attacks

and accompanied by cyanosis, discomfort and also by temporary cessation of respiration. Other signs of the spasmophilic diathesis, such as Trousseau's sign, the facial phenomenon, exaggerated reflexes, and changed electrical reactions are also present. In congenital laryngeal stridor the crowing sound is present at birth, is continuous, is not accompanied by any evidences of discomfort or obstruction, and there are no signs of exaggerated nervous irritability. Obstruction in the nose or nasopharynx, as by *adenoids*, *nasopharyngitis*, or *retropharyngeal abscess*, is sometimes accompanied by noisy inspiration, especially if there is a retropharyngeal abscess low down in the pharynx. The sound is, however, never the same as in congenital laryngeal stridor, it is not present at birth, the infant is manifestly sick and uncomfortable, and physical examination shows the evidences of some disease causing the obstruction. *Laryngitis*, whether catarrhal or diphtheritic, may cause a crowing sound with inspiration. This sound is, however, always of recent development and is associated with a loss of or a change in the character of the voice and cough. The baby is also sick. *Hypertrophy of the thymus*, while it may be congenital and cause interference with respiration, is almost never great enough to produce a crowing sound in respiration. If it did, the sound would be heard in both inspiration and expiration, and there would be marked evidences of interference with the breathing. The cry would be clear, but the larynx would not move in respiration. There would be all the physical signs of enlargement of the thymus. *Enlargement of the tracheobronchial lymph nodes*, while it may cause interference with respiration, can not cause a crowing sound in inspiration. The only conditions which can simulate almost exactly congenital laryngeal stridor are a *papilloma* or a *cyst of the larynx*, situated just above the vocal cords. Both of these may cause interference with inspiration without interference with expiration and a continuous crowing sound. Both may also be present at birth. I have seen both of these conditions mistaken for congenital laryngeal stridor. The diagnosis can only be made by examination of the larynx.

Treatment.—No treatment is necessary or possible. Nothing can be done to hasten the growth of the larynx and epiglottis. It is important, however, to avoid as far as possible catarrhal processes in the respiratory tract, because, if there is a catarrhal laryngitis, the symptoms are likely to be exaggerated.

CONGENITAL MALFORMATIONS OF THE ESOPHAGUS

Congenital Atresia of the Esophagus

A number of congenital malformations of the esophagus, resulting in atresia, have been described. These malformations are more common than is usually supposed. In the one which occurs by far the most frequently the upper portion of the esophagus is somewhat dilated and ends sharply and roundly at or just above the level of the bifurcation of the trachea. The lower portion is normal at the stomach end, tapers gradually at the upper end and almost always opens into the trachea at or just above the bifurcation. Sometimes it opens into a bronchus. The two portions of the esophagus are usually connected by a fibrous cord.

Symptomatology.—The symptomatology of this malformation is very characteristic. Nevertheless, almost everyone, the first time he meets it, fails to recognize it before the autopsy, but he never misses it again. The baby appears perfectly normal at birth, but the obstetrician notices that it is unusually full of mucus and that it is hard to get the upper respiratory

passages clear. It either has frequent attacks of coughing, strangling and cyanosis or the saliva dribbles almost continuously from its mouth. When put to the breast, it nurses vigorously for a short time, then coughs, strangles, and vomits up mucus alone or a mixture of mucus and milk. It then takes hold again vigorously and repeats the performance until it is too tired to nurse any more. Sometimes, when there is very little milk, it will nurse several times without vomiting, and then vomit up milk and mucus with the same strangling and coughing. The attacks are the same when water or any form of food is given. These symptoms continue without improvement. The baby is always hungry, there is no distension of the abdomen, the bowels are constipated, the stools are made up entirely of meconium, loss of weight and strength is progressive and death takes place in about a week, although some babies are said to have lived as long as two weeks. There is often a little rise in temperature in the first few days from lack of fluid, the so-called *inanition fever;* after this the temperature is subnormal. The skin becomes dry and wrinkled, the fontanelle depressed, the cry feeble, the motions slow and infrequent and the mind apathetic, that is, the baby shows all the evidences of progressive starvation.

Diagnosis.—It is evident that the only condition which can explain the attacks of coughing, strangling and cyanosis, with the vomiting of mucus and ingested liquids, is some obstruction to the passage of the food and saliva into the stomach. It is also evident that, as the symptoms occur in attacks, the obstruction must be part way down the esophagus, thus allowing the retention of a certain amount of liquid, which does not cause trouble until it fills up the cavity and begins to run over. If the obstruction was high up, symptoms would appear as soon as anything was taken. The other symptoms are simply confirmatory of the conclusion that nothing enters the stomach or those of secondary starvation.

The diagnosis should always be confirmed by an attempt to pass a soft rubber catheter into the stomach. The distance from the gums to the cardia in the new-born is about 17 cm. The obstruction is usually met at about 12 cm. It is important to use a catheter large enough to fairly well fill the esophagus. A #12 American is about the right size for a new-born baby.

Prognosis and Treatment.—When the malformation is the usual one the prognosis is absolutely hopeless. In rare instances, however, the obstruction may be due to a membrane placed transversely across the esophagus. In one of my patients, in whom the symptoms were characteristic, an obstruction, presumably of this sort, was felt to give way suddenly under the pressure of the catheter, which then passed easily into the stomach. The symptoms were immediately relieved and the baby did uninterruptedly well. It is useless to try to correct the malformation by operation, because it is impossible to restore the continuity of the esophagus. Life may be prolonged a short time by a gastrotomy, but it is impossible to raise a baby by feeding it in this way. The only thing to do is to keep it as comfortable as possible and to allow it to die peacefully.

Congenital Stenosis of the Esophagus

Congenital stenosis or stricture of the esophagus, while very rare, is more common than congenital atresia. The stricture may occur at any part of the esophagus and may be narrow or several centimeters in length. There is usually not much dilatation above the stricture, although there may be considerable, or even sacculation.

Symptomatology.—The chief symptom is, of course, vomiting. This may begin as soon as the baby begins to take food or not until after the attempt is made to give solid food. When it begins in early infancy, there is nothing characteristic about it. It usually, however, occurs as soon as the milk is taken or not at all. It is never explosive. It may occur with more or less severity at almost every feeding or it may occur only occasionally, or in attacks lasting a few days at a time at intervals of weeks. It makes very little difference whether the baby is on the breast or the bottle or what the character of the milk mixture. It is evident that secondary spasm must play an important part in the causation of the vomiting, especially when it is intermittent.

In other instances vomiting does not begin until the attempt is made to take more solid food. Some children are unable to swallow even soft

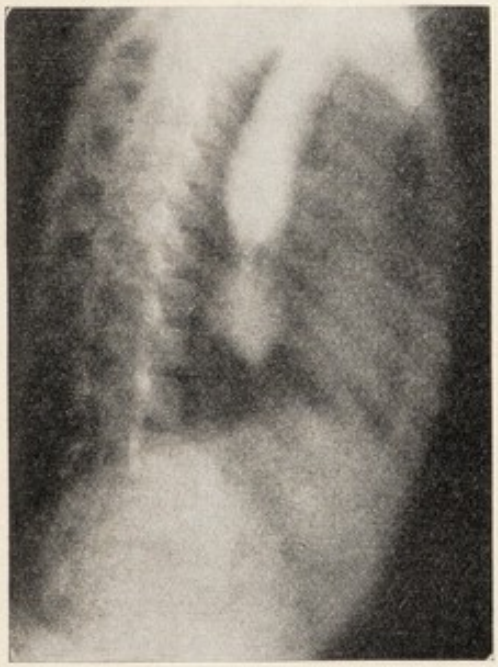

FIG. 12.—Congenital stenosis of esophagus. Lateral view after opaque meal.

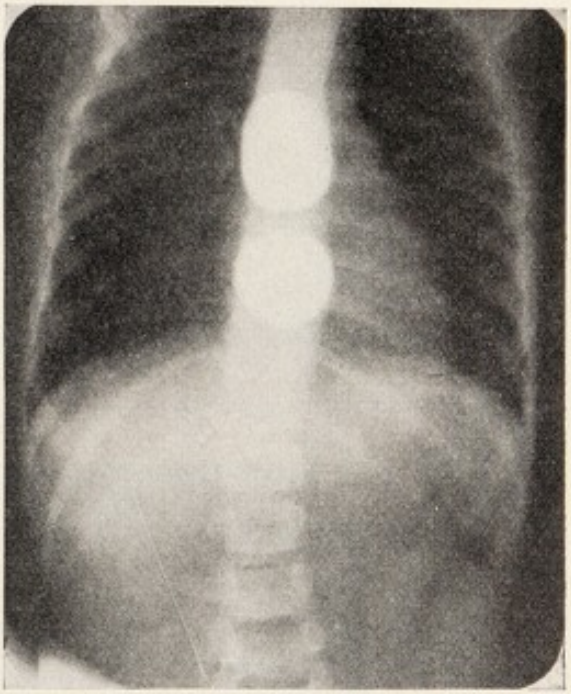

FIG. 13.—Congenital stenosis of esophagus. Front view after opaque meal.

solids; others can take them, but are unable to swallow solid food. As there is usually but little dilatation above the stricture, vomiting occurs at once or after only a little food has been taken. The retention of food to be vomited later is very uncommon. The vomiting is not explosive. Not infrequently articles of food which can be taken at one time are vomited at others, presumably because of secondary spasm. Attacks often occur in which nothing, not even liquids, can be swallowed for several days. In such instances the attacks are due to irritation with secondary spasm or the impaction of food or of some foreign substance, like a coin, which has been swallowed. The nutrition is more or less impaired according to the severity of the symptoms and the care which has been exercised in the selection of the food given. Deformity of the jaws is likely to develop from lack of use.

Diagnosis.—Congenital narrowing of the esophagus is seldom recognized as early as it should be. In infancy, the symptoms are almost always attributed to indigestion, while even in childhood they are attributed to poor appetite, capriciousness about the food or indigestion.

There is a certain amount of excuse for overlooking the true condition in infants that are taking only liquid foods, but in childhood there is no excuse for not suspecting it and finding out whether it is present or not. This can be done easily by using the fluoroscope or taking roentgenograms after an opaque meal, with bougies or, best of all, with the esophagoscope. The only condition with which congenital stenosis of the esophagus may be justifiably confused is *spasm of the esophagus*, which may give almost identical symptoms. The diagnosis between these conditions can always be made, however, with bougies or the esophagoscope.

Prognosis.—The prognosis in this condition is, in general, very good, if it is treated, as it should be, by an expert esophagoscopist.

Treatment.—Dilatation is usually sufficient to relieve and cure this malformation, but in certain instances operation is necessary. The feeding of these patients, both before and after operation, is very important. They must not only be given food which they can swallow, but the diet must be so planned as to give a sufficient number of calories and to contain the various food elements in the proper proportions and a sufficient amount of all the accessory food factors. The nutrition can be built up and kept up, even in the worst cases, if sufficient care is exercised in the regulation of the diet.

CONGENITAL MALFORMATIONS OF THE STOMACH

Hypertrophic Stenosis of the Pylorus

The only congenital malformation of the stomach of any importance is hypertrophic stenosis of the pylorus. The symptoms of this malformation are not present at birth, but develop in a few weeks.

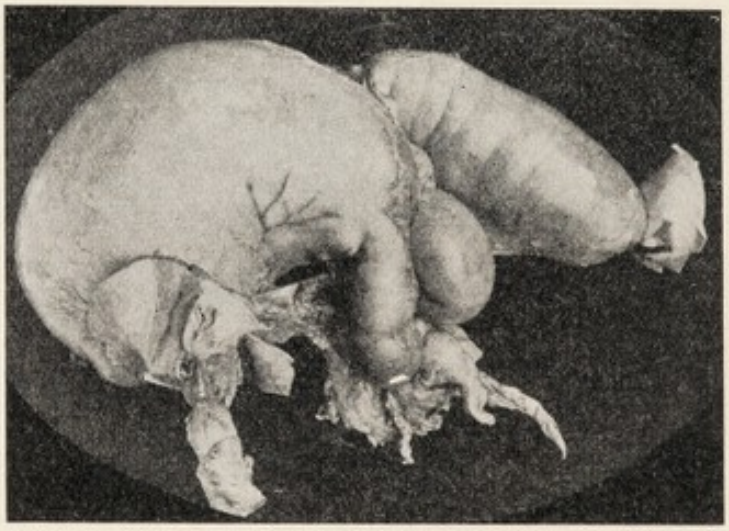

Fig. 14.—Hypertrophic stenosis of the pylorus. Anterior view after gastro-enterostomy, showing tumor.

Pathology.—The pathologic lesion consists of a simple hypertrophy of the circular, muscular fibres of the pylorus, which is often two or three times thicker than normal. The normal thickness of the circular, muscular coat in infants under three months of age is not over 2.5 millimeters, with an average of 1.6 millimeters. In hypertrophic pyloric stenosis the thickness varies between 3 and 7 millimeters, with an average of 4.4 millimeters (Wollstein). There is little, if any, involvement of the longitudinal muscular fibres. The submucosa is often edematous and thus appears wider than normal. This and the mucosa are often

thrown into folds, which increase the obstruction. The mucosa and serosa are normal and there is no formation of new connective tissue. There are never any inflammatory lesions. Macroscopically, the tumor

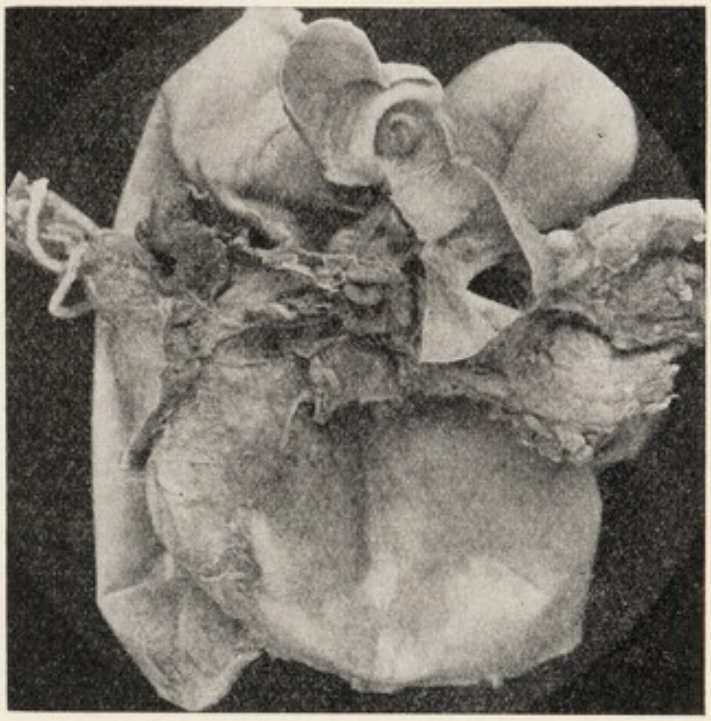

FIG. 15.—Hypertrophic stenosis of the pylorus. Shows tumor from the duodenum.

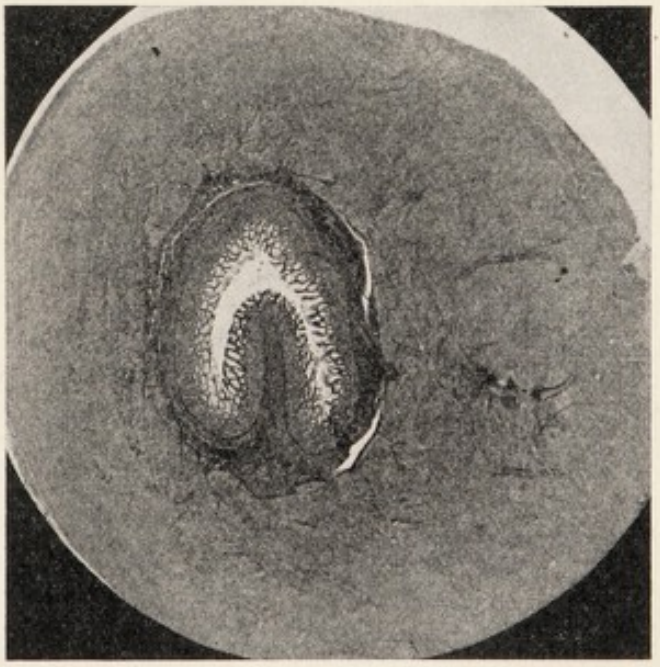

FIG. 16.—Hypertrophic stenosis of the pylorus. Cross section. Note folding of mucosa and dilatation of glands.

is about the size and shape of a dressed olive, pale, smooth and hard, like cartilage.

Hypertrophy of the pylorus has been found in the fetus, in premature and in new born infants. Furthermore, it would seem to be impossible that the degree of hypertrophy which is found at operation a few weeks

after birth in these cases could have developed in so short a time. The lesion is, therefore, unquestionably congenital. Hypertrophy of the muscles of the stomach develops after a time as the result of their attempt to overcome the obstruction. When they weaken, as they must in time, dilatation takes place. A secondary catarrhal condition of the gastric mucosa also sometimes develops as the result of retention of the gastric contents. The intestine becomes narrow and shrunken from lack of use. The pathological changes of starvation finally develop, as less and less food is retained.

Etiology.—In spite of the fact that many theories have been advanced as to the causation of this malformation, it is safe to say that nothing is really known about it. The explanations which are given are simply collections of words, which merely serve to cover up, more or less successfully, our real ignorance.

This malformation is more common in boys than in girls and is said to occur more frequently in first-born children than in those born later. It is at least as common in breast-fed babies as in those fed artificially.

Symptomatology.—Babies with hypertrophic pyloric stenosis appear perfectly normal at birth and show no symptoms of their trouble. Usually at some time during the second week they begin to vomit. The vomiting may, however, begin earlier than this. Its appearance is rarely delayed beyond a month, although in a case reported by Scott, in which the diagnosis was proved at operation, it did not begin until the baby was more than twenty-one months old. At first, there is usually nothing characteristic about the vomiting, but after a few days or weeks it becomes explosive. In rare instances it is explosive from the first. The vomiting is sometimes so violent that the vomitus may be thrown two or three feet. When the symptoms are well established, vomiting usually occurs but once after a meal, as the stomach is emptied at that time. Sometimes several meals may be retained and vomited together. The vomiting usually occurs immediately or within half an hour after food is taken. The baby is likely to show evidences of discomfort or pain just before vomiting. The character of the food, unless it be a thick cereal gruel, the amount given at a feeding or the intervals between feedings make but little difference. The vomitus usually shows no evidence of any catarrhal inflammation of the stomach, but consists of the food, either just as it was taken or partially digested.

The baby soon becomes constipated, because of the small amount of food which enters the intestine. The stools at first show evidences of milk remains, but later, being made up of bile, the intestinal secretions and bacteria, consequently have the characteristics of meconium or of the starvation stool. The urine becomes scanty from lack of fluid.

There being no indigestion, the tongue is clean and the breath sweet. As the baby is starved, it is naturally very hungry and ready to eat again as soon after vomiting as it has overcome the fatigue due to the effort. Being hungry, it cries and fusses as long as it has the ability. The manifestations of starvation develop rapidly, according to the amount and progress of the obstruction in the individual case. The baby loses weight and strength. The skin is dry, the fontanelle depressed, the mouth dry, the cry feeble. The temperature usually becomes subnormal, or may become elevated, because of the lack of fluid, the so-called "inanition fever."

Physical Signs.—The physical signs to be looked for, in addition to the general manifestations of malnutrition and starvation, are dilatation of

the stomach, visible gastric peristalsis and tumor of the pylorus. In the earlier stages, the stomach simply appears more prominent than usual after food is taken. In the later stages, if dilatation has occurred, it can be seen and palpated below the navel. Unless it is below the navel, it is not safe to consider it enlarged. Percussion of the stomach is of little use at this age. If much obstruction has developed, waves of peristalsis can be seen running across the stomach from left to right. These appear soon after food is taken and, in my experience, can always be seen, if they are looked for carefully enough. They can sometimes be started up by stroking the epigastrium or by putting a piece of ice or a cold cloth on it. There may be only one wave or two or three following each other across the stomach. They often make the baby uncomfortable and sometimes make it cry out. They are usually about the size of half an egg.

The tumor can almost always be felt, if it is carefully sought for. It is usually palpable about an inch to the right of a point midway between the tip of the ensiform and the navel, but may be found anywhere in the

FIG. 17.—Hypertrophic stenosis of the pylorus. Waves of peristalsis.

upper right abdomen. It feels like a dressed olive, is movable and is not tender. It does not vary in size. If there is visible peristalsis, the tumor is usually found where the waves stop. Sometimes it is more easily felt when the stomach is full, sometimes when it is empty. If it is not easily felt, the stomach should be filled, either by giving the baby a meal or with a tube. If it is not found, the abdomen should be examined during the relaxation which follows vomiting. It is more easily palpated then than at any other time. Although the tumor can almost always be found, its absence does not absolutely exclude pyloric stenosis, because, in rare instances, it may be situated so high up under the liver that it cannot be felt. Moreover, a certain amount of skill and experience is necessary to recognize it. It is possible to mistake an enlarged gland for it, but not likely, as enlarged abdominal glands are most unusual at this age and the combination of an enlarged gland and the characteristic symptoms of hypertrophic pyloric stenosis would be a most unusual coincidence.

Diagnosis.—The symptomatology and physical signs of hypertrophic pyloric stenosis are so characteristic that it is almost impossible to mistake it for anything else. There are, however, a number of conditions which may be mistaken for it. The most important of these is pyloric spasm. Many pediatrists do not believe in spasm of the pylorus as an entity, but think that it occurs only as a complication of hypertrophic pyloric

stenosis. Some of them think that it is the cause of hypertrophic pyloric stenosis and others speak of them as if they were the same thing. Personally, I believe that pyloric spasm may occur as an entity. I also believe that it increases the symptoms in true hypertrophic pyloric stenosis, often accounts for the variation in their intensity and is responsible for their sudden onset in some cases. Pure spasm is, however, relatively uncommon in comparison with hypertrophic stenosis. The vomiting appears either earlier or later, usually later, in spasm than in stenosis. It may be explosive, but is usually not as constant. Constipation is, as a rule, less severe and the stools contain more food residue than in stenosis. Malnutrition is usually not as marked. Visible peristalsis may be, and often is, just as marked. Ordinarily, there is no palpable tumor. If there is a tumor, it is usually smaller and not as hard. Finally, it may be intermittent and vary in size.

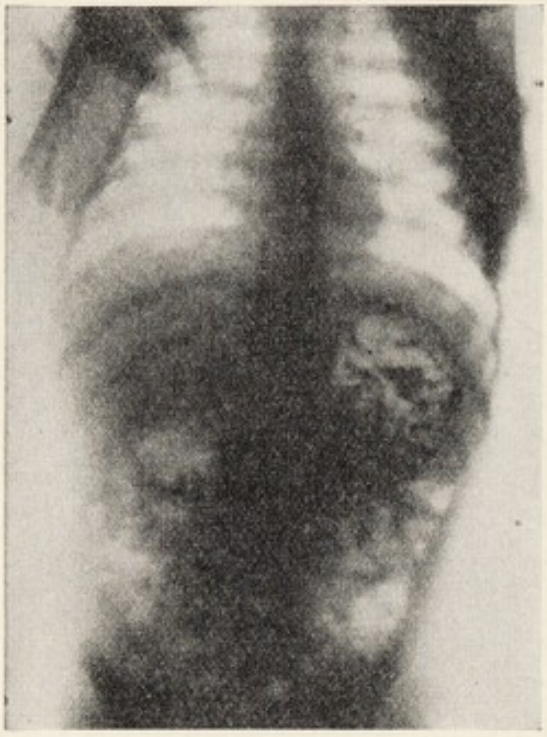

FIG. 18.—Hypertrophic stenosis of the pylorus. Taken immediately after an opaque meal. Nothing has left the stomach.

Fluoroscopic examination or Roentgenographs taken after a bismuth or barium meal are sometimes of great assistance in the diagnosis between stenosis and spasm. Normally, of course, the food begins to leave the stomach at once and the stomach is empty in about three hours. In spasm, nothing may leave the stomach for some time or only a little at long intervals, but eventually, if the meal is not vomited, the pylorus relaxes and the food passes out. In stenosis, nothing leaves the stomach for hours or, at best, only a little trickles through, the pylorus never relaxes and eventually, the meal is vomited.

Chronic indigestion is sometimes mistaken for pyloric stenosis. It should not be, however, as the symptomatology of the two conditions is quite different. The vomiting of indigestion may begin at any time, is not explosive, usually occurs several times after the same feeding and depends on the character of the food. The vomitus almost always shows evidences of indigestion. The bowels may be constipated, but the stools always contain milk remains and are usually undigested. There is no visible gastric peristalsis and, of course, no tumor. Examination with the Roentgen ray shows that food begins to leave the stomach at once.

Another condition which is sometimes mistaken for hypertrophic pyloric stenosis is that in which vomiting occurs as the result of lack of tone in the cardia. In this condition the vomiting usually occurs soon after the food is taken, but, as a rule, only when the baby moves or cries. The vomiting is never explosive, the stools are composed of milk residue, there is no visible peristalsis or tumor, the nutrition is little, if at all, impaired. The Roentgen ray shows no signs of obstruction at the pyloric orifice.

Prognosis and Treatment.—Many authorities believe that this is a self-limited condition and that, if a baby can be tided over a few months,

the pylorus will again become patent as the result of the growth of the parts. In favor of this belief are the good results reported by many observers from medical treatment. On the other hand, it is well known that no food passes through the pylorus after a gastroenterostomy for this malformation, and that there is no change in the pylorus for many months after operation. In one of my patients food is still passing through the new opening after thirteen years. Furthermore, when gastroenterostomy is done for other conditions, the tendency is for the new opening to close and for the food to pass through the pylorus again. There is no data, as far as I know, as to the condition of the pylorus years after apparent recovery under medical treatment. There is, moreover, some reason to believe that the symptoms of obstruction may recur in later childhood or adult life.

FIG. 19.—Hypertrophic stenosis of the pylorus. Taken six hours after an opaque meal. Nothing has left the stomach.

Various forms of medical treatment are recommended by different observers, the most important of which are lavage, frequent small feedings, breast milk and the prevention of dehydration by the administration of salt solution or Ringer's solution by enema, the Murphy drip, or subcutaneously. Papaverine, cocaine and other sedatives have been recommended. Haas (Journal American Medical Association, 1922, **79,** 1314), who believes that this condition is simply a single manifestation of a general state, whose etiological factor is an overaction of the vagus portion of the autonomic nervous system, and usually a hyperexcitability of all the motor functions, believes that it can be cured with atropine. He reports forty cases treated in this way with only two deaths. He uses a 1–1000 solution, which must be prepared frequently from the crystals, as it deteriorates rapidly. He begins with 1–1000 grain at each feeding and increases the dose until the vomiting is relieved or there is flushing of the face, keeping on with this dose. He has given

as much as 16–1000 grain at a dose, or $\frac{1}{8}$ grain in twenty-four hours. Neither his premises nor his results have been verified by others.

Sauer (American Journal Diseases of Children, 1921, **22,** 166) believes that a large proportion of these cases can be cured by feeding thick cereal gruels. Porter and others have also used this method successfully. His explanation is that the thick, immobile cereal cannot be ejected from the stomach by the sudden explosive contractions that produce vomiting of milk feedings. The thick cereal remaining in the

Fig. 20.—Hypertrophic stenosis of the pylorus. Thirteen years after posterior gastroenterostomy. Nothing passes through the pylorus.

stomach can be moved along by the slow peristaltic contractions of the stomach wall. His mixture is made up of

Skimmed Milk	9 ounces
Water	12 ounces
Farina or Rice Flour	6 tablespoonfuls
Dextri Maltose	3 ounces

The mixture is boiled in a covered double boiler for an hour. He gives from two to six tablespoonfuls from six to seven times daily, and places the baby on the right side for a time after feeding. He puts the food in the mouth with a tongue depressor and scrapes it off with another. Porter puts the mixture into a Hygeia nipple, the end of which has been slit with a very sharp knife, with a spoon.

Equally good results have been obtained recently in these cases by operation, provided the operation has not been put off too long, because the condition was unrecognized or medical treatment persisted in. Gastroenterostomy has been superseded by splitting of the pylorus

(Rammstedt operation), which is a much simpler and better operation. Goldbloom and Spence (American Journal Diseases of Children, 1920, **19,** 263) found that the mortality with this operation was only one third as great when the symptoms had lasted less than four weeks as when they had lasted four weeks or longer, and that it was almost six times as large in babies that had lost 20% or more of their best weight than in those that had lost less than 20%. Babies can begin to take food within a few hours after this operation and, in a few days, are able to take considerable amounts of reasonably strong foods. Wollstein's studies (American Journal Diseases of Children, 1922, **23,** 511) show that the wound in the pylorus is healed within nine days and that the pylorus is relaxed within two weeks. The stomach returns to normal size within a month and the gap between the cut ends of the muscle coat has practically disappeared in six weeks. The conditions have been found to be normal at the end of two years, except for a fine, linear scar.

Although there is no doubt that the symptoms of hypertrophic pyloric stenosis in infancy disappear in many instances under medical treatment, it seems to me that the Rammstedt operation is a better method of treatment for this malformation. Recovery under medical treatment is always slow and by no means certain. Operation is often necessary after all, and, because of the delay, is less likely to be successful. The symptoms cease in a day or two after a successful Rammstedt operation and the baby is well in a couple of weeks. The mortality is no higher under surgical than under medical treatment. Surgeons competent to do the operation are at least as common as pediatrists and physicians competent to regulate the diet and properly treat this condition. It is not known whether or not the hypertrophy at the pylorus ever entirely disappears after medical treatment. There is reason to suppose that it may not and that it may be responsible for a recurrence of the symptoms in later life. It has been proved, on the other hand, that the pylorus is anatomically and functionally normal after the Rammstedt operation. I believe, therefore, that the wisest thing to do is to operate on these babies as soon as a positive diagnosis is made. If the baby is dehydrated, it is advisable, if possible, to delay operation for a day or so, in order to restore some of the lost fluid.

It is advisable to begin to feed babies within twelve hours after the Rammstedt operation. Diluted breast milk is the best food, next to this, whey or a whey mixture. The food should be given at intervals of two or two and one half hours and in small amounts, one half to one ounce at first. This is advisable, not because of any lack of patency at the pylorus, but because the intestine is unaccustomed to food and cannot, in the beginning, take care of much of it. The supply of fluid must also be kept up in some way, by mouth, rectum, or under the skin, for several days. In most instances the amount and strength of the food can be rapidly increased after the first two days.

CONGENITAL MALFORMATIONS OF THE INTESTINES

Stenosis or Atresia of the Intestines

Stenosis or atresia may occur at any part of the intestinal tract and may be sharply localized or occupy a considerable area. Atresia is more common than stenosis. The lesions are found most often in the duodenum or rectum.

Symptomatology.—The babies appear normal at birth. Vomiting, distention of the abdomen and the failure to pass stools containing milk remains are symptoms common to all. It is evident that the higher up the obstruction, the sooner vomiting will occur. If the obstruction is above the entrance of the common bile duct, the vomitus will not contain bile; if it is below it, the vomitus will contain bile; if it is very low down, the vomitus may be "fecal." As always, when vomiting is excessive, the vomitus may contain red or brown streaks of fresh or changed blood. If the obstruction is above the entrance of the common bile duct, the stools will contain bile and be like meconium; if the obstruction is below it, the stools may or may not contain bile, according to the time in fetal life at which the obstruction took place. Usually they do not contain it and are dry and grey. If the obstruction is very low down, no stools will be passed. The intestines become distended above the obstruction and collapsed below it. Therefore, if the obstruction is high up, there is very little distention of the abdomen, while there is much, if it is low down. There is, of course, rapid and progressive loss of weight and strength, and all the symptoms of starvation develop.

Prognosis and Treatment.—Except in some of the minor malformations of the rectum, these conditions are invariably fatal in a few days, unless the malformation is one which can be remedied by operation. The duration of life depends on the location of the obstruction, the rule being that the lower the obstruction, the longer the life. The outlook being hopeless in these conditions without operation, the abdomen should be opened in every instance as soon as the diagnosis is made, even though the chance of finding a remediable condition is very small.

Malformations of the Rectum

The abnormality may consist simply in an absence of the anus, the opening of the rectum being closed by skin. The anus, however, is usually present. There may then be an obstruction in the lumen of the rectum or the rectum may end at any level in a blind pouch connected with the anus by a fibrous cord. In other instances there may be simply a narrowing of the rectum of greater or less extent, but almost always at its lower end. In still other instances the rectum opens abnormally into the perineum, vulva, vagina or bladder.

Symptomatology.—The symptoms of these conditions are obvious, varying from those of intestinal obstruction to constipation and the discharge of feces in an unusual location, according to the nature of the malformation. It is evident, since such malformations are possible, that the examination of the new-born baby should include an examination of the anus, and that it should always be noticed whether the baby has passed feces during or after birth.

Prognosis and Treatment.—Strictures of the rectum can usually be readily overcome by dilatation. Abnormal openings must, if possible, be closed and a new opening be made in the normal situation. Operation should be delayed, however, until the baby is older and the parts larger than at birth, even though there is danger of infection of the mucous membranes in the meantime. Unless it is absolutely certain that the atresia is due only to an imperforate anus, it is far wiser to open the abdomen at once than to attempt to remedy the condition from below. It goes without saying that when there is atresia the operation must be performed immediately.

Patency of Meckel's Diverticulum

Meckel's diverticulum, which is a persistent omphalomesenteric duct, may maintain its connection with the navel and remain patent. If it is closed at its intestinal end, there will be a slight glairy discharge from the navel. If the intestinal end is open there will usually be a discharge of intestinal contents from the navel, that is, there is an *umbilical fistula*. If the mucous membrane protrudes, it forms what is called an *adenoma of the navel* or a *mucous polyp*. The tumor may vary from the size of a pea to that of a walnut. It has, of course, the appearance of a mucous membrane, and, like any exposed mucous membrane, is likely to become inflamed.

Diagnosis.—The only condition for which this malformation can be mistaken is a granuloma of the navel. Such a mistake is impossible, if there is a fecal discharge, but may be made through carelessness, if there is not. The protruding duct is symmetrical and looks like mucous membrane, while the secretion is colorless and glairy. A granuloma is irregular and looks like granulation tissue, while the discharge is a thin pus. An opening can be found in a protruding duct with a fine probe; there is no such opening in a granuloma.

Prognosis and Treatment.—When there is no protrusion of the mucous membrane at the navel, there is a chance that the canal may eventually become obliterated and that the discharge will cease, but the chances are that it will not. There is no chance of spontaneous recovery, when there is a protrusion of the mucous membrane. The only rational treatment, therefore, is the removal of the diverticulum from the inside of the abdomen and the closure of the navel. It is safe to delay the operation until the baby is several months old.

CONGENITAL MALFORMATIONS OF THE BILE DUCTS

Congenital malformation of the bile ducts is a relatively uncommon malformation. It is more common in males than in females and may occur in several members of the same family. Babies with this malformation usually appear normal at birth, but are sometimes jaundiced. If jaundice is not present at birth, it develops in two or three days and always under five days, and increases progressively, becoming very marked. After the jaundice has developed the urine contains bile. The stools may, at first, appear like meconium, if the bile ducts were not closed until late in fetal life. Usually, however, they are gray or white and remain so. If colored at first, they become gray or white after a few days. The liver is almost invariably much increased in size, but in a few cases has not been. The spleen is always enlarged, usually considerably. The nutrition is surprisingly well maintained. Itching is not marked. Death usually occurs from hemorrhage in a few weeks or months. If not, death occurs from malnutrition, almost always in less than eight months, although one case has lived ten months.

Pathology.—All sorts of malformations of the bile ducts have been found, varying from an absence of most of the biliary passages to localized atresia. The most common malformation is the absence or atresia of the common duct. It is, of course, unimportant if the cystic duct is alone involved, and there are no symptoms. The pancreatic duct is also often malformed. If so, the symptoms of indigestion are more marked. There are almost always cirrhotic changes of the biliary type in the liver.

These are probably secondary to the obstruction to the outflow of bile. In some cases, however, they may possibly be primary and the lesions of the ducts due to a descending cholangitis. It is possible that there may be two varieties of congenital malformation of the bile ducts; one a congenital error in development and the other secondary to cirrhosis of the liver from some sort of toxemia. Syphilis plays no part in the etiology of this condition.

Diagnosis.—There are several conditions with which congenital malformation of the bile ducts may be confused. These are *congenital syphilis, septic infection of the new-born, icterus neonatorum* and *catarrhal jaundice.*

The liver and spleen are sometimes much enlarged in *congenital syphilis.* There may also be jaundice. The jaundice is, however, not as marked as in congenital malformation of the bile ducts. The stools contain bile. The urine often does not contain bile. There are also other signs of congenital syphilis, usually marked. The Wassermann test is positive.

Jaundice is a not uncommon symptom of *septic infection of the new-born.* It is, however, almost never as marked as in congenital malformation of the bile ducts. The liver and spleen may be enlarged, but seldom to the degree which is usual in congenital malformation of the bile ducts. The stools contain bile; the urine does not. The temperature is elevated and the baby is manifestly seriously ill, whereas in congenital malformation of the bile ducts there is no fever and the child does not seem ill.

The time of the appearance of the jaundice is the same in *icterus neonatorum* as in congenital malformation of the bile ducts and the jaundice may be just as deep. There is, however, no enlargement of the liver and spleen in icterus neonatorum. The stools contain bile and the urine does not. Even if there was a question as to the diagnosis in the beginning, it would be plain in a short time, because icterus neonatorum is a temporary condition and congenital malformation of the bile ducts a permanent one.

Catarrhal jaundice is most unusual in infancy, especially in early infancy. In both catarrhal jaundice and congenital malformation of the bile ducts there is jaundice and bile in the urine and no bile in the stools. The liver, however, is but slightly, if at all, enlarged in catarrhal jaundice and much enlarged in congenital malformation of the bile ducts. The spleen is never enlarged in catarrhal jaundice and is always enlarged in congenital malformation of the bile ducts. There is usually fever and the baby is sick with catarrhal jaundice, while there is no fever and the baby is not sick in congenital malformation of the bile ducts.

It must be remembered in differential diagnosis that there are two possible sources of error as regards the stools. There is a form of bile pigment which is colorless, leucohydrobilirubin, and it is possible that traces of bile may be excreted through the intestinal mucosa. In doubtful cases, therefore, it is well to try the corrosive sublimate test for bile on the stools before assuming that there is no bile in them. On the other hand, the finding of traces of bile does not necessarily mean that the bile ducts are patent.

Treatment.—Congenital malformation of the bile ducts cannot be cured by medical treatment. Babies with this malformation can digest and utilize proteins and carbohydrates fairly well, but are not able to utilize more than from one fourth to one half of the normal amount of fat. It is, therefore, advisable to keep the fat in their food low or to give

bile salts. Holmes (American Journal Diseases of Children 1916, **11**, 405) found, in his analysis of all the reported cases, that the condition might possibly have been remedied in about 15%. Hence, it is not only justifiable, but advisable, to operate on these cases in the hope that a remediable condition may be found. No harm, at any rate, can be done, because there is no chance of recovery under medical treatment. If the pancreatic duct is also obstructed, the condition is, of course, absolutely hopeless. It is possible to determine with the duodenal catheter whether or not pancreatic secretion is entering the intestine. If it is impossible to make this examination, these cases should be operated upon. They should not be, if no pancreatic secretion reaches the intestine.

CONGENITAL CYSTIC KIDNEYS

Congenital cystic malformation of the kidneys is due to an error in development. The urinary passages below the kidneys are normal, but other malformations in various parts of the body are not uncommon. The cysts vary much in size and replace some or all of the true renal tissue. The kidneys may be normal in size or nearly fill the abdomen. Both kidneys are almost always affected.

If the kidneys are much enlarged, they may interfere with delivery and perhaps render an operation, of course, fatal, necessary. If the baby is born alive and the kidneys are enlarged, the abdomen is more or less distended, according to the size of the kidneys, the enlargement being primarily in the flanks and extending forward. These tumors have the characteristics of other solid tumors of the kidney, being peculiar only in that they are bilateral. The surface is irregular, they are dull on percussion, fill up the flanks and can usually be pushed forward a little. The only other tumor with which they are likely to be confused is sarcoma of the kidney, which is unilateral and which, moreover, is not congenital. They cannot be removed, because the removal of both kidneys is necessarily fatal. If the kidneys are not enlarged, the malformation cannot be recognized.

The onset and the severity of the symptoms due to the lesions in the kidneys depend upon the amount of kidney tissue which is left, not on the size of the kidneys. If there is little or none, as may be the case, whether the kidneys are large or small, death occurs from uremia in a few hours or days after birth. If there is a great deal, this malformation is not incompatible with normal growth and development, even to adult life. In most of the cases which are not rapidly fatal, however, interstitial changes gradually develop in the kidney tissues which are left, bring on the symptoms of chronic nephritis and finally cause death. In such cases, unless the kidneys are enlarged, the true cause of the trouble is never suspected. The treatment is, of course, the same as when the nephritis is due to other causes.

CONGENITAL MALFORMATIONS OF THE URACHUS

The intraembryonic portion of the allantois is a tube which extends from the lower end of the intestine to the umbilicus. The middle segment of this tube dilates in the second month and forms a spindle-shaped sac, which becomes the urinary bladder. The part of the tube between this sac and the navel remains small, loses its lumen and, at birth, is an impervious cord, known as the urachus.

Fistula of the Urachus

If the part of the tube between the bladder and the navel does not close, there is, of course, a free connection between the bladder and the navel, and urine runs from the navel. If the tube is closed at the bladder, but open above, there will be a glairy discharge from the opening at the navel. The mucous membrane lining the canal usually protrudes a little. Sometimes, however, it does not, and there is simply a dimple or a small slit where the urachus joins the skin. In rare instances calculi have been found in the canal.

Diagnosis.—If the discharge at the navel is watery, the proper tests will show that it is urine. If it is glairy, it may be from either the urachus or a patent Meckel's diverticulum. If it is from the latter, a probe will pass straight into the abdomen; if it is from the urachus, it will pass downward toward the bladder.

Treatment.—The treatment is purely surgical. The urachus should be dissected out and, if the canal connects with the bladder, it should be amputated at the bladder.

Cyst of the Urachus

If the tube closes at both ends, but does not close in the middle, it may become distended by the secretion of the lining membrane and form a tumor in the median line of the abdomen, below the navel and not far from the surface. This tumor will have the characteristics of an unilocular, encapsulated, non-inflammatory collection of fluid. It is conceivable that the tube may close above, but remain open in the middle and at the lower end. If so, it will be filled with urine when the bladder is full and form a tumor which will disappear when the bladder is emptied.

Diagnosis and Treatment.—If these tumors are borne in mind, they are easy to recognize and can hardly be mistaken for anything else. The treatment is purely surgical. They should be entirely removed.

CONGENITAL MALFORMATIONS OF THE EXTERNAL GENITALS

Hypospadias

In this malformation, which is due to an arrest of development occurring before the end of the fourth month of fetal life, the opening of the urethra is somewhere in the median line of the lower surface of the penis, instead of at the end of the glans, as normally. The portion of the unclosed urethra beyond is represented by a groove in the median line on the lower surface of the penis. This deformity is described as perineal or scrotal, penile, and balanic, or glandular, according to the location of the opening. It causes no symptoms, unless the opening is constricted, but simply makes the passing of urine inconvenient. If the opening is far back, it may, however, interfere in adult life with procreation.

The treatment is, of course, purely surgical and extremely difficult. It should not be undertaken until the penis is sufficiently developed to make a satisfactory result possible. It seems to be questionable whether it is advisable to interfere, if the opening is in the glans.

Undescended Testicles. Cryptorchidism

During the fifth and sixth months of fetal life the testicles are in contact with the anterior abdominal wall, near the inner abdominal ring. They enter the inguinal canal in the eighth month and pass into the scro-

tum a short time before birth. In many full-term babies, however, one or both testicles are in the canal and not very infrequently in the abdomen. In the vast majority of the cases in which the testicle is in the canal, it passes into the scrotum within a few weeks or months, and always within five or six years. When the testicle is within the abdomen at birth, it may come into the canal in a short time and finally come into the scrotum. The chances are, however, that it will not, unless it comes into the canal within a few weeks or months after birth. There are no symptoms from this anomaly in early life, unless the testicle, which is exposed to internal injury when it is in the canal, is struck or bruised. The misplaced testicle is, however, quite liable not to develop properly This is not of great importance, if one testicle is in its normal position in the scrotum. Malignant growth is not infrequent in testicles which are retained in the abdomen.

Treatment.—When the testicle is in the canal, something can be done to hasten its descent into the scrotum by manipulation. A truss should never be applied above it to prevent its sliding back. If it does not pass into the scrotum before six years, an operation, which will usually be successful, should be performed. If one testicle is in the abdomen and the other one is in the scrotum, operation, which may or may not be successful, is not necessary, but not contraindicated. If both testicles are in the abdomen, operation may be performed in the hope that, if the testicles can be brought out and down, the chance of sterility may be diminished. These operations should be done when the child is five or six years old.

Diagnosis.—It must not be forgotten that in the infant and young child, the testicle easily slips back into the abdomen. The diagnosis of undescended testicle is not justified, therefore, unless the testicle is constantly absent or constantly in the canal. If the testicle is in the abdomen, it is possible that, on account of the absence of the testicle from the scrotum, an enlarged gland or encysted hydrocele of the cord may be mistaken for it in the canal. It is easy to distinguish between an encysted hydrocele of the cord and a testicle in the canal because one is a sac filled with liquid, while the other is a solid body. It is harder to distinguish between an enlarged gland and a testicle in the canal, as both are solid. A gland is usually harder than a testicle, however, not exactly the same size as the other testicle, and varies in size.

Adherent Prepuce and Phimosis

In the normal male infant at birth the inner membrane of the prepuce is almost invariably adherent to the glans. The adhesions gradually become loosened by the motion and growth of the parts, so that in a few years the prepuce can usually be easily retracted. An adherent prepuce causes no interference with the passage of urine. It may favor the development of balanitis, but the adhesions have usually disappeared before the time at which this condition is likely to develop.

Phimosis is the condition in which the prepuce cannot be retracted over the glans. It varies greatly in degree. In some cases the opening is no more than a pinhole, in others a part of the glans is visible. When the prepuce is also elongated, the condition is called hypertrophic phimosis. Phimosis is usually accompanied by adhesions of the prepuce to the glans. When the prepuce can be pulled back so that the opening of the urethra is plainly visible, there is no interference with or discomfort in the passage of urine. If it cannot be, there may be ballooning of

the prepuce and pain in passing urine. Phimosis predisposes to the development of balanitis, but this rarely happens in infancy. If balanitis develops, it will cause discomfort in the same way as any other mild inflammatory process. It is conceivable that long-continued mild balanitis may get a baby or young child into the habit of playing with the penis. It is also possible that balanitis may reflexly cause or keep up incontinence of urine. I am convinced, however, that there are no other symptoms from an adherent prepuce or phimosis. I have seen many instances in which nervous symptoms of various sorts were attributed to them, but have always been able to find some other cause for them, on the removal of which the symptoms ceased. I have also seen many children who had been previously and, of course, unsuccessfully, circumcised for the relief of the very symptoms for which they were brought to me.

Treatment.—Although probably unnecessary, it is usually advisable, in the interest of cleanliness, when the prepuce is simply adherent, to break up the adhesions by pulling back the foreskin some time during the first few weeks of life. If the adhesions are at all firm, they can be broken with a director or probe. The glans should be greased with vaseline daily for one or two weeks. After this, the prepuce should be pulled back and the parts washed at least twice a week. Circumcision should never be considered for the relief of simple adhesions. If there is phimosis, it is usually possible to gradually dilate the prepuce by working it back a little at a time or by stretching it slowly with forceps. When there has been a phimosis, it is most important to guard against the development of *paraphimosis*, the first few times that the prepuce is pushed completely back. This can be readily avoided by not leaving the foreskin pushed back but an instant. The reformation of adhesions can be prevented by pulling the foreskin back daily for a week or two and greasing the glans with vaseline. If the foreskin is very long and thick and cannot be readily stretched, an operation is advisable. Circumcision is preferable to slitting, as the cosmetic results are better and simple slitting does not do away with the redundancy of the prepuce.

I am not in favor of routine circumcision, even though it is endorsed and urged by nasty-minded nurses and surgeons in need of business. The prepuce serves to protect the sensitive glans and should be kept, if possible. The presence of the prepuce does not of itself predispose to masturbation. Even if it makes the development of balanitis easier, this can be prevented by ordinary cleanliness. The removal of the prepuce for masturbation usually stops the habit, while the wound is healing, but no longer. There is no evidence to show that Jews masturbate less than Christians. Circumcision does not cure epilepsy, chorea or nervous habits or make intellectual prodigies out of idiots, because a long prepuce, whether or not adherent, has nothing to do with the causation of these diseases.

The prepuce is also almost always adherent to the clitoris in female infants. For some reason or other it has not yet become the custom to strip it back as a routine, as is the case with boys. It has also usually been overlooked as a cause of nervous symptoms and diseases, except by the Freudians. As a matter of fact, adherence of the prepuce causes no symptoms, of course, unless smegma collects beneath it. Then there may sometimes be local discomfort, which may lead to handling of the parts. Pseudomasturbation is more common in girls from this cause than in boys. In older children it may also be responsible to some extent for incontinence.

It is easy to strip back the prepuce from the clitoris and remove the smegma. The parts should then be washed once or twice a week. Circumcision is almost never necessary.

Atresia of the Vulva and Hymen

Either the labia majora or the labia minora, more often the labia minora, may be adherent. The union may be firm, but the adhesions are usually slight and easily broken up with the fingers. In some instances, the labia are joined by a thin membrane, which is also easily ruptured.

The opening in the hymen is often very small in babies and young girls. Occasionally the opening is filled by a thin membrane, which is easily ruptured. In very rare instances the hymen closes the vagina. No symptoms are produced, however, as a rule, until menstruation begins.

Hydrocele

A pouch of the parietal peritoneum passes through the inguinal canal into the scrotum, before the testicle begins to descend behind it. This is known as the processus vaginalis. Normally, this pouch is obliterated to form a fibrous cord, except in the lower part, where it forms a sac, known as the tunica vaginalis, which surrounds the testicle, except above and behind. When there is an accumulation of serous fluid in some part of this peritoneal pouch, it is called a hydrocele. There are several varieties of hydrocele, according to the location of the fluid.

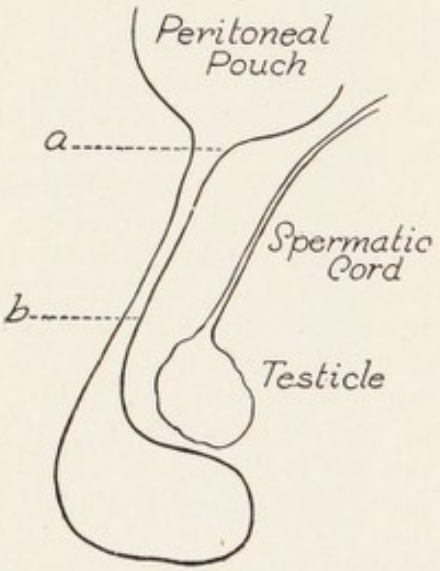

Fig. 21.—Anatomy of hydrocele.

If the peritoneal pouch remains patent throughout, the condition is known as *congenital hydrocele*. The tumor is long and tubular above, but larger and rounded below in the scrotum. It is elastic, fluctuant and translucent. The testicle can be felt high up posteriorly. When the baby is on its back and the scrotum is lifted, the fluid runs back into the abdomen, or, if it does not, can be pushed back. Congenital hydrocele may be confused with inguinal hernia or complicated by it. If there is a hernia, the tumor is less elastic, does not fluctuate and is not translucent. Gurgling can usually be felt in it, at any rate, when it is pushed back. The differential diagnosis is harder when there are both a congenital hydrocele and a hernia, but must be made on the same principles. If both are present, the hydrocele is of relatively little importance.

If the peritoneal pouch is closed where it leaves the abdomen, (a), and open below, the condition is known as *infantile hydrocele*. The characteristics of the tumor are the same as in congenital hydrocele except, of course, that the fluid cannot be returned to the abdomen.

If the peritoneal pouch is open above, but is closed where it should be below (b), the condition is known as *funicular hydrocele*. The swelling is cylindrical, elastic, fluctuant and translucent. The fluid can be returned into the abdomen. This form of hydrocele may also be complicated by or mistaken for an inguinal hernia. The diagnosis between hernia and

funicular hydrocele is the same as between hernia and congenital hydrocele.

If the canal is closed both above and below (a and b), the condition is known as *encysted hydrocele of the cord.* The tumor is tubular, elastic, fluctuant and translucent. The canal is often closed in part and the tumor is, in consequence, smaller. It may be mistaken for an enlarged gland or an undescended testicle. An enlarged gland is hard, inelastic, does not fluctuate and is opaque. If the testicle is in the canal, it is not in the scrotum. The testicle is semi-elastic, but is harder than a hydrocele, is opaque and not fluctuant.

A similar process of the peritoneum may extend, in connection with the round ligament, into the inguinal canal in females, and is known as the canal of Nuck. This canal, while cut off above, may remain patent and become filled with serum. The condition is then analgous to an encysted hydrocele of the cord and is known as *encysted hydrocele of the canal of Nuck.* This condition is often mistaken for an enlarged gland. The differential diagnosis between an enlarged gland and an encysted hydrocele of the canal of Nuck is the same as between an enlarged gland and an encysted hydrocele of the cord.

If the canal is closed, and there is a collection of fluid in the tunica vaginalis the condition is commonly spoken of as hydrocele, but is more properly termed *hydrocele of the tunica vaginalis.* The tumor is, of course, entirely in the scrotum. It is elastic, fluctuant and translucent. The testicle can be felt high up posteriorly. It is hard to mistake this condition for anything else, if it is borne in mind. If it is not, it may carelessly be mistaken for an inguinal hernia or an enlarged testicle.

Prognosis and Treatment.—The tendency of all forms of hydrocele is to spontaneous recovery. This tendency is less marked in the congenital and funicular forms. The application of a truss may help to cure the congenital and funicular forms by preventing the entrance of fluid into the canal. The truss also diminishes the danger of hernia. If the truss does not cure the hydrocele in a year, an operation is necessary. Tapping will usually cure infantile hydrocele, encysted hydrocele of the cord and of the canal of Nuck and hydrocele of the tunica vaginalis. If it does not, after several trials, operation may be performed. It is wise, however, not to hurry about it. It is wiser not to inject iodine or other irritants into an infantile hydrocele, encysted hydrocele of the cord or hydrocele of the tunica vaginalis as they may cause too much irritation. It is very dangerous to inject them into the congenital and funicular forms, because of the possibility of setting up peritonitis.

CONGENITAL MALFORMATIONS OF THE EXTREMITIES

There are many congenital malformations of the extremities, most of which are so rare as to be unimportant. Among the more common malformations are *supernumerary digits* and *webbing of the fingers and toes.* The treament of these conditions is, of course, purely surgical.

CLUB-FEET

Clubbing of the feet is more common and more important. It may be of various degrees. Many clubbed feet can be cured or, at any rate, helped by proper treatment in early infancy. Manipulation is often sufficient in mild cases. In more severe cases plaster casts should be applied. It is wise, however, to consult an orthopedic surgeon, except in the mildest cases.

Deformities from Amniotic Bands

The formation of annular grooves in the extremities or even the amputation of a finger, toe or a large part of an extremity by amniotic bands is not very uncommon. These deformities are usually attributed to maternal impressions. They cannot, of course, be due to maternal impressions, because it is impossible for a mother to "mark" her child, even if she wished. Furthermore, in most cases, the impression which is supposed to have caused the deformity occurred long after the limbs were formed.

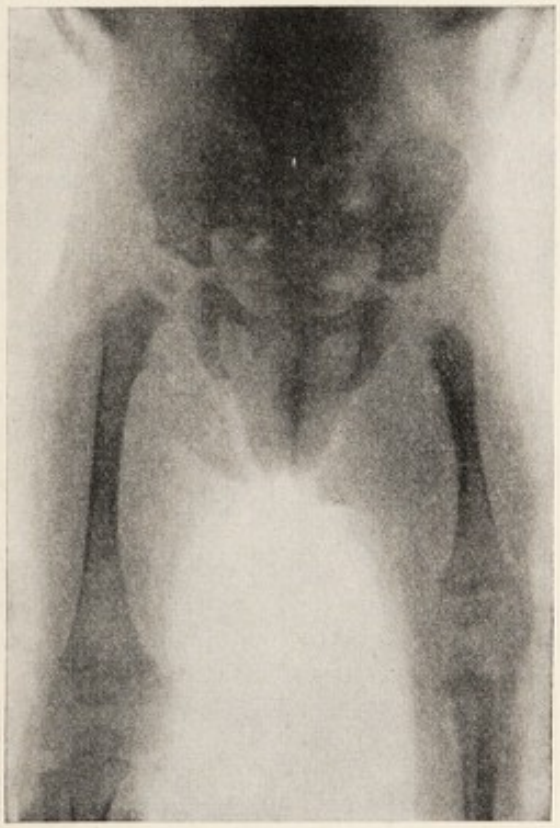

Fig. 22.—Congenital malformation of thigh.

Fig. 23.—Deformities from amniotic bands.

Congenital Dislocation of the Hips

Congenital dislocation of the hip is not very uncommon. It is usually overlooked until the child begins to walk. It should, however, always be looked for as a part of the general physical examination of a baby. With the baby lying on its back, the legs should be fully extended. If there is a congenital dislocation of one hip, that leg will apparently be shorter than the other. It can, however, be pulled down into place, but will slip up again when the pull is relaxed. When there is any question of congenital dislocation of the hip, Nelaton's line should be drawn, that is, a line from the anterior superior spine of the ilium to the tuberosity of the ischium on the same side. If there is a dislocation, the trochanter will be found above this line. If there is a double congenital dislocation, it may be missed on comparing the legs. It is always wise, therefore, to determine whether the trochanters are in the proper position or not.

Treatment.—Much can be done for congenital dislocation of the hip by proper manipulations in infancy. In some instances, the condition may be cured at this time without operation. It is very important, therefore, to recognize it early.

NEVI

By the term nevus is meant a congenital overgrowth of some element of the skin. These overgrowths are commonly known as birthmarks. Nevi may be pigmented or vascular. The etiology of nevi is unknown. They are certainly not due to maternal impressions, as is so often erroneously supposed.

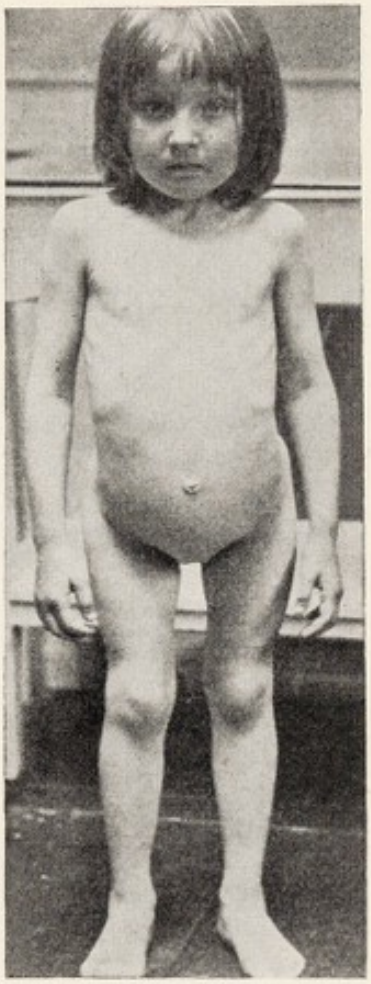

Fig. 24.—Congenital dislocation of the hips.

The pigmented forms may be simply brownish discolorations of the skin, or raised and warty, or hairy. When they occur over the lumbar region of the spine they usually indicate that there is a spina bifida occulta. The pigmented forms of nevi seldom disappear spontaneously.

Treatment.—It is wise to leave the smooth, pigmented moles alone, unless

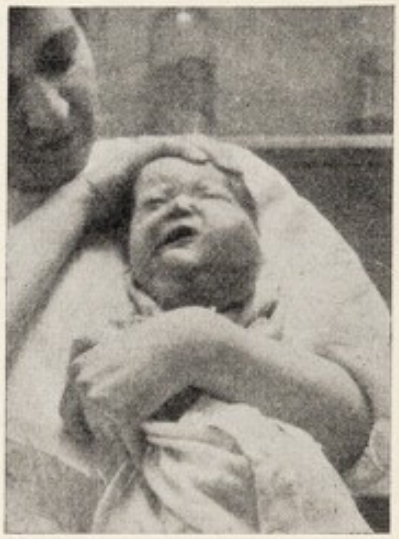

Fig. 25.—Large vascular nevus of cheek.

they are in a situation where they are disfiguring. The warty ones should be removed, if complete removal is possible. If not, it is wiser to leave them alone, as there is considerable danger of metastasis if they are not completely removed. The hairy moles may be excised or treated by electrolysis.

The vascular forms of nevi vary from small, superficial discolorations of the skin to large, bulging, red areas covering half of the face or a whole extremity. The small, superficial discolorations usually disappear spontaneously in a few years. If they do not, or if it is desired to hasten their disappearance, they can be cured by freezing with liquid air or carbon dioxide snow. All nevi, even if small and superficial, should be watched carefully to see if they are enlarging. If they are, they should be treated at once. If vascular nevi are large and raised, they should be treated as soon as possible, because they almost never decrease in size,

but usually increase, often very rapidly. They may rupture and bleed profusely, or, by increasing in size, cause pressure on other structures. Great judgment is required as to the proper procedure to be used in the individual case. Electrolysis by multiple punctures, freezing with liquid air or carbon dioxide snow, or the actual cautery may be tried. The best method of treatment, however, is the removal of the nevus. In many cases, however, this is impossible, and in others dangerous, because of the difficulty in getting at all the enlarged vessels.

MONGOLIAN SPOTS

Mongolian spots are blue or grayish blue spots on the skin. They vary in size from that of a small coin to that of the hand. They are most common in the sacral and lumbar regions or on the buttocks, but are sometimes found on the shoulders and extensor surfaces of the extremities. They are usually present at birth, but may not appear until several weeks after birth. The color deepens for a few months and then gradually fades away. They almost always disappear before the child is six years old, but sometimes persist into adult life. They are present in at least 90% of the Mongolian and African races, but may be found on the babies of all races. It was formerly supposed that they indicated an admixture of Mongolian or African blood, but it is now known that they occur more or less commonly in all races.

They are due simply to the persistence in a rudimentary form of a deep layer of pigmented cells in the corium, which was probably well developed in some of our prehistoric ancestors. This layer is well developed in certain monkeys and accounts for the peculiar bluish color of their skins. These pigmented cells can be found microscopically deep in the corium in many white children that do not show the spots. The difference in the frequency and in the size of the spots in different races and individuals apparently depends entirely on the general tendency to pigmentation of the skin in the given race and individual.

These spots are harmless, cause no symptoms, disappear of themselves and require no treatment. They are important only in that they are sometimes mistaken for ecchymoses or give rise to the unwarranted suspicion, when they occur in white babies, that there has been some admixture of Mongolian or African blood.

SECTION II

BIRTH INJURIES

CAPUT SUCCEDANEUM

A caput succedaneum is a swelling of the scalp due to edema in the soft tissues. It is situated over the presenting part and is due to the lack of pressure in this situation. It is rounded, and largest at the base. It is covered with skin and hair, pits on pressure and is neither fluctuant nor translucent. It is not hot, red or tender. Pressure on it causes no symptoms of cerebral irritation nor bulging of the anterior fontanelle. It gradually diminishes in size and is gone in a few days.

The only conditions with which it can be confused are *cephalhematoma, meningocele* and similar conditions, and a *lipoma* or a *nevus*. The differential diagnosis between these various conditions has been taken up in detail in the section on *meningocele*.

CEPHALHEMATOMA

A cephalhematoma is caused by a hemorrhage between the periosteum and one of the cranial bones. As the periosteum is attached to the edge of the bone, the swelling is always limited to one bone, usually one of the parietals. There may, however, be more than one cephalhematoma on the head at the same time. A cephalhematoma is not present at birth but develops soon after. It is rounded, and largest at the base. It is covered with skin and hair and is fluctuant, but not translucent. It is not hot or tender. It is never red, but sometimes has a slight bluish tinge. Pressure on it causes no symptoms of cerebral irritation and no bulging of the anterior fontanelle. A boney ridge is usually palpable at the edge of the tumor after a few days.

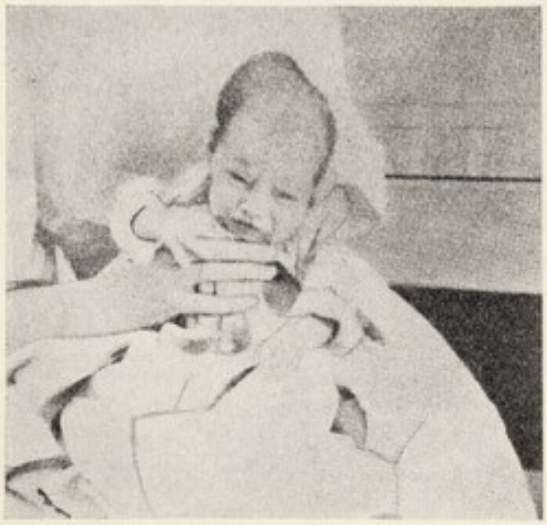

FIG. 26.—Cephalhematoma.

Diagnosis.—The only conditions with which a cephalhematoma can be confused are *caput succedaneum, meningocele* and similar conditions, a *lipoma* or a *nevus*. The differential diagnosis between these various conditions has been taken up in detail in the section on *meningocele*.

Prognosis.—The tumor begins to diminish in size in a few days or a week and is gone in a few weeks. The boney ridge often increases in size and sometimes may be felt for months after the disappearance of the tumor. It, however, eventually disappears. In rare instances, a cephalhematoma becomes infected and an abscess forms. This almost never happens, however, unless some attempt at treatment has been made.

Treatment.—The treatment of a cephalhematoma is to leave it entirely alone. If it is left alone, it will almost invariably disappear without causing any trouble. Pressure on it cannot possibly do any good. Tapping it may possibly hasten its disappearance, but the chances

FIG. 27.—Cephalhematomata.

of infection and subsequent abscess are so great that it is never advisable to tap it.

HEMATOMA OF THE STERNOCLEIDOMASTOID

A hematoma of the sternocleidomastoid muscle is due to a rupture of the blood vessels and muscle fibers of the muscle as the result of stretching of the neck during labor. It is usually unilateral, but may be bilateral. The rupture usually occurs at the junction of the middle and lower thirds of the muscle. There are usually no evidences of the injury at birth, although there may be a soft, somewhat fluctuant swelling at the seat of the injury. There is no tenderness, and the head is held in the normal position.

FIG. 28.—Hematoma of the sternocleidomastoid.

The blood is gradually absorbed and scar tissue forms at the seat of the injury. This makes a hard lump in the muscle, and it is at this time, a few weeks after birth, that the injury is usually first noticed. At this time, the head is usually still held in the normal position.

The scar tissue soon begins to contract and consequently shortens the muscle. As the result of the shortening, the head is pulled downward toward the shoulder on the same side and the face is turned a little toward the other side. At this time

the tumor in the muscle is usually much less noticeable. At no time does the baby have any discomfort from this injury.

Diagnosis.—It seem hardly reasonable that this injury can be mistaken for anything else. Nevertheless, I have known it to be mistaken for an enlarged gland and a sarcoma. If the situation of the tumor is borne in mind, it cannot be mistaken for these other conditions. The tumor is in the muscle itself; enlarged glands and sarcomata may be near the muscle, but they are not in it.

Prognosis.—In a good many instances in which the injury has been small, the blood is absorbed and the injury repaired without the development of any deformity. In most instances, even if some torticollis has developed, it eventually disappears. It is only in the rarest instances that permanent deformity results.

Treatment.—Nothing should be done for two or three weeks. Regular massage of the muscle should then be begun and the neck should be stretched in order to prevent the development of contractures. If contractures are developing, these measures should be kept up and, in addition, a pad or Thomas collar applied to prevent further contracture. If a permanent contracture has developed, operative interference is necessary.

OBSTETRICAL PARALYSIS

Obstetrical paralyses are peripheral paralyses due to injuries during labor. They are of two types, the facial and the arm.

Facial Paralysis

Facial paralysis is caused by pressure, usually of the forceps, on the trunk of the facial nerve after it has left the stylo-mastoid foramen. All the branches of the nerve are, therefore, involved and there is a complete paralysis of one side of the face. The baby is unable to close the eye on the affected side or to wrinkle the forehead. The mouth is drawn to the opposite side. The paralysis is most marked when the baby cries. The conjunctiva is usually somewhat irritated on the affected side.

Fig. 29.—Obstetrical paralysis of face.

Diagnosis.—The only other form of facial paralysis which a new-born baby can have is one due to a lesion of the facial nerve within the skull. When the lesion is central, the upper branch of the nerve is not involved and the baby can, therefore, close its eye and wrinkle its forehead. Furthermore, if the paralysis is central, there is almost invariably paralysis of one or more of the extremities, in addition.

Prognosis.—Recovery almost invariably takes place within a few weeks or, at most, months. Occasionally, however, a patient does not recover entirely and a certain amount of paralysis persists through life.

Treatment.—No treatment is necessary. In fact, it is advisable not to disturb the baby by any attempts at massage or the application of electricity.

Paralysis of Arm

Obstetrical paralysis of the arm is due to injury to the brachial plexus by the separation of the head and shoulder during delivery, which causes stretching of the nerves. It is not due to the pressure of the forceps. Asphyxia, with the consequent relaxation of the muscles, favors the development of this injury. As the result of the stretching, the nerve trunks are stretched, frayed out or ruptured. There is also hemorrhage from the torn vessels into the nerve sheaths.

The brachial plexus is made up of the fifth, sixth, seventh and eighth cervical and the first dorsal nerves. The fifth and sixth cervical nerves are the ones most commonly affected. Injury to these trunks causes the upper arm type of obstetrical paralysis, which corresponds to Erb's paralysis in later life. The lower trunks are less often affected. When they are, the stress of the injury falls on the first dorsal and eighth cervical nerves. Injury to these nerves causes the lower arm type of paralysis, often known as Klumpke's paralysis. When all the trunks are affected, the paralysis is of the combined type. In other instances, in which the injury to the nerve trunks has been unusual, various modifications of these types occur.

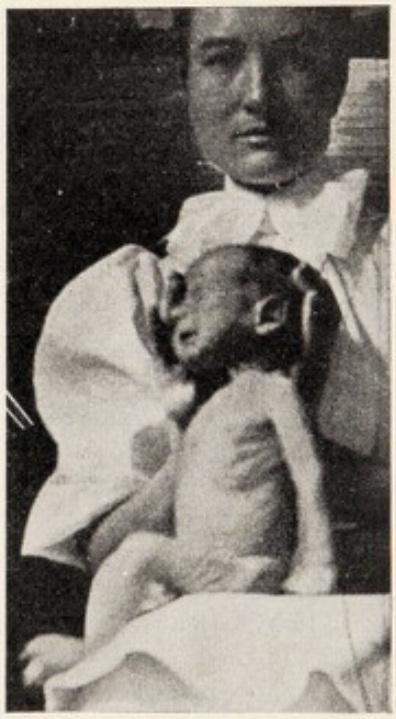

Fig. 30.—Obstetrical paralysis of arm.

Upper Arm Type.—In this type, which is due to injury to the fifth and sixth cervical nerves, the deltoid, biceps, brachialis anticus, supinator longus, infraspinatus and supraspinatus, and the serratus magnus are the muscles chiefly involved. As the result of the involvement of these muscles, the arm hangs limp at the side, extended at the elbow and with the palm turned back.

Lower Arm Type.—In this type the muscles chiefly involved are those of the forearm. The triceps is usually also affected. The arm is flexed at the elbow, the hand is flaccid, extended at the wrist, with the fingers flexed.

Combined Type.—In the combined type the position is a combination of those found in the two other types and necessarily varies somewhat according to which nerves and muscles are most affected.

The paralysis in all cases is at first flaccid. Later, contractures of the non-paralyzed muscles develop as the result of the lack of opposition. The reaction of degeneration is present in the affected muscles. These muscles gradually atrophy. The reflexes which depend on the nerves and muscles involved in the paralysis are absent or diminished. Sensation is usually normal.

Diagnosis.—At first, fracture of the clavicle or dislocation of the shoulder may be mistaken for obstetrical paralysis. When the lack of use is due to fracture of the clavicle, it is from pain; not from paralysis. Careful examination will reveal the fracture and show that the baby can move the arm. When the loss of use is due to dislocation of the shoulder, careful examination will show that the head of the bone is not in the normal position and that the failure to use the arm is due to pain,

not to paralysis. Obstetrical paralysis of the arm may possibly be confused with paralysis due to an intracranial lesion. When the paralysis is due to an intracranial lesion, however, it soon becomes spastic, instead of flaccid, and the reflexes are exaggerated, rather than diminished. Furthermore, paralysis in a new-born baby, due to an intracranial lesion, is almost never limited to one extremity. If it was, moreover, all the muscles of the extremity would be involved, not certain special ones.

In after years, it is sometimes difficult to distinguish between obstetrical paralysis of the arm, infantile paralysis and cerebral paralysis. Both infantile paralysis and obstetrical paralysis are of the flaccid type with contractures of the antagonists. Infantile paralysis is, however, not congenital. While the paralysis might conceivably involve the same muscles which are affected in obstetrical paralysis, it almost never does. In old cerebral paralysis, all the muscles of the extremity are involved, not certain special ones. The contractures are of the stronger muscles and the reflexes are exaggerated.

Prognosis.—The prognosis must, necessarily, depend on the amount of injury done to the nerves. If the nerves have been simply stretched or frayed out a little, complete or nearly complete repair and restoration of function is possible. If the nerve trunks have been much frayed or ruptured and hemorrhage has occurred between the ends, there will be retraction of the broken nerves and the formation of scar tissue in the frayed-out nerves, or between the ends, which will prevent the transmission of nerve impulses. Clinically, many of the milder cases recover to a considerable extent, but almost never completely. The severe cases usually improve somewhat, but, as a rule, are left with very considerable paralysis. Improvement is most rapid and greatest in the first three months. Little improvement can be expected after a year. In the severe cases there is a retardation of the growth of the bones of the extremity, with shortening.

Treatment.—In the beginning, the treatment consists entirely in support, by a sling or some other means, of the arm. After two or three weeks massage should be begun with the object of keeping up the muscular tone. Electricity may be used for the same purpose. If the muscles react to Faradism, this should be used. If they do not, galvanism should be used.

There is much difference of opinion as to whether operation on the nerve trunks is advisable or not in these cases and, if so, at what time the operation should be done. Certain surgeons feel that, at any rate in cases in which the paralysis is complete or nearly complete, operation should be done on the plexus at one month, on the ground that the baby is able to stand the operation at that time as well as later, that the retraction of the ruptured nerves is less marked and scar tissue less abundant and firm, and that the operation is, therefore, easier. Others believe that the operation should be delayed until the child is a year old, on the ground that it is then possible to know just what the final extent of the paralysis is and just what operation must be done. They also believe that, as the parts are larger at this time, the operation is easier. I, personally, am inclined to agree with those who advocate delay.

Muscle training should be begun as early as possible. It is surprising how early this can be started and how much can be accomplished by it. Apparatus should also be applied, if necessary, to prevent the development of deformities. Much may be done in old, neglected cases, by operations on bones, muscles and tendons.

INTRACRANIAL HEMORRHAGE IN THE NEW-BORN

Intracranial hemorrhages of varying extent are found very frequently at autopsies on new-born infants. Some authors go so far as to say that from 35 to 40% of the infants who die in the first week of life die from intracranial hemorrhage. It seems to me, however, that these figures are too large and that the proportion of deaths in the first week from intracranial hemorrhage is much smaller than this. It also seems to me that the importance of small intracranial hemorrhages found at autopsy is exaggerated, as it is very probable that many babies that do not die have small intracranial hemorrhages without showing any symptoms at the time or in after life.

Etiology.—The causes of intracranial hemorrhage in the new-born may be divided into three classes; trauma, cerebral congestion and disease.

Hemorrhage from trauma usually occurs because in the moulding of the head the bones are forced to ride over each other. When the bones override each other the sinuses may be ruptured or the meninges torn. More often, however, the small veins are torn from the sinuses. This is possible because the veins are very numerous in the new-born, are unsupported between the pia and the sinuses and are very thin-walled. The moulding of the skull and the consequent overriding of the bones is due to the pressure of the mother's parts. Even if the forceps is applied, the pressure exerted by them is little more than that exerted by the mother's parts. Intracranial hemorrhage from trauma in forceps deliveries should not be attributed, therefore, to the forceps. In rare instances, the skull may be fractured by the forceps. The number of cases of intracranial hemorrhage due to trauma is relatively small.

Cerebral congestion is present in all labors. It must be very marked in many. It is due, in large part, to asphyxia. As the result of the cerebral congestion, the intracranial pressure is increased and the thin walls of the capillaries give way. Cerebral congestion is probably the cause of the vast majority of cases of intracranial hemorrhage. When the congestion during labor and the delicacy of the cerebral capillaries are borne in mind, it is most surprising that hemorrhage from the capillaries does not occur in every case.

Intracranial hemorrhage from disease is very seldom due to syphilis. It is sometimes due to toxemia from sepsis or from some unknown cause. Hemorrhagic disease of the new-born accounts for most of the intracranial hemorrhages due to disease. It is considered the most common cause of intracranial hemorrhage by certain authors. It seems to me, however, that they exaggerate its importance. The basis of this opinion is that I have never seen any evidences of intracranial hemorrhage in hemorrhagic disease of the new-born or other evidences of hemorrhagic disease of the new-born when there was an intracranial hemorrhage. When the intracranial hemorrhage is due to hemorrhagic disease of the new-born, there is an increase in the bleeding and coagulation times of the blood. When hemorrhagic disease of the new-born is the cause of the hemorrhage, the symptoms usually do not appear before the second or third day.

Location of the Hemorrhage.—The hemorrhage may occur anywhere in the cranial cavity. It may be cortical, basal, into the ventricles or anywhere throughout the brain substance. The dura being adherent to the skull in the new-born, cortical hemorrhages must be subdural and in the tissues of the arachnoid and pia. When the hemorrhage is due to

trauma, it is most often cortical or basal. When it is due to hemorrhagic disease of the new-born, it may occur anywhere. The old division of these hemorrhages into supratentorial and infratentorial is unimportant.

Symptomatology.—The babies usually appear normal at birth. The symptoms usually develop in the first twenty-four or forty-eight hours, but may be delayed until the fifth day. They appear earlier in the cases due to trauma, because the hemorrhage is usually large at first. The symptoms develop slowly in the cases due to congestion and disease, because the bleeding is usually slower. In general, they appear latest in the cases due to hemorrhagic disease of the new-born.

The first symptoms are usually moaning, crying, restlessness or refusal to nurse. In some instances, edema of the face and hemorrhage from the nose or into the pharynx are early symptoms. More definite symptoms usually soon develop, such as bulging of the anterior fontanelle, spasm or paralysis of one or more of the muscles supplied by the cranial nerves, rigidity or flaccidity of the extremities, twitching, convulsions, stupor and disturbance of the rate of the pulse and respiration.

The symptoms vary markedly in different cases. They are probably due much more to a general secondary increase in the intracranial pressure than to pressure or irritation from the hemorrhage itself. There is, presumably, always a secondary hydrocephalus. Little can be told from the symptoms as to the location of the hemorrhage, because the general symptoms of pressure disguise the localizing symptoms and because of the imperfect development of the infant's brain and central nervous system. It is impossible to locate the hemorrhage from the condition of the reflexes or the evidences of cranial nerve involvement. In a general way, however, convulsions, rigidity and bulging of the fontanelle are more likely to be present if the bleeding is largely above the tentorium and respiratory disturbances, if it is below.

Diagnosis.—Continuous moaning or crying, restlessness, unwillingness to nurse, pallor, stupor or the signs of cerebral irritation should always suggest the possibility of intracranial hemorrhage. Bulging of the fontanelle makes the diagnosis more certain, as do convulsions and twitching. All these symptoms may, however, be due to cerebral edema and most of them may be caused by intestinal toxemia. Prolongation of the bleeding and coagulation times of the blood (see Hemorrhagic Disease of the New-born) makes the diagnosis of intracranial hemorrhage more probable, but does not prove that it is present, because many babies have prolonged bleeding and coagulation times, but do not have cerebral hemorrhage. Lumbar puncture will show an increase in the intracranial pressure. If the cerebrospinal fluid is bloody, and it is certain a vein has not been tapped, the diagnosis is positive. Munro (American Journal Diseases of Children, 1922, **24,** 273) has found that the normal cerebrospinal pressure in new-born infants is about 6 millimeters of mercury and that anything above 10 millimeters is abnormal.

Treatment.—The bleeding and coagulation times of the blood should always be done. If they are prolonged, the treatment for hemorrhagic disease of the new-born should be instituted (see Hemorrhagic Disease of the New-born). Lumbar puncture should be done to diminish the intracranial pressure. According to Munro, if the pressure is above 18 millimeters of mercury, it is also necessary to do a ventricular puncture or a subtemporal decompression. Unless a surgeon, expert in this work, is at hand, and the facilities for testing the cerebrospinal pressure, the practitioner will have to be satisfied with removing the fluid by repeated

lumbar punctures and, if the patient is not doing well, by ventricular puncture. In ventricular puncture the needle should be introduced into the lateral angle of the anterior fontanelle and pushed downward and a little inward and forward. Repeated lumbar punctures may be necessary. Munro recommends introducing the needle a little to one side of the median line, pushing forward, inward and upward, as he thinks that there is less danger of wounding the venus plexuses than when the needle is introduced in the median line. This is a revival of the old method of lumbar puncture which is much more difficult than the introduction of the needle in the median line. If care is taken not to push the needle in too far, there is little danger of bleeding when it is introduced in the median line. The fluid should be allowed to run off until the fontanelle is depressed or, if it was not distended, until the fluid drops only slowly, unless symptoms of cerebral irritation develop before this time.

Prognosis.—It is probable that many cases of intracranial hemorrhage in the new-born, in which the hemorrhage is slight, are never recognized and never cause any symptoms in later life. Many cases in which the condition is recognized or, at least, suspected and which are not treated, if the hemorrhage is small, recover entirely and never show any symptoms later. When the hemorrhage is large and no treatment is instituted, many of the babies fortunately die. A very few may not show any after effects, but most of them will have a spastic paraplegia or diplegia. Many of them will be feeble-minded and some of them epileptic. Epilepsy not uncommonly develops in the cases in which the hemorrhage was small and in which the evidences of paralysis and impaired mentality are slight or absent. The symptoms of paralysis are often not marked enough to be recognized until the baby is some months or one or two years old.

The chances of complete recovery are somewhat better when treatment is instituted, and death is less common. Rather more of the babies do not develop any symptoms in after life, but many of them, although their lives are saved by the treatment, develop paralysis and epilepsy later, or are feeble-minded. The best results are likely to be obtained when the bleeding is due to hemorrhagic disease of the new-born and the condition is recognized early.

VISCERAL HEMORRHAGE IN THE NEW-BORN

Minute and unimportant hemorrhages into the viscera are probably very common in the new-born as the result of the friability of the vessels, the congestion due to asphyxia during labor, and trauma. Hemorrhage into the abdominal organs is especially likely to occur in breech presentations and when attempts at resuscitation by swinging are made. Hemorrhages are more common in the suprarenals than elsewhere; next in the liver or abdominal cavity. Hemorrhage into the suprarenals may be due to compression of the vena cava between the liver and the vertebral column by the contraction of the uterus. If the hemorrhage in the suprarenals is large, it may rupture into the perirenal tissues or peritoneal cavity. Visceral hemorrhages, occurring after birth, may be due to septic infection or hemorrhagic disease of the new-born. They are very seldom due to syphilis.

Symptomatology.—Large hemorrhages at birth, wherever located, cause death in a short time or the baby is born dead. If the hemorrhage does not cause death at birth, a hemorrhage in the lungs gives the physical signs of atelectasis or pneumonia. If it is in the liver or suprarenals there

are no physical signs. If a hemorrhage in a suprarenal is large, it usually perforates and causes death before there is time for the signs of suprarenal insufficiency to develop. If it is small, the symptoms are so slight that they are attributed to something else. If there is a sudden, large hemorrhage into some organ a few days after birth, the symptoms are simply those of acute hemorrhage . If the hemorrhage is smaller, the symptoms are obscured by those of the causative condition and are, therefore, usually not recognized.

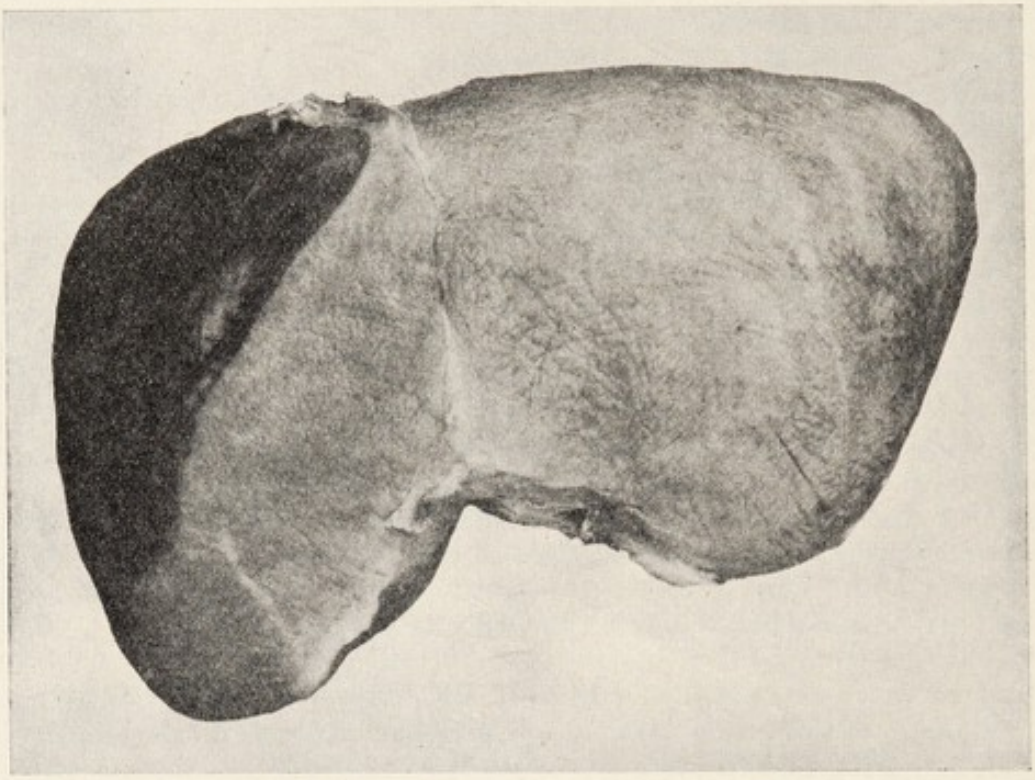

FIG. 31.—Hemorrhage into liver at birth.

Treatment.—As the condition usually cannot be recognized, there is no treatment except preventive and of the causative conditions. It is conceivable, however, that if a hemorrhage into the abdominal cavity was recognized, it might be stopped by an operation.

FRACTURES

It hardly seems necessary to call attention to the fact that the bones of the extremities may be fractured during delivery, or that the skull may be fractured when the forceps is applied. Nevertheless, many physicians neglect to look the new-born baby over for these injuries. The bones most commonly broken are the clavicle, humerus and femur. Fractures of the clavicle are usually greenstick; those of the humerus and femur, complete. When these injuries are properly treated, the results are usually perfect.

SECTION III

DISEASES OF THE NEW-BORN

PHYSIOLOGIC ENGORGEMENT OF THE BREASTS

Swelling of the breasts of new-born babies usually begins on the second or third day of life, but sometimes not until the fifth day. The swelling reaches its height about the tenth day and has usually disappeared in about three weeks. The appearance of the breasts is that of the full, virgin breast. The breasts are not hot, red or tender. The baby suffers no discomfort. The swelling is due to the secretion of milk. The milk sometimes runs out of itself and can always be squeezed out. The appearance, composition and microscopic characteristics of this secretion are much like those of colostrum. The secretion of milk and swelling of the breasts occurs in both boys and girls. It is probably due to the presence in the fetal blood of certain bodies derived from the placenta and chorion which are present in the blood of the mother and which, in her, cause the secretion of milk. This explanation is, however, not entirely satisfactory.

Treatment.—If the breasts are let alone the milk is gradually absorbed, the engorgement disappears and the breasts resume their normal size. If they are bruised or squeezed they often become infected and mastitis results. They should, therefore, be let alone and protected against injury.

MASTITIS

The milk ducts of the infant contain bacteria. The nipples are easily cracked and bacteria are always present on them. The physiologic engorgement of the breasts predisposes to infection. If the engorged breasts are squeezed or injured or the nipple is cracked, bacteria may gain a foothold and cause inflammation and abscess. The appearance and symptomatology of mastitis in the new-born are the same as in adults.

Treatment.—Mastitis seldom develops if the breasts are protected from injury and kept clean. If inflammation appears, cold applications should be tried. If an abscess develops, it should be incised as in the adult. The incision should be made near the periphery in a radius of the gland. It must not be forgotten that the milk ducts may be cut in the infant as well as in the adult and that a careless and unnecessarily long incision in a female infant may cause irreparable damage.

EDEMA NEONATORUM

Etiology.—There is little or no justification for setting apart edema in the new-born as a special disease. Local edema may occur in the new-born from the same causes as in later life. More or less generalized edema may develop in the course of any severe disease, such as septic infection of the new-born, or in any condition accompanied by impairment of the circulation or blood, such as congenital weakness or anemia, or malnutrition. It is almost never due to congenital heart disease and very seldom to disease of the kidneys.

Diagnosis.—The swelling has the characteristics of edema, that is, it is not red, hot or tender, but often pale, is most marked in dependent portions, is usually symmetrical and pits on pressure. It should not be confused with sclerema, in which the swelling is not symmetrical, is hard and does not pit on pressure, and in which the affected portions are rigid. In sclerema the temperature is always much below normal, in edema it may or may not be.

Prognosis.—The prognosis is that of the causative condition.

Treatment.—The treatment is essentially that of the causative condition and symptomatic. When the cause is congenital weakness or debility, it consists in regulation of the diet and surroundings, much as in prematurity. When it is congenital anemia, the treatment is along the same lines as that of anemia in later infancy.

SCLEREMA NEONATORUM

Sclerema is a condition in which there is hardening of the skin and subcutaneous tissues. It may occur in circumscribed areas or extend over the greater part of the body. It occurs most commonly during the first few days or weeks of life, but may develop under suitable conditions at any time during the first year. Although fairly common in Europe, it is very rare in this country. It is due, in all probability, to lowering of the body temperature either as the result of exposure or of feeble circulation. Exposure to cold, neglect, bad hygienic surroundings, congenital feebleness, atelectasis of the lungs and congenital heart disease are the most common causes in the first few weeks of life; later, debility, especially as the result of acute diarrhea. In a number of cases it has developed in connection with an acute general bacterial infection and on this account a number of authors are inclined to consider it an infectious disease. A more reasonable explanation is that in such cases it develops as the result of the lowered vitality caused by the infection.

Pathology.—The induration of the tissues is due to hardening of the subcutaneous fat. This induration is rendered possible by the peculiar composition of the fat in infancy. At this age it contains a much larger proportion of palmitin and stearin and a correspondingly smaller proportion of olein than in later childhood and in adult life, and hence solidifies at a much lower temperature. According to Langer and Knöpfelmacher, the fat in the new-born contains only about 43% of oleic acid compared with 65% in adult life. These proportions change rapidly, however, so that the adult relations are nearly attained by the end of the first year. These figures coincide very closely, therefore, with the age incidence of the disease. This explanation is not entirely satisfactory, however, as sclerema sometimes occurs when the temperature has not fallen to the point necessary for the solidification of the fat. That the fat must be concerned, however, is shown by the fact that fat-free portions of the skin, as the prepuce and scrotum, and those portions of the body, as the palms and soles, in which the proportion of oleic acid is relatively high, are never involved. There are no characteristic lesions in the skin.

Symptomatology.—The first symptom is the induration of the skin, which usually appears in the legs and extends upward. It may, however, appear first in the cheeks. In severe cases it involves practically the whole body. In others it is less extensive or is limited to circumscribed areas. It is most marked where the adipose tissue is most abundant. The indurated areas are hard and firm and do not pit on pressure, feeling, as Northrup says, like a half-frozen cadaver. The skin is usually smooth,

the color normal or somewhat bluish. As the result of the induration of the tissues, the extremities are more or less rigid and the motions limited. The respiration may also be embarrassed by the rigidity of the thorax. The surface of the body is cold and the internal temperature low, the tendency of the temperature being to conform to that of the surrounding atmosphere. On account of the lowered vitality and the feeble circulation, there is usally cyanosis, superficial respiration and feeble pulse and cry.

Diagnosis.—The condition with which sclerema is most likely to be confounded is *edema neonatorum*. In edema the swelling appears first in the eyelids, the dorsa of the feet and dependent portions of the body. The color is pale or waxen, there is pitting on pressure, absence of rigidity and less lowering of the temperature. Cases, intermediate between edema and sclerema or in which both edema and sclerema are present, may occur, however, which are difficult to classify.

Another condition which may be confused with sclerema is the very rare one, known as *scleroderma of the new-born*. In this condition, which is supposed to be due to a fatty necrosis of the subcutaneous fatty tissue associated with inflammation, large or small areas of thickening of the skin and subcutaneous tissues may develop anywhere on the face, body, or extremities during the first few weeks of life. These areas are somewhat reddish or bluish and are not as hard as in sclerema. The infants are, moreover, in good condition and the temperature is not lowered. Recovery is the rule. Atrophy of the skin does not take place as in the scleroderma of adults.

Prognosis.—Death usually comes slowly and quietly in from two to five days. Recovery practically never occurs, except in the milder and limited cases.

Treatment.—The treatment must be directed to the relief of the causative conditions and to the elevation of the temperature. The body heat is best kept up by the same measures that are used in the case of premature infants. In fact, these cases should be treated as regards food, stimulation, handling and care, in practically the same way as premature infants. Massage with olive oil may possibly be of some benefit, provided the infant is not too much weakened by the consequent handling and exposure.

DISEASES OF THE NAVEL

Granulomata of the Navel. Umbilical Polyp

The navel sometimes does not heal properly and there is in consequence a growth of granulation tissue. This may project a little or may be situated deep down in a normally retracted navel. It varies in size from that of the head of a pin to that of a pea. There is a discharge which is more or less purulent, and which is irritating to the skin. When the granuloma is large and has a pedicle, it is known as a polyp.

Diagnosis.—Granulomata of the navel may be confused with a patent Meckel's diverticulum, or mucous polyp, and a patent urachus. The discharge in granuloma is more or less purulent; in patent Meckel's diverticulum, glairy or fecal; in patent urachus, glairy or urine. There is a central canal in a patent Meckel's diverticulum and a patent urachus; none in granuloma. The tumor is small or lacking in a patent urachus and is made up of mucous membrane. It may be quite large in a patent Meckel's diverticulum and is covered with mucous membrane. There is no mucous membrane present in a granuloma.

Treatment.—Granulomata usually dry up and disappear in one or two weeks, if they are kept clean and powdered with aristol or dermatol. They dry up more quickly if they are touched every day or two with a nitrate of silver stick. They may be tied off at the base with a small ligature or snipped off with scissors and the base treated with the nitrate of silver stick or the actual cautery. It is simpler to tie a ligature about a granuloma than to snip it off, and safer, because of the danger of hemorrhage when it is snipped off. It is most important to be sure that the trouble is a granuloma and not a projecting Meckel's diverticulum before a ligature is applied or the tumor cut off.

OMPHALITIS

In this condition there is an inflammation of the navel which usually appears toward the end of the first week of life. The folds of the navel are swollen and there is a purulent discharge. The superficial veins of the abdomen become enlarged and lymphangitis develops. The inflammation may extend to the skin and subcutaneous tissues and result in cellulitis. The inflammation may become definitely erysipelatous or eventuate in abscess or gangrene. Omphalitis is due to infection of the navel as the result of lack of proper care of the navel at birth or afterward. It may develop if the navel is strapped for hernia before it is completely healed. There is more or less fever, according to the severity of the trouble, and all the symptoms of a local infection.

Prognosis.—The prognosis depends on the severity of the local process and on whether the infection spreads inward through the blood vessels or remains localized. If it remains localized, there is a fair chance of recovery.

Treatment.—The most important part of the treatment is to prevent infection by proper care of the navel. If infection has taken place, a 1–5000 solution of corrosive sublimate should be applied. If an abscess develops, it should be opened. General treatment, by regulation of the diet and care, is most important.

INFLAMMATION OF THE UMBILICAL VESSELS

Inflammation of the umbilical vessels is often a sequela of omphalitis, but may be the primary lesion. The arteries are more often involved than the vein. The inflammation of the arteries may extend a short or a long distance from the navel. The arteries may contain pus or septic thrombi. It is self-evident that the conditions are favorable for a general blood infection with all its possible manifestations. Extension to the peritoneum, with a consequent local or general peritonitis is not uncommon. Sometimes pus runs out or may be squeezed out from the navel.

The vein is usually involved all the way to the liver, and the inflammation often extends into the branches in the liver, setting up a suppurative inflammation of the liver. There is sometimes a discharge of pus from the navel, but in both phlebitis and arteritis the local manifestations may be very slight. When present, they are, except for the discharge of pus from deep down, much the same as those of omphalitis. The general symptoms are, of course, those of septic infection.

Prognosis.—Inflammation of the umbilical vessels is always a serious condition. If the infection remains localized, there is a fair chance for recovery. If the infection extends or becomes general, recovery seldom takes place.

Treatment.—The main treatment of this condition is preventive. If the navel is properly taken care of at and after birth, there will be no infection and no occasion for further treatment. If there is infection and it does not extend too deeply, an attempt should be made to give an opportunity for external drainage. Nothing else can be done, except to apply wet corrosive dressings of the strength of 1–5000 and to feed and care for the baby as well as possible.

SEPTIC INFECTION OF THE NEW-BORN

Acute pyogenic infection of the new-born is, perhaps, a more accurate term than the more usual septic infection of the new-born. It is far more common than is generally realized. It may vary from a mild infection with very few and slight symptoms to a very severe infection, running its course in from twenty-four to forty-eight hours. The infection may involve any and all parts and organs of the body.

Etiology.—The infection is, in most instances, caused by the common pyogenic bacteria, usually the staphylococcus aureus and the streptococcus. The most serious infections are usually due to the streptococcus, but may be due to the staphylococcus aureus. Infection is sometimes due to the pneumococcus and sometimes to various other organisms. The gonococcus is, in my experience, not a very common cause.

It is comforting to physicians and nurses to think that infection may take place before birth. This is possible, but probably very rarely happens. Infection may take place during birth, from the mother's secretions, or after birth. It almost always happens after birth. It is not necessarily due to lack of care. Infection occurs most often through the navel, but the portal of entry may be the mouth, anus or skin. Any lesion of any mucous membrane or of the skin affords an opportunity for infection. The wonder is, not that infection occurs as often as it does, but that it does not happen in every baby, when the opportunities for and the susceptibility of babies to infection are considered.

Types of Infection.—Whatever the portal of entry, the manifestations may be either local or general. They may remain localized at the seat of entrance or may later become general. There may be no signs of infection at the portal of entrance, the manifestations being localized somewhere else, or general from the first. Septic infection cannot be ruled out because there is no evidence of a primary focus of infection.

Systemic Infection

When there is a general, systemic infection the symptoms are almost always very marked. The temperature is elevated and irregular. The pulse and respiration are rapid. Loss of weight is rapid and progressive, and all the symptoms of marked malnutrition develop. There is anorexia and usually more or less vomiting and diarrhea. The liver is usually enlarged and the spleen sometimes. There is often jaundice, but usually no bile in the urine. Hemorrhages into the skin are not uncommon. Pustules often develop on the skin and in many cases there is desquamation of the skin. In the most severe cases dry gangrene of the fingers and toes develops. Leucocytosis is usually marked, unless the resistance is overcome, when it is absent. Death occurs in from one to four or five days. The symptoms of systemic infection are the same when the general infection is secondary to a local infection. In some cases the infection is less severe and the symptoms are limited to fever and dis-

turbances of nutrition and digestion with, perhaps, a few pustules on the skin. Recovery is very rare, if there is jaundice or hemorrhages. Pathologically, there are granular and fatty degeneration of all the organs and the evidences of hemorrhages.

Diagnosis.—Mild cases, with only slight constitutional symptoms and no local symptoms, may be mistaken for inanition or dehydration fever. The ingestion of fluid causes the symptoms to disappear in dehydration fever. It has no effect in sepsis. In sepsis, moreover, local symptoms develop or the general symptoms increase. The only other disease in which jaundice is associated with fever at this age is catarrhal jaundice. This is very uncommon, is never associated with hemorrhage, the baby is not severely ill and there is no bile in the stools. Hemorrhage may suggest hemorrhagic disease of the new-born. While there is some fever in this condition, there is never jaundice and there are no symptoms outside of the temperature, the hemorrhages and the results of the hemorrhages.

Prognosis.—The outlook in all, except mild cases, is practically hopeless. Cases with jaundice and hemorrhage seldom recover.

Treatment.—The treatment must be entirely symptomatic, unless there is a focus of infection which can be removed. Breast milk is the best food; water should be pushed; stimulation used as necessary. Vaccines are useless.

WINCKEL'S DISEASE OR EPIDEMIC HEMOGLOBINEMIA OF THE NEW-BORN AND BUHL'S DISEASE OR ACUTE FATTY DEGENERATION OF THE NEW-BORN

These conditions are, in all probability, simply varieties of septic infection of the new-born and ought to be dropped as special entities. The chief peculiarities of Winckel's disease are deep jaundice, marked cyanosis, hemoglobinemia and hemoglobinuria, and that it usually occurs in epidemics in institutions. The temperature is high and death occurs in a few days.

In Buhl's disease the babies are asphyxiated and cyanotic at birth. The cyanosis continues, and jaundice and hemorrhages develop. The temperature is low and death is almost certain. Pathologically, there is fatty degeneration of all the parenchymatous organs.

Local Infections

The manifestations of a local infection may be in the neighborhood of the portal of entry, as in omphalitis, inflammation of the umbilical vessels, localized abscesses on the surface of the body and stomatitis, or in organs at a distance. The organs most commonly involved are the lungs, liver, peritoneum and bones. The meninges and pericardium are also sometimes affected.

Infections of the Lungs.—The most common manifestation in the lungs is a purulent bronchitis which usually develops into a diffuse bronchopneumonia. There is nothing characteristic about the physical signs, which are not different from those in bronchitis and bronchopneumonia from other causes. They may be very slight, however, and, in general, the physical signs are not as marked as they would be expected to be from the fever and the severity of the symptoms of infection. In some instances, the lesions are largely limited to one lobe and the physical signs are, therefore, those of lobar pneumonia.

Diagnosis.—In the absence of a lesion at the portal of entry, the diagnosis of septic infection of the lungs from an ordinary bronchitis,

bronchopneumonia or pneumonia must rest on the temperature being higher and the constitutional symptoms more severe than would be expected from the physical signs in the lungs.

Prognosis and Treatment.—The outlook is always very grave and few recover. The treatment is entirely symptomatic and supportive.

Infections of the Liver.—The liver is usually involved secondarily as the result of an extension of the infection from the umbilical vessels. If the baby lives long enough, multiple small abscesses are formed, sometimes a single large one. In rare instances there may be abscesses in the liver when the portal of entry is not at the navel. It is impossible to recognize small abscesses of the liver, and even a large one is almost certain to be missed, unless it is on the surface in a place where it can be palpated. Enlargement of the liver when there is inflammation of the navel and umbilical vesels is suggestive of infection of the liver, but not proof, because in septic infection of the new-born the liver is almost always enlarged as the result of fatty change.

Treatment.—There is, of course, no possibility of surgical interference, unless the abscess is single. Even then, the baby's condition will probably be such as to make an operation inadvisable or fatal.

Infections of the Peritoneum.—Infection of the peritoneum is most common when there is infection in or about the navel or in the umbilical vessels. It may occur, however, when there are no evidences of infection at the navel or elsewhere. There may be a general purulent peritonitis, a localized peritonitis with adhesions, or walled-off abscesses. Localized peritonitis with adhesions is very difficult to recognize, but should be suspected if there is marked, localized tenderness in the abdomen in association with lesions at or about the navel. The signs of general purulent peritonitis are the classical signs of this condition at any age but, as always in infancy, are extremely hard to recognize. Rigidity of the abdomen, constant flexion of the thighs on the abdomen and putting the hands on the abdomen when it is touched are the most important signs at this age. Localized abscesses are even harder to recognize but, when a new-born baby shows the symptoms of a collection of pus, there are no evidences of an abscess elsewhere and more or less marked abdominal symptoms, there is, in all probability, a localized abscess in the peritoneum. This is the more probable, if there is or has been inflammation about the navel. The most common locations are in the region of the navel, deep in the pelvis or behind the stomach.

Prognosis and Treatment.—Localized peritonitis with adhesions may subside. It is perhaps helped somewhat by local applications of heat or cold. General purulent peritonitis is almost always fatal, whether operated upon or not. There is some chance of recovery, if a localized abscess is opened and drained.

Infections of the Bones and Joints.—Infection almost always takes place in the diaphysis, but sometimes in an epiphysis. It then extends, with or without separation of the epiphysis, to the joint. The most marked physical signs are usually in the joints. The infection is usually multiple. Rheumatism, tuberculosis of joints and scurvy do not occur at this age. Syphilitic epiphysitis seldom appears as early and, if it does, does not show the symptoms of acute septic infection and is almost always accompanied by other manifestations of syphilis.

Prognosis and Treatment.—The prognosis is better than in most of the other forms of local infection. The treatment is surgical.

Infections of the Meninges.—The meninges are seldom infected. If they are, any or all of the physical signs of meningeal infection may be present, but they are quite likely to be obscured by the general evidences of septic infection and the meningeal condition overlooked. The cerebrospinal fluid is turbid or purulent, contains large numbers of polynuclear cells and many of the causative organisms. The condition is hopeless and there is no treatment.

PEMPHIGUS NEONATORUM

This is the term applied to *impetigo contagiosa* in new-born infants. It is almost always caused by the staphylococcus aureus but may be by the staphylococcus albus or the streptococcus. It is contagious and most commonly occurs in epidemics in institutions. It may be spread by physicians, nurses and midwives. It is sometimes simply one of the manifestations of a general septic infection. The eruption is not present at birth, but appears in from four or five days to three weeks after birth.

The lesion is a bulla, which is usually situated on a red base. The bullae are at first seldom larger than a split pea or a dime, but in severe cases may be much larger and involve considerable areas. The palms and soles are never affected in the beginning and, if they are later, the lesions involving them are very large. The blebs contain serum at first, but the contents may become more or less purulent. The smaller lesions either dry up and form crusts or rupture and become crusted with secretions. The larger bullae rupture and the epidermis comes off, leaving a moist, red, denuded surface. In such cases the condition is essentially the same as in a superficial burn.

When the lesions are few and small, there are practically no constitutional symptoms. When they are numerous and large, the infant is much depressed, just as it is with a large burn. The temperature is then often subnormal. If the baby lives long enough for suppuration to take place, it shows the constitutional symptoms common to such a condition. Secondary infections often take place in severe cases and add their symptoms to the picture.

Diagnosis.—There is a bullous form of syphilitic eruption, also known as pemphigus, which is present at birth or appears within a few days after birth. There are always lesions on the palms and soles, whether there are any elsewhere or not, while in pemphigus neonatorum there are seldom any lesions on the palms and soles and, if there are, they appear late in the disease and are very large. In syphilitic pemphigus the infection is always a severe one and consequently the nutrition is much impaired and there are always other manifestations of syphilis, such as snuffles, mucous patches and enlargement of the liver and spleen. In pemphigus neonatorum, however, the nutrition is, at any rate in the beginning, good and there are no other evidences of syphilis.

Prognosis.—The prognosis, both as to duration and outcome, depends entirely on the severity of the infection in the given instance. The disease may be only trifling and run from a few days to a few weeks or it may be very serious and terminate fatally in one or two days. Unfortunately, mild cases may change suddenly to the severe type. A baby with this disease cannot be considered out of danger, therefore, until it is well.

Treatment.—The most important treatment, as in so many of the diseases of the new-born, is the preventive. It will never occur, if everything about the baby and its attendants is clean. The second case

in a short time in an institution or in the practice of a physician or nurse means carelessness somewhere, the source of which must be found and eliminated.

The first element in treatment, when the disease has developed, is cleanliness. The baby's clothes must be sterilized as long as the eruption persists. Great care must be taken not to spread the infection in bathing. The skin about the lesions should be washed with a 1–5000 solution of corrosive sublimate or with 95% alcohol. The bullae should be broken and, if not too large, dressed with the officinal ammoniated mercury ointment. If large surfaces are denuded, they should be treated as any large superficial burn, that is, either with some oily dressing or exposed to the air without dressing of any sort. It goes without saying that breast-milk should be given, if possible, and the greatest attention paid to every detail of the care and nursing.

DERMATITIS EXFOLIATIVA. RITTER'S DISEASE

This disease, like pemphigus neonatorum, is due to an infection of the skin, usually with the staphylococcus aureus. There is at first redness about the mouth, which soon extends to the face. There are also fissures about the mouth, which are quite characteristic. Soon after the redness appears on the face, it extends to the trunk and extremities and finally involves the whole surface of the body. The skin becomes everywhere hyperemic, swollen, thickened and scaly. After a time the epidermis comes off in large flakes, leaving the red corium exposed. There is usually more or less exudation of serum from the denuded corium. The constitutional symptoms are usually marked, being a combination of those due to sepsis and those resulting from the denudation of large areas of the body. Death occurs in a few days in most instances. Occasionally, however, a baby recovers.

Treatment.—The treatment for the skin is like that for an extensive superficial burn, either by oily applications or by exposure to the air. The latter method is probably preferable. Breast-milk should be given, if possible, and every attention paid to the details of the care and nursing.

ERYSIPELAS NEONATORUM

Erysipelas of the new-born may, perhaps, be regarded as simply another manifestation of septic infection of the new-born. Even so, as it is usually not associated with any other evidences of infection, it may be considered as if it was a separate entity. There is always, of course, some lesion of the skin which serves as the portal of entry. This is most often at the navel, about the external genitals or on the face. The characteristics of the erysipelatous infection are the same as at other ages. The skin is thickened, indurated, rosy red in color, shiny, hot and somewhat tender. The edges of the indurated area are distinct and elevated above the normal skin. The edematous swelling may be considerable, but the tendency to the formation of blebs and abscesses is less marked than in later life. The most striking thing about erysipelas in the infant is its tendency to continue to spread from one part to another, the so-called *erysipelas migrans*. I have seen it start on the face and gradually run down to the toes and then run back again to the head. The symptoms are those of any septic infection; high and often irregular temperature, anorexia, disturbance of the digestion, rapid loss of weight and leucocytosis.

Diagnosis.—Erysipelas is easy to recognize at any age, if it and its characteristics are borne in mind. In *erythema neonatorum* the reddening of the skin is usually more diffuse, the skin is not thickened and the edges of the reddened area are not elevated. In *cellulitis* the lesion evidently extends more deeply and the edges of the swelling are usually not as much elevated and are less sharply defined.

Prognosis.—Erysipelas is a very serious disease in the new-born and also throughout infancy. At least 90% of young infants with erysipelas die, chiefly because of its tendency, at this age, to spread from one part of the body to another. If they are able to withstand the disease at first, their resistance is finally overcome and they succumb. Complications in the heart or kidneys are unusual at this age.

Treatment.—There is no local treatment which has much, if any, effect on the progress of the disease. Ichthyol may perhaps be more efficient, or less inefficient, than the others. A 30% ointment of ichthyol, made up with vaseline, may be applied to the affected areas every three or four hours. Iced compresses are often very comforting to older children, but, if used with babies, great care must be taken not to chill them. A saturated solution of camphor in ether, painted on with a brush, also sometimes makes children more comfortable.

There is no drug which influences in any way the course of erysipelas. It is possible that in the more chronic cases, streptococcus vaccines, especially if autogenous, may do some good, but I have myself never seen any benefit from their use. The most important parts of the treatment are the feeding and general care of the baby. Breast-milk is most important for it. If the lesion is on the face, it cannot be put to the breast because of the danger of infecting the mother's breast. If the lesion is somewhere else, it may be put to the breast, if the most minute precautions are taken against contagion. In general, however, it is wiser to get the milk with a pump or by expression and feed it to the baby in a bottle. The best substitute for breast-milk is modified milk, preferably a whey mixture.

INTESTINAL TOXEMIA OF THE NEW-BORN

I am in the habit of applying this name to a condition with the following symptom-complex. It seems to me to be a rather important condition, but, as no one has ever paid any attention to a paper which I published about it in 1912, very likely it is not as important as I think.

Symptomatology.—The babies are normal at birth and continue to do well up to the second, third, fourth or even fifth day, when they become rather suddenly ill. They are likely to cry and moan a good deal, although not infrequently they are unusually quiet. Attacks of cyanosis are a common and early symptom. Twitching of the extremities, slight, general rigidity and retraction of the head are not uncommon, while convulsions occur occasionally. The temperature is usually only moderately elevated, but may be high. Vomiting is uncommon, as is diarrhoea, constipation being the rule. In most instances the symptoms develop before the baby has ceased to pass meconium and it is usually found that it has not passed as much as the average baby. If the stools are not composed entirely of meconium, they are usually small in amount, loose, dark brown and contain small curds and mucus. They are often offensive. The abdomen may be distended, but usually is not. Loss of weight is rapid, the face becomes pinched and, in all but the mildest

cases, it is evident that the baby is seriously ill. If the bowels are thoroughly cleaned out, all food stopped and water given freely, recovery is usually rapid and complete. If the bowels are not cleaned out and food is continued, a fatal termination is not uncommon.

Etiology.—My interpretation of the etiology of these cases is that a bacterial infection of the meconium through either the mouth or anus takes place within the first twenty-four or forty-eight hours after birth and that, on account of the incomplete evacuation of the intestines, the toxic products formed in the meconium as the result of this infection are absorbed into the circulation and that these toxic products cause the symptoms. Corroborative evidence in favor of this conception is that as the meconium is made up of proteins, the products of bacterial action in it must necessarily be putrefactive in character and, therefore, toxic. The early onset of the symptoms, the absence of any nidus of infection and the absence of other signs of sepsis, together with the rapid and complete recovery after the evacuation of the bowels, seem sufficient to exclude an ordinary septic infection. The symptoms are evidently not due to starvation or dehydration, because they occur both in babies that have been fed and in those that have not and because recovery takes place when the bowels are evacuated, even though food is stopped.

Treatment.—The treatment consists in the administration of one or two teaspoonfuls of castor oil, the withdrawal of food for from twelve to twenty-four hours and the feeding of water or water sweetened with saccharin. It is also well to irrigate the bowels in the beginning. Bromides or stimulants may be used, if necessary.

The best food, after the period of starvation, is human milk, either plain or diluted, according to the condition of the baby. If this cannot be obtained, a mixture of cow's milk, low in fat, high in milk sugar and with a moderate amount of proteins, part of them preferably in the form of the whey proteins, is best. It is important to give a high percentage of milk sugar in order to change the bacterial flora from the proteolytic to the fermentative type.

Diagnosis.—The diseases for which this condition is most likely to be mistaken are intracranial hemorrhage, meningitis, septic infection of the new-born and dehydration fever. In intestinal toxemia the symptoms usually appear earlier than in septic infection and the temperature is usually lower. There is no local nidus of infection. The marked general and local symptoms of infection, such as hemorrhages, jaundice and furuncles, are absent. There is a tendency to constipation and the stools are usually meconium-like in character. In many instances, however, a positive diagnosis can only be made after the bowels have been thoroughly moved.

Meningitis is extremely rare at this age and when it occurs is usually part of a general septic infection. There is usually bulging of the anterior fontanelle in meningitis; never in intestinal toxemia. The symptoms in meningitis are not relieved by catharsis. Lumbar puncture will settle the diagnosis in doubtful cases.

The symptoms of intracranial hemorrhage in the new-born may be very similar to those of intestinal toxemia. Moaning, crying and refusal to nurse are more prominent symptoms in intracranial hemorrhage than in intestinal toxemia. Edema of the face and hemorrhages from the nose or into the pharynx never occur in intestinal toxemia nor does bulging of the anterior fontanelle. Symptoms of cerebral irritation, such as twitching and convulsions, may occur in both. The symptoms

in intracranial hemorrhage are not relieved by catharsis. In some cases the diagnosis can only be made by lumbar puncture.

In dehydration fever the babies are hungry or thirsty and the symptoms of dehydration, such as loss of weight and dryness of the skin are usually marked. Symptoms of intoxication and meningeal irritation occur later, as a rule, than they do in intestinal toxemia. The symptoms are exaggerated by catharsis in dehydration fever, while they are relieved by catharsis in intestinal toxemia of the new-born.

(This chapter is copied, in most part, from my paper on intestinal toxemia in the new-born published in the American Journal of the Diseases of Children, 1912, **4,** 229.)

ICTERUS NEONATORUM

Icterus neonatorum is often known as physiologic or idiopathic icterus. It may properly be regarded as a physiologic condition, but in severe cases it is on the borderline of the pathologic. According to various statistics, it occurs in from forty to eighty per cent of all new-born infants. It is most common in premature and feeble infants and is also most severe in them. It is equally common in boys and girls.

Symptomatology.—Jaundice is not present at birth. It may develop on the first day and seldom develops after the seventh day. In 75% it appears on the third, fourth or fifth day. It appears first on the face and chest, then in the conjunctivae, and finally spreads over the whole body. The color of the skin varies from a pale to a deep yellow. The jaundice lasts three or four days in mild cases, from seven to ten days in moderate cases, and several weeks in the severe cases.

The general condition is little, if at all, disturbed. There is no fever. The stools are normal and contain bile. There is almost never any bile in the urine. The liver and spleen are not enlarged. There are no hemorrhages and seldom any evidences of itching.

Etiology.—Although many theories as to the etiology of icterus neonatorum have been advanced, it is safe to say that even now the real cause is unknown. Hess's explanation (American Journal Diseases of Children 1916, **11,** 294) that there is a sudden increase in the secretion of bile soon after birth and that the excretory powers of the liver are not, at first, equal to the secretory seems as reasonable as any and more probable than most.

Diagnosis.—If a new-born baby has jaundice the chances are that the trouble is icterus neonatorum, because jaundice is more often due to this cause than to all others combined. Jaundice may, however, be due to a number of other causes. In giving the differential diagnosis between icterus neonatorum and these other conditions, I will give only the points of difference. In *septic infection of the new-born* the baby is sick, has a fever, hemorrhages are common, the liver is enlarged and often the spleen and there are other evidences of sepsis. In *congenital malformation of the bile ducts* the liver and spleen are enlarged, there is bile in the urine, the stools, at first white or composed of meconium, are later always white, jaundice is often present at birth and steadily increases, hemorrhages are not uncommon. In *catarrhal jaundice* the baby is somewhat sick, has some elevation of the temperature, the liver is often slightly enlarged, there is bile in the urine and the stools are white or gray. In *congenital syphilis* the nutrition is poor, the liver and spleen are much enlarged and there are other marked signs of syphilis. In *congenital family* or *acholuric*

jaundice the jaundice is present in other members of the family, the spleen is much enlarged and the liver somewhat enlarged. In another set of cases, which is very rare, the jaundice has been found to be associated with an interstitial hepatitis with enlargement of the spleen. This hepatitis may or may not be syphilitic. Babies with this condition may or may not recover. Holt and others have described cases of this sort and I have myself seen several of them.

Prognosis.—Icterus neonatorum does not affect the general condition or interfere with growth. The jaundice disappears in a few days or weeks, according to its severity.

Treatment.—No treatment is necessary. Icterus neonatorum is a physiological condition and is probably due to the mechanical difficulty which the bile encounters in passing through the bile capillaries. There is, therefore, no object in giving cathartics. Cleaning out the intestine cannot affect the conditions in the bile capillaries. None of the so-called cholagogues, including calomel, increase the flow of bile. If they did, they would cause harm in this condition. There is no indication for changing the food, because sufficient bile to carry on the processes of digestion enters the intestine, only the excess passing into the circulation.

ERYTHEMA NEONATORUM

The skin of the new-born infant is normally redder than it is after a few weeks. It is very sensitive to irritation and, if irritated, the condition which is known as erythema neonatorum sometimes develops. In this condition the skin is everywhere uniformly reddened. The redness appears in the first two or three days of life. It usually fades away in about a week, but may persist somewhat longer. As it fades, there is quite likely to be a profuse, branny desquamation. There are no constitutional symptoms, but in some instances there seems to be itching.

Diagnosis.—The chief importance of erythema neonatorum is that it may be mistaken for other more serious conditions. Among these are dermatitis exfoliativa, erysipelas and scarlet fever. In all of these conditions, however, the baby is definitely sick and has an elevation of temperature, while in erythema neonatorum there are no constitutional symptoms and the temperature is normal. In dermatitis exfoliativa the epidermis comes off in large flakes disclosing the denuded, oozing corium, while in erythema neonatorum there is a branny desquamation, leaving a normal skin. The redness in erysipelas is always localized and the area of redness is raised, indurated and has a definite border, while in erythema neonatorum the redness is general, the skin is not indurated and there is no border. Scarlet fever is very uncommon at this age. The rashes are very similar, but in scarlet fever the tongue and throat are involved, while they never are in erythema neonatorum. The chief danger is that a mild case of scarlet fever may be mistaken for erythema neonatorum and, consequently, the proper precautions not taken.

Treatment.—When babies have erythema neonatorum it is important to be careful while bathing them not to have the water too hot or to rub them too vigorously. No soap should be used on the skin and the skin should be thoroughly and carefully dried. A dusting powder should then be applied over the whole body. A very good one is:

Pulv. zinci oxidi	℥ii
Pulv. amyli ad	℥i

Stearate of zinc powder is also very good.

DEHYDRATION FEVER OF THE NEW-BORN. INANITION FEVER

Many babies have a rise of temperature between the second and fifth days of life without any other evidences of disease. It ordinarily does not go above 102° F., but may reach 105° F. The babies are restless and uncomfortable and not infrequently act hungry or thirsty. There is considerable loss of weight. The skin becomes dry and inelastic. The urine is often scanty. In severe cases the baby becomes much prostrated and may develop symptoms of meningeal irritation. When the breast-milk comes in or the baby is given a sufficient amount of an artificial food, the temperature falls, often very rapidly, and the baby quickly becomes normal. If the breast-milk does not come in and the baby is not given sufficient food and water, it may die.

Etiology.—It has been supposed for a long time that this rise in temperature was due to starvation, the reasons being that it occurs at the time when the supply of breast-milk is insufficient and when loss of weight is marked, and that the temperature returns to normal when the supply of food is sufficient. Hence, it has been called inanition fever. This explanation is not entirely satisfactory because the height of the temperature does not always coincide with the amount of the loss of weight, and babies that have no temperature often lose as much or more weight than those that do.

A more reasonable explanation is that the fever is due to the loss of water. It is known that fever may be produced experimentally in animals by dehydration. The rise in temperature is due to a diminution in the plasma water, which is equivalent to a concentration of the plasma or an increase in the serum protein per cent. Bakwin has found in a considerable series of cases that when there is fever without other evidences of disease, the plasma concentration is increased and that when babies have fever from infection or other causes, the plasma concentration is not increased. He found the plasma concentration increased, even when the loss of weight was not marked. He calls attention to the fact that the water content of babies varies much at birth and that one baby with a large reserve of water may lose a considerable amount of water and, therefore, of weight, without being dehydrated and having an increase in the plasma concentration, while another baby, with a small reserve of water, will become dehydrated and have an increase in the plasma concentration after a small loss of water and of weight. He also found that even the impairment of the elasticity of the skin did not always coincide with the plasma concentration. Finally, it has been found by him and others that the administration of water alone, without food, will bring down the temperature. It seems evident, therefore, that the rise in temperature is due to dehydration rather than to starvation.

Diagnosis.—The temperature of all new-born babies should be taken regularly during the first week, so that, if there is a rise in temperature, it will be known at once. If it is due to dehydration, there will be no other evidences of disease. Other common causes of temperature at this time are septic infection and intestinal toxemia of the new-born. The temperature in septic infection, as a rule, does not begin before the fourth or fifth day and there are other evidences of infection. In intestinal toxemia the stools are scanty and composed of meconium, the baby is manifestly toxic and the symptoms are relieved when the intestines are emptied. A new-born baby may, of course, have many diseases attended with fever, but with all of them it will be manifestly ill and show the symptoms or physical signs of some definite ailment.

Treatment.—The treatment is primarily preventive. New-born babies should be watched more carefully than they sometimes are to be sure that they are getting enough milk from the breasts. If they are not, they should be given either an artificial food or water, as the case may be. If the temperature rises, they should be given water or water sweetened with saccharin. As there is no vomiting or diarrhoea and the dehydration is seldom very severe, it is almost always sufficient to give the water by mouth. Eight to twelve ounces in twenty-four hours is usually sufficient. In very severe cases, which have been neglected, physiologic salt solution may be given subcutaneously or intraperitoneally. From two to four ounces may be given under the skin, the injection being repeated as soon as it is absorbed. From four to six ounces may be given intraperitoneally. It is almost never necessary to use this route, however, and it should not be used again in less than twenty-four hours.

HEMORRHAGIC DISEASE OF THE NEW-BORN

The chief characteristic of this condition, which is often and perhaps better called spontaneous hemorrhage of the new-born, is that hemorrhages occur spontaneously, without injury or obvious cause, from the mucous membranes and into the skin and organs of the body. In uncomplicated cases there are no symptoms, except those due to hemorrhage, and no pathological lesions, except those resulting from hemorrhage. Hemorrhagic disease of the new-born is equally common in males and females. It is a self-limited disease. There is never a recurrence or a tendency to bleeding in later life.

Symptomatology.—The first symptom is spontaneous hemorrhage. It sometimes appears on the first day, usually on the second or third day, almost never after the seventh day and never after two weeks. The bleeding may begin in the skin or be from the cord or from any of the mucous membranes. It is quite likely to occur where there was some abrasion of the skin at birth. When a place once begins to bleed, it continues to ooze. It never bleeds copiously, but keeps on oozing. The bleeding almost always occurs from several places. Under the old methods of treatment which had no effect, except sometimes to stop the bleeding, if it was on the surface of the body where it could be reached, from 60 to 80% of the babies died. One-half of those that died, died in the first day and all inside of a week. The bleeding stopped in two- thirds of the babies that recovered in five days or less and never lasted over nine days. If the hemorrhage is from the gastric mucous membrane, the blood may be vomited or passed in the stools. If vomited, it is almost always dark brown, having been changed by the hydrochloric acid of the stomach. It is possible that no blood will be vomited, even when the hemorrhage is from the stomach, and that the baby may die before blood has been passed in the stools. It is also possible that the blood which is vomited may have been swallowed. Blood passed from the bowels, whether it came from the gastric or the intestinal mucous membrane, is always dark brown in color. It is usually mixed with the stool, but in any case is not in the form of clots. It is sometimes difficult to tell whether the stool is composed of blood or meconium. If there is blood, the napkin is usually stained or, if the stool is mixed with water, the water will be bloody. It is seldom necessary to examine the stool microscopically or to try any of the tests for occult blood. If there is not enough blood to be recognized by the naked eye, the bleeding is usually not serious. Hemorrhagic disease of the new-born may be a cause of

intracranial hemorrhage, but is, I think, much less often so than many recent writers believe. Hemorrhages into the internal organs are seldom large enough to cause either physical signs or special symptoms. I have never seen hematuria in this condition, although it is described.

If the loss of blood is considerable, the infant becomes pale and weak. It loses weight and the skin becomes dry. If the loss of blood is still greater, the respiration becomes rapid and sighing, the pulse rapid and feeble, and finally all the symptoms of severe hemorrhage develop, ending in death. The temperature, at first normal, becomes subnormal as the loss of blood increases. It is not elevated, unless there is some complication.

Etiology.—There is a definite prolongation of the coagulation time during the first four or five days of life, with a gradual reduction to the normal by the tenth day. That is, the normal condition in the blood in the new-born is favorable to the development of the hemorrhagic disease of the new-born. If the normally prolonged coagulation time is exaggerated, the conditions are even more favorable for the development of spontaneous hemorrhage. Hemorrhage develops, however, in only a small proportion of the cases in which the coagulation time is prolonged, even if it is much prolonged. Furthermore, hemorrhage sometimes develops in babies in whom the coagulation time is not prolonged. There is much difference of opinion as to whether the bleeding time is prolonged or not, and, if it is, whether it plays any part in the etiology of spontaneous hemorrhage in the new-born. If it does, it is probably not the primary factor, but merely secondary or contributory.

There is a definite diminution in the prothrombin of the blood during the period in which the coagulation time is prolonged, and when the prothrombin reaches the normal level the coagulation time becomes normal and the tendency to hemorrhage ceases. The calcium content of the blood of the new-born is greater than that of older children, the blood platelets are normal in number, there is no defect in the retractility of the clot and no fibrinolysis. These are exactly the conditions present in the blood in hemophilia, which is due to a qualitative defect in the platelets. It seems probable, therefore, that spontaneous hemorrhage in the new-born is due to a deficiency of prothrombin, which is, in turn, due to a qualitative defect in the blood platelets. It is evident that the blood platelets soon become normal and contain a normal amount of prothrombin. Why the platelets are deficient in prothrombin at birth and why they cease to be deficient after a few days is unknown.

Most of the methods for the determination of the coagulation time are too complicated and require too much apparatus to be of use to the general practitioner. Rodda's method (American Journal Diseases of Children, 1920, **19,** 269) is the simplest and is quite accurate. His method requires a spring lance (a simple scalpel will suffice), two one and one-half inch watch glasses and No. 6 lead shot. The glasses and shot should be cleaned, preferably by washing with soap and water, followed by ether. The lance should be cleaned and sterilized. The infant's heel is sponged with ether. The puncture is made, with the lance blade set (about 0.5 cm.) to produce a free flow of blood without the slightest pressure. A clean watch glass containing a No. 6 shot receives the second drop of blood. A second watch glass is inverted over the first. The watch glasses are gently tilted every thirty seconds until the shot no longer rolls, but is fixed in the clot. The end result is sharply defined. The shot is firmly imbedded so that the glass can be inverted without

dislodgement of the shot. The time is reckoned from the moment that the first drop falls. The time consumed in the formation of the second drop is considered a part of the coagulation time. Determinations are made to the nearest half minute. The average coagulation time in the new-born by this method is seven minutes. A time of more than ten minutes represents delayed coagulation.

Another method, recommended by Larrabee (Boston Medical & Surgical Journal, 1920, **183,** 151) is quite simple, except that the blood must be obtained by venipuncture. Two c.cm. of blood are taken from a vein, put into a test-tube having a diameter of 1 cm., both the syringe and tube having been previously rinsed out with normal salt solution. The tube is allowed to stand upright. Every minute it is turned sideways. When it can be inverted without spilling, the clotting is considered to be complete. The normal time is from four to eight minutes. Anything over twelve minutes is decidedly abnormal.

The simplest way to determine the bleeding time is that recommended by Duke (Journal American Medical Association, 1910, **55,** 1185). A small cut is made in the lobe of the ear. At one-half minute intervals the blood is blotted up on absorbent paper. This gives a series of blots of gradually decreasing size. Each blot represents one-half minute's outflow of blood. The rate of decrease in the size of the blots shows the rate of decrease of the hemorrhage. The cut should be made of such a size that the first half minute's outflow of blood makes a blot of 1 or 2 cm. in diameter. The total duration of such a hemorrhage is called the bleeding time. The normal bleeding time varies from one to three minutes. Larrabee says that anything over eight minutes is distinctly abnormal. The bleeding time is independent of the coagulation time.

Diagnosis.—Hemorrhagic disease of the new-born is not hemophilia. Hemophilia is a family disease, occurs only in males and the tendency to bleeding persists throughout life. Hemorrhagic disease of the new-born is a sporadic disease, occurs in males and females alike, is a temporary condition and there is no tendency to bleeding in later life. Moreover, the tendency to bleeding in hemophilia is not present at birth, but develops in childhood.

New-born babies may have hemorrhages from sepsis or congenital syphilis. The hemorrhages in these conditions are not severe. In sepsis they are associated with other symptoms of infection, jaundice, fever and the evidences of local infection. The hemorrhages are due to some abnormality in the antithrombin factor. In syphilis the nutrition is poor and there are other signs of syphilis, such as snuffles, pemphigus, jaundice and enlargement of the liver and spleen. In syphilis, moreover, the hemorrhage is most likely to be nasal.

Hemorrhage from the stomach, due to the swallowing of blood from cracks in the nipple of the mother, is often mistaken for hemorrhagic disease of the new-born. If this is the cause, hemetemesis will be the only evidence of hemorrhage. The baby will seem otherwise well. A careful search will disclose the lesion of the nipple. If a baby vomits blood the mother's nipples should, therefore, always be examined to be sure that there is no lesion there before the diagnosis of hemorrhagic disease of the new-born is made.

Bleeding from the vagina is not uncommon in new-born female infants. It is the result of the intense congestion of the parts at birth. If there is no hemorrhage elsewhere, *vaginal hemorrhage* is, therefore,

probably not a symptom of hemorrhagic disease of the new-born. It does not mean menstruation nor is it a sign of precocious maturity.

Treatment.—It has been suggested that the coagulation and bleeding times ought to be determined in all new-born infants and that, when one or both is prolonged, treatment instituted. This procedure seems hardly necessary, however, when it is remembered that the coagulation time is normally prolonged, that the line between normal and abnormal prolongation is indefinite, that only a small percentage of the infants in whom it is supposed to be abnormally prolonged have spontaneous hemorrhage and that treatment, after hemorrhage has appeared, is almost invariably successful.

The methods of treatment, in order of efficiency, are the injection of fresh animal or human blood serum or of whole human blood, and the transfusion of whole human blood. Animal serum is not as good as human. Their action is due to the prothrombin which they contain. This disappears in a few hours. They are useless, therefore, unless they are fresh. Old serums, such as diphtheria antitoxin, are worse than useless as they may do harm in other ways. The dose is one or two ounces into the tissues, preferably of the buttocks. When whole blood is used, it should be obtained by venipuncture and immediately injected into the buttocks. One or two ounces is sufficient. It is unnecessary to consider compatibility when either serum or whole blood is injected into the tissues. In the majority of instances, the injection of fresh serum or whole blood will stop the hemorrhage. If it does not, the injection may be repeated in a few hours or a transfusion done. A single transfusion almost invariably stops the hemorrhage at once. Transfusion is, therefore, preferable. Many authors say that a transfusion should always be done and that it is not justifiable to inject blood, because of the possibility that the hemorrhage may recur or that it may suddenly increase or appear in some other place, if the injection is not efficient. The injection of whole blood is much quicker and simpler than transfusion, however, and can be done by many physicians who are not competent to do a transfusion. Unless someone accustomed to doing transfusions in infants is at hand, therefore, it is ordinarily safe to trust to the injection of whole blood, provided that the baby has not already lost a large amount of blood, which must be returned to it.

Direct transfusion is a very difficult operation with babies and is not necessary. Indirect transfusion is best performed with paraffined tubes, although if the operator is expert, the wet syringe may be used. Theoretically, uncitrated blood should be preferable to citrated blood, because the effect of citration is to prevent coagulation and the defect in hemorrhagic disease of the new-born is in coagulation. Practically, citrated blood has been used by many operators with success. Two ounces of blood are sufficient to stop the hemorrhage. More should be given, of course, if there has been much loss of blood.

It has been supposed that the blood of new-born infants was indeterminate as regards grouping and that it was, therefore, unnecessary to determine compatibility before selecting a donor. It has recently been shown that, with a sufficiently delicate technic, 80% of new-born infants can be placed in one of the four recognized groups and that one-third as many infants as adults have iso-hemolysins in their blood. There apparently should be some danger in doing transfusions in the new-born without determining the compatibility of the blood before selecting a donor. Practically, however, the experience of many men over many

years has shown that the danger is so slight that it is negligible. Nevertheless, if there is time and the facilities for making the tests are at hand, it is advisable to determine the compatibility and to use only a suitable donor. If the time is limited or the facilities are not at hand, transfusion should be done with any donor available.

The administration of drugs by the mouth, of adrenalin subcutaneously, or of gelatin by any method is useless. Bleeding from the surface of the body can sometimes be temporarily diminished or stopped by the application of Monsel's salt, thromboplastin or adrenalin. Constant pressure, however, usually gives better results. If the cord is bleeding, it should be retied, but no wounds should be made in it in retying.

CONGENITAL ATELECTASIS OF THE LUNGS

Congenital atelectasis is due to the persistence of the fetal state as the result of the failure of the whole or part of a lung or lungs to expand. The failure to expand may be due to asphyxia with insufficient methods of resuscitation, so that the baby has not cried and breathed deeply enough to expand the lungs, or to congenital feebleness of the chest muscles which are not strong enough to produce complete expansion. The latter cause is especially common in premature infants. Congenital atelectasis is more common than is usually supposed.

Pathology.—The first parts of the lungs to expand are the anterior borders of the upper lobes. Then follow the anterior portions of the upper lobes, the anterior portions of the lower lobes and the posterior portions of the upper lobes. The atelectatic portions are, therefore, most often the posterior portions of the lower lobes, and next, the posterior portions of the upper lobes. One or both lungs may be involved. Usually parts of both are involved, but sometimes the whole of one lung and none of the other. The parts of the lungs which are expanded are likely to be emphysematous. The unexpanded portions of the lungs are brownish-red, vascular and do not crepitate. There may be secondary dilatation of the right side of the heart and passive congestion of the liver and spleen.

Symptomatology.—There may be no symptoms of atelectasis. A baby may live for days or weeks and show no symptoms, except, perhaps, feebleness, and then die suddenly. The autopsy, however, will show atelectasis of the lungs. Sudden death without symptoms of atelectasis is especially likely to happen in premature infants. On the other hand, there may be marked symptoms from the first; cyanosis, dyspnoea, gradual failure and death from asphyxia or in convulsions. In other instances there are symptoms, but these are not marked. The baby does not thrive well, the cry is never hard and strong and there may be slight cyanosis all the time or fleeting attacks of slight or moderate cyanosis. Sometimes there are attacks of marked cyanosis with prostration. In many of these cases death finally occurs in an attack of cyanosis or with a convulsion. In many instances, however, even when the symptoms are pronounced, the babies gradually improve and entirely recover. This may happen both with and without treatment. Others improve rapidly and recover with proper treatment.

Physical Signs.—The physical signs are often very indefinite, because of the small size of the lungs and the transmission of sounds from one side of the chest to the other. I have repeatedly searched for atelectasis in premature infants because of symptoms suggesting its presence, and been unable to find anything wrong. Nevertheless, at autopsy a whole

lung or parts of both lungs were not expanded. Inspection seldom shows any diminution in the respiratory excursion on the affected side. Percussion is unreliable because the lungs are so small, the chest is so elastic and the emphysematous areas obscure the dullness which would be expected from the atelectatic areas. The respiration is usually superficial and the normal sound in the expanded areas prevents the bronchial sound from the unexpanded areas, into which little air enters, from being noticed. While theoretically there should be bronchial respiration over the atelectatic areas, practically the sound which is heard is vesicular or, at most, bronchovesicular. The voice sounds should be bronchial over the atelectatic areas, but the baby seldom cries loudly, the sounds are mixed with those from the expanded emphysematous areas, and, in consequence, they are at most only bronchovesicular. Moist râles are sometimes heard over the atelectatic areas.

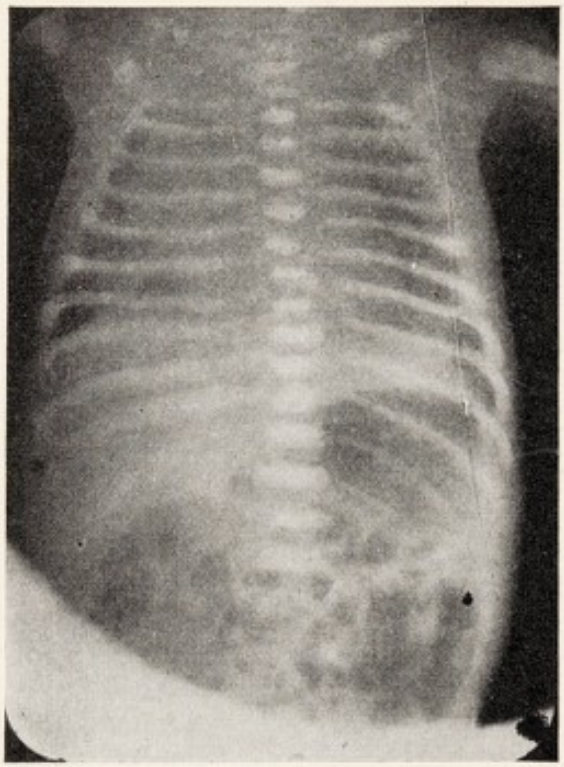

FIG. 32.—Congenital atelectasis of the lungs. Taken on first day of life.

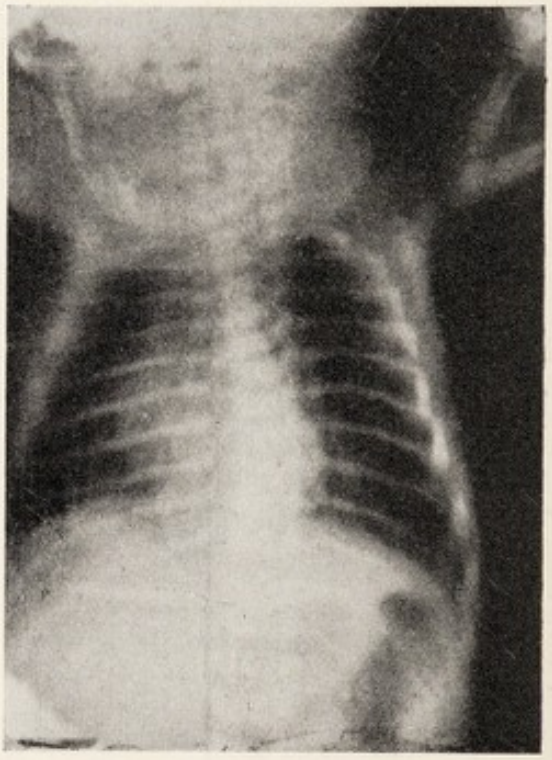

FIG. 33.—Congenital atelectasis of the lungs. Same baby. Taken on fourth day.

The Roentgen ray, however, gives most valuable information as to the presence or absence of atelectasis and as to whether the lungs expand or not as time goes on. The atelectatic areas, of course, show a shadow.

Diagnosis.—Atelectasis of the lungs should be suspected when a newborn baby is cyanotic, either constantly or intermittently, does not cry loudly, does not breathe normally or is unusually feeble without apparent cause. It is often very difficult to recognize it, however, because of the unreliability of the physical signs. A Roentgenogram of the chest should be taken in all doubtful cases. It is sometimes difficult to distinguish between a congenital heart lesion without a murmur and atelectasis. The Roentgenogram will usually settle the diagnosis. In one baby that I saw, who had a patent ductus arteriosus without a murmur, the lungs were engorged with blood and gave all the physical signs of atelectasis. The physical signs of septic infection of the lungs and of the

acute inflammatory diseases of the lungs may be the same as those of atelectasis and may be accompanied by cyanosis. In all of these conditions, however, there is an elevation of the temperature, which in atelectasis is normal or subnormal.

Treatment.—The most important form of treatment is the preventive. It is impossible, of course, to always prevent babies from being born prematurely or congenitally feeble. Almost all full-term babies can, however, be made to cry vigorously at birth and to thoroughly expand their lungs. Babies whose lungs have not expanded should be immersed, unless they are too feeble, in water at 110° F. for a few minutes, plunged into water at 60° F. for a few seconds, put back into hot water and so on, as is done in resuscitating new-born infants, several times daily. Spanking is also efficacious. The object of both the plunging in cold water and the spanking is, of course, to make the baby cry vigorously and thus expand the lungs. Babies with atelectasis should not be allowed to lie in one position long at a time, but should be turned frequently from side to side. They must be kept warm and properly fed, in fact, treated like a premature infant. Oxygen is useful when there is cyanosis. It should be given whenever there is cyanosis, not for a few minutes every hour or so, as is the usual custom.

URIC ACID INFARCTIONS

Uric acid infarctions are probably present in the kidneys of all new-born infants, but it is only occasionally that they produce any symptoms. The primary cause of these infarcts is presumably the large proportion of uric acid in the urine of young infants but, as these infarcts are not found in later life when the urine contains large amounts of uric acid, there must be some other contributory cause which is active only in early infancy.

Pathology.—The infarctions are made up of collections of uric acid crystals or of amorphous and crystalline urates in the straight tubules of the kidneys, that is, in the pyramids. An infarct, therefore, looks like a number of yellow, orange or brownish-yellow streaks arranged in the form of a fan. There are usually several infarcts in each kidney. There may be a slight secondary irritation or even inflammation of the tubules or pelves of the kidneys.

Symptomatology.—In most instances there are no symptoms, unless it may be that these infarcts account in part for the scanty excretion of urine during the first few days of life. It is much more probable, however, that the scanty excretion accounts for the increased density of the urine and the consequent precipitation of the uric acid and urates in the tubules. When these collections of uric acid and its salts are washed out in the urine they often make reddish or reddish-yellow stains on the diapers. These stains are most common during the first week of life, not uncommon during the first few weeks, and may occur during the whole of infancy, if the urine becomes very acid. The passage of the crystals may cause pain and sometimes priapism. The urine may contain albumen, red cells, renal epithelium and an occasional cast as the result of the irritation caused by the crystals. It is, of course, highly acid and concentrated.

Diagnosis.—The reddish and reddish-yellow stains on the diapers are sometimes mistaken for blood stains. It is usually easy to distinguish them by visual inspection, but, if there is any doubt, the question can be settled with the microscope. The pinkish stain, due to some unknown

form of bile pigment, which is so often seen on the diapers about a stool, is sometimes attributed to a uric acid infarction. This stain is always a diffuse one, however, and evidently connected with the stool rather than with the urine. The stains due to infarctions are localized and thicker. The bile stains are seen more often in late infancy; the uric acid stains in early infancy.

Treatment.—The formation of large infarcts can probably be hindered and the development of any symptoms, except of the stains on the diapers, prevented by the administration of water during the first few days of life. The new-born baby should get from eight to twelve ounces of liquid daily. If symptoms have developed, it is advisable to give from ten to twenty grains of the citrate or acetate of potash every twenty-four hours.

UMBILICAL HERNIA

Hernia into the umbilical cord is a very unusual congenital malformation due to a defect in the development of the anterior abdominal wall. I have never seen a case. The sac is covered with membrane and may contain any or all of the abdominal organs. It is practically a hopeless condition, unless an operation is performed at once. If it is, the chances are considerably in favor of recovery.

The ordinary umbilical hernia is not present at birth, but usually develops soon after the cord has come off. It may develop at any time during infancy as the result of crying, coughing or distention of the abdomen with gas. It is more common in poorly nourished infants. It is due to the protrusion of a sac of peritoneum containing intestine through the abdominal ring, which is weak in its upper portion. The protrusion is not infrequently through and just above this weak, upper portion of the ring. The tumor is from one-fourth to three-fourths of an inch in diameter and seldom protrudes more than an inch.

An umbilical hernia seldom causes any discomfort, although when it is large and becomes distended with gas, it is, apparently, sometimes a little painful. Strangulation is most unusual; I have never known of a case.

Prognosis.—The tendency of umbilical hernia in infancy is to recovery, even without treatment. An adult is almost never seen with an umbilical hernia which has persisted from infancy, which shows that recovery must be the rule. Recovery is quicker under treatment. Most cases are cured in from six months to two years, if properly treated. The earlier that treatment is begun, the quicker is the recovery. Occasionally, however, the hernia persists in spite of treatment. If it is still present at five years, it probably will persist.

Treatment.—It is useless to try to prevent or cure an umbilical hernia with a pad and a tight flannel binder. It is impossible to put the binder on tightly enough to do any good and, if it is put on tightly, it merely makes the baby uncomfortable if it has gas or has taken a full meal. The best treatment is with a plaster strap. The ordinary zinc oxide plaster is usually satisfactory. It should be two inches in width. The hernia should be pushed in and then the skin on each side of the navel folded in to act as a splint. The strap should then be applied. The strap should be long enough to almost reach the edge of the ribs. It should not touch the ribs and should not go around the body. If it does, it may cause much discomfort if the abdomen is distended from any cause. Any intelligent mother or nurse is capable of applying the strap. It should

never be put on, if there is a granuloma of the navel. It is most important to be sure that the baby is quiet and the hernia reduced before the strap is applied. The baby may be bathed in a tub with the strap on. It should be changed when it begins to loosen. It is less irritating to the skin to wet it with ether or gasolene before pulling it off. The hernia must not be allowed to come out while the strap is being changed. The strap usually causes no irritation of the skin, but sometimes does. If it does, moleskin plaster may be tried. The length of the strap may be changed, if the irritated area is near the end of the strap. If it is not, a piece of cloth may be put over the irritated area, under the strap. It is

FIG. 34.—Umbilical hernia. Plaster strap applied; is too long.

not advisable to use a coin or a button over the navel under the strap, because, although a coin or a flat button may prevent the hernia from coming out, it does not favor the closing of the ring in the way that folding in the skin does. If one side of the button is rounded and that side is applied to the navel, it pushes into the opening and prevents its closure. A truss with a hard rubber pad and nipple should never be used because the nipple pushes into the opening and prevents healing. It is impossible for the umbilical ring to contract when a truss of this sort is worn.

If an umbilical hernia has not healed in two years, it probably will not. An operation is then necessary. It may be performed at that time or deferred until the child is four or five years old, according to circumstances.

VENTRAL AND EPIGASTRIC HERNIAS

Small ventral hernias are not very uncommon in the median line above the navel. They may occur anywhere in the abdominal wall, but

usually in the upper portion. They seldom cause any discomfort and are usually easily cured. The treatment is by strapping, as in umbilical herniæ. Operation is seldom necessary.

Diastasis of the Recti Muscles

Separation of the recti muscles, most often above, but sometimes below the navel, is not uncommon in feeble and poorly nourished babies or when the abdomen is much distended. The separation may extend from the ensiform nearly to the pubes. The separation is seldom more than one-half inch in width at the widest point. It causes no symptoms. The muscles always come together again as the baby grows older and stronger.

INGUINAL HERNIA

Inguinal hernia may be present at birth or develop later as the result of crying, coughing, distention of the abdomen with gas or straining at stool from constipation. I have never seen it develop as the result of phimosis. Inguinal hernia is not uncommon in males. In females the hernia may take place into the canal of Nuck and is then known as *hernia of the canal of Nuck*. Inguinal hernia is more common on the right than on the left, but is not infrequently double. The reason that inguinal hernia is so common in infancy is that the anatomical conditions in infancy oppose but little resistance to the protrusion of the intestine. The internal ring is almost directly behind the external and, in consequence, the canal is short. Furthermore, the internal ring is often not entirely closed at birth. Several varieties of inguinal hernia are described. The acquired form of adults is uncommon. The hernia is more often into an open funicular process. If the process is closed above the tunica vaginalis, the intestine cannot get into the scrotum around the testicle and the hernia is described as funicular. If the process is not shut off, the intestine surrounds the testicle in the scrotum and the hernia is called congenital. As a matter of fact, it makes no difference in either the prognosis or the treatment which form it is.

Symptomatology.—In general, there are comparatively few symptoms from an inguinal hernia, whether it is large or small. Sometimes it causes pain and discomfort. An inguinal hernia may become incarcerated or strangulated, but seldom does. Nevertheless, whenever a baby has abdominal pain or discomfort, shows evidences of pain somewhere or the symptoms of intestinal obstruction, inguinal hernia should always be thought of as a possible cause of the symptoms and looked for.

Diagnosis.—If an inguinal hernia is small it may be confused with a small *funicular hydrocele*, an *encysted hydrocele of the cord*, a partially descended testicle in the canal or an enlarged gland. The contents of both a small funicular hydrocele and a hernia can be pushed back into the abdomen. A hydrocele is usually more elastic and fluctuant than a hernia. A hernia often contains gas. A hydrocele is translucent and a hernia is not, but, if the tumor is small, it is difficult to determine whether it is translucent or not. An encysted hydrocele of the cord cannot be reduced, is elastic, translucent, if large enough, and does not contain gas. If the tumor is a testicle in the canal, the testicle is absent from that side of the scrotum. The tumor is firm, does not vary in size, does not contain gas and can be pushed back into the abdomen. An enlarged gland is always of the same size, is firm, not reducible and does not contain gas.

If the hernia is larger, but does not extend into the scrotum, it may be confused with a large, funicular hydrocele. The differential diagnosis is based on the same points as when the hydrocele is small, but translucency will be of greater importance.

If the hernia is large and extends into the scrotum it may be confused with a congenital hydrocele. A hernia is less elastic than a hydrocele, does not fluctuate and is not translucent. Gurgling can usually be felt in it, at any rate, when it is pushed back. Both are reducible.

A hernia into the canal of Nuck may be confused with a *hydrocele of the canal of Nuck*, an *encysted hydrocele of the canal of Nuck* or an

FIG. 35.—Inguinal hernia. Worsted truss applied.

FIG. 36.—Inguinal hernia. Worsted truss applied.

enlarged gland. The principles of the differential diagnosis are the same as in the case of a small inguinal hernia in the male.

Prognosis.—Many small inguinal hernias recover under treatment with a truss, but few of the large ones. There is a marked tendency for hernias to recur after apparent recovery. It is not safe to consider a case recovered until at least six months have elapsed after the truss has been left off without a recurrence of the hernia. Even then, a hernia may recur, if there is any unusual strain.

Treatment.—I thoroughly disapprove of all forms of trusses made by the instrument makers. They do not fit well, are uncomfortable, hard to keep clean, cause atrophy of the muscles from pressure and seldom hold the hernia. The only suitable truss for infants is the worsted truss made from a skein of Germantown yarn. The method of application is

best understood from the accompanying photographs. The loop goes over the ring on the affected side. The yarn must be rewound to fit the individual infant. The skein should be long enough to go around the body, back through the crotch and be tied by a tape behind. It should be thick enough to afford firm support. Several trusses must be provided for the sake of cleanliness, so that they may be washed whenever soiled. The truss can be protected somewhat against soiling by a piece of rubber tissue. The hernia must never be allowed to come out while the truss is being changed. If the hernia is not cured in six months, an operation should be performed. If it is apparently cured, the truss should be worn at least six months longer and then taken off. If at any time thereafter there is a return of the hernia, operation should be done. If the hernia ever becomes incarcerated, it should be operated upon. Operation is obligatory, of course, if the hernia becomes strangulated.

FEMORAL HERNIA

Femoral hernia is very uncommon in infancy, because the opening through which a femoral hernia may come is protected by the formation of the parts, the anterior, superior spine of the ilium and the spine of the pubes being close together and Poupart's ligament being relatively large. A femoral hernia can be easily distinguished from an inguinal hernia, because the origin of the tumor is on the outer side of the pubic spine instead of on the inner, as in inguinal hernia. The tumor is situated at or just below the saphenous opening. The treatment is operative.

SECTION IV

NUTRITION

GROWTH AND DEVELOPMENT

The average full-term, American baby weighs between seven and seven and one half pounds at birth. Girls are likely to weigh a few ounces less than boys. During the first few days after birth there is always a loss of from five to twelve ounces. The birth weight is usually not regained until the baby is two weeks old. If it is entirely artificially fed, the birth weight may not be regained until somewhat later than this. The loss in weight after birth is due in part to the passage of meconium and in part to lack of food and water. It is greater when babies are not kept sufficiently warm and there is, in consequence, greater loss of heat and, therefore, of body tissue.

The average baby usually doubles its birth weight at five months and trebles it at a year. Many apparently normal babies do not, however, treble their birth weight before they are fifteen months old. Babies who are small at birth ought not to be expected, therefore, to weigh as much at any time during infancy as those who are large at birth. In some instances, however, babies that are below the average weight at birth because of lack of sufficient nourishment while *in utero*, gain very rapidly after birth and weigh as much, or more, than the average.

The average baby during the first five months should make an average gain of from six to eight ounces a week. The gain is never as rapid during the rest of the first year and a weekly average of from four to six ounces is satisfactory. The gain each week is hardly ever the same, especially in artificially fed babies. A small gain or lack of gain for one week need not cause worry. If it continues for several weeks, however, there is something wrong. A baby almost never makes a large gain regularly each week. It ought not to be expected to do so, moreover, because every baby occasionally has some slight indisposition or for some reason its food is temporarily not sufficient.

Babies should be weighed regularly during the first two years. It is advisable to weigh them daily during the first two weeks. Once a week is often enough after this until the baby is a year old. Once in two weeks or once a month is often enough during the second year. It causes unnecessary worry, if a baby is weighed daily, because there is never a steady gain in weight from day to day. There may be no gain one day, a loss the next, and a marked gain the next, the total, however, being a gain each week. Children should be weighed once a month.

Balance scales are much preferable to spring scales, which are not at all accurate. It is a good plan to get a pair of ordinary bathroom scales at the start, as they can be used for the whole family and for the baby as it grows older. It is often easier to weigh a baby by having it held in someone's arms and then subtracting the weight of the person holding it, than to try to keep it still in a basket.

Growth continues rapidly during the second year, but it is not as rapid as in the first year. It continues to be rapid up to the fourth year.

Growth in length is also very rapid during the first two years. The average increase in length during the first year is eight inches and during the second year four inches.

Growth in both height and weight is less rapid after the third year and is almost uniform from that time until the prepubertal acceleration. This begins in girls at about eleven years and in boys at about thirteen years, and lasts several years. Roughly, children gain about two inches in height and from five to seven pounds in weight annually after the third year until the prepubertal acceleration. The increase in weight at this time is thus relatively greater than the increase in height. In general, height and weight increase hand in hand. If, however, there is a disturbance of the nutrition, growth in weight is retarded more than growth in height. The urge toward growth in height is so great in childhood that the height often increases when there is no increase in weight and, not infrequently, when there is a little loss in weight. Growth in height is influenced more by heredity than growth in weight. Growth in weight usually corresponds to growth in height.

The rate of growth in height and weight are not synchronous during childhood, but show marked seasonal differences. Growth in height is most rapid during the spring and the first half of the summer and is often associated with a little loss of weight. It is less rapid during the latter part of the summer and autumn. Growth in weight is most rapid during the late summer and autumn and less rapid during the late spring and early summer. In fact, during this period, there is often a loss of weight which is equal to or greater than the gain during the winter and early spring. It is very possible, however, that the relatively small gain in weight during the late spring and early summer is the result of the fatigue incident to the school year and the sudden increase in exercise at this time, while the relatively greater increase in the late summer and autumn is the result of freedom from care and out-door life.

Many figures have been compiled as to the average gain in height and weight at various ages. None of these are entirely satisfactory. The best at present available are those used by The Nutrition Clinics for Delicate Children, devised by Dr. W. R. P. Emerson and Mr. Frank A. Manny of Boston. These tables are based upon the results of the work of a committee of the National Child Health Council, which has endeavored to produce a single weight and height table to take the place of the various tables now in use. The material finally presented consists of Baldwin & Wood's tables for boys and girls of school age and Woodbury's tables for infancy and early childhood. Dr. Emerson believes that the standards in these tables still fall short of those which will be secured when data are obtained only from children who are in normal condition. In the meantime, these tables will serve as a working standard. The boundaries of the safety weight zone, 20% overweight for height and 7% underweight for height, have been determined from clinical evidence. Seven per cent. underweight for height represents a retardation of about one year in growth.

Weights of thirty-four pounds and under have been recorded without any allowance for clothing. For weights of boys from 35 to 65 pounds, 3.5% of the net weight has been added for clothing. Above 64 pounds, 4% of the net weight has been added. For girls the additions are—from 35 to 65 pounds, 3%; from 66 to 82 pounds, 2.5%; for 83 pounds and over,

TABLE I

TABLE OF AVERAGE WEIGHTS OF CHILDREN AT VARIOUS HEIGHTS
Also Showing Weights 7% Underweight for Height

Boys

Height inches	Aver. weight for height lbs.	7% Underweight lbs.	Height inches	Aver. weight for height lbs.	7% Underweight lbs.	Height inches	Aver. weight for height lbs.	7% Underweight lbs.	Height inches	Aver. weight for height lbs.	7% Underweight lbs.
*20.00	8.0	7.5	*34.00	27.0	25.0	48.00	53.0	49.5	62.00	104.0	96.5
*20.25	8.5	8.0	*34.25	27.5	25.5	48.25	53.5	50.0	62.25	106.0	98.5
*20.50	9.0	8.5	*34.50	28.0	26.0	48.50	54.0	50.0	62.50	108.0	100.5
*20.75	9.5	9.0	*34.75	28.5	26.5	48.75	54.5	50.5	62.75	110.0	102.5
*21.00	9.5	9.0	35.00	29.5	27.5	49.00	55.0	51.0	63.00	111.0	103.0
*21.25	9.5	9.0	35.25	30.0	28.0	49.25	56.0	52.0	63.25	112.5	104.5
*21.50	10.0	9.5	35.50	30.5	28.5	49.50	57.0	53.0	63.50	114.0	106.0
*21.75	10.5	10.0	35.75	31.0	29.0	49.75	57.5	53.5	63.75	115.5	107.5
*22.00	10.5	10.0	36.00	31.0	29.0	50.00	58.0	54.0	64.00	117.0	109.0
*22.25	11.0	10.0	36.25	31.5	29.5	50.25	59.0	55.0	64.25	118.5	110.0
*22.50	11.5	11.0	36.50	32.0	30.0	50.50	60.0	56.0	64.50	120.0	111.5
*22.75	12.0	11.0	36.75	32.0	30.0	50.75	60.5	56.0	64.75	121.5	113.0
*23.00	12.0	11.0	37.00	32.0	30.0	51.00	61.0	56.5	65.00	123.0	114.5
*23.25	12.5	11.5	37.25	32.5	30.0	51.25	62.0	57.5	65.25	124.5	116.0
*23.50	13.0	12.0	37.50	33.0	30.5	51.50	63.0	58.5	65.50	126.0	117.0
*23.75	13.5	12.5	37.75	33.5	31.0	51.75	63.5	59.0	65.75	127.5	118.5
*24.00	13.5	12.5	38.00	34.0	31.5	52.00	64.0	59.5	66.00	129.0	120.0
*24.25	14.0	13.0	38.25	34.5	32.0	52.25	65.0	60.5	66.25	130.0	121.0
*24.50	14.5	13.5	38.50	35.0	32.5	52.50	66.0	61.5	66.50	131.0	122.0
*24.75	15.0	14.0	38.75	35.0	32.5	52.75	67.0	62.5	66.75	132.0	123.0
*25.00	15.0	14.0	39.00	35.0	32.5	53.00	68.0	63.0	67.00	133.0	123.5
*25.25	15.5	14.5	39.25	35.5	33.0	53.25	69.0	64.0	67.25	134.5	125.0
*25.50	16.0	15.0	39.50	36.0	33.5	53.50	70.0	65.0	67.50	136.0	126.5
*25.75	16.5	15.5	39.75	36.0	33.5	53.75	70.5	65.5	67.75	137.5	128.0
*26.00	16.5	15.5	40.00	36.0	33.5	54.00	71.0	66.0	68.00	139.0	129.5
*26.25	17.0	16.0	40.25	36.5	34.0	54.25	72.0	67.0	68.25	140.5	130.5
*26.50	17.5	16.5	40.50	37.0	34.5	54.50	73.0	68.0	68.50	141.5	131.5
*26.75	18.0	16.5	40.75	37.5	35.0	54.75	73.5	68.5	68.75	143.0	133.0
*27.00	18.0	16.5	41.00	38.0	35.5	55.00	74.0	69.0	69.00	144.0	134.0
*27.25	18.5	17.0	41.25	38.5	36.0	55.25	75.0	69.5	69.25	145.0	135.0
*27.50	19.0	17.5	41.50	39.0	36.5	55.50	76.0	70.5	69.50	146.0	136.0
*27.75	19.5	18.0	41.75	39.0	36.5	55.75	77.0	71.5	69.75	146.5	136.5
*28.00	19.5	18.0	42.00	39.0	36.5	56.00	78.0	72.5	70.00	147.0	136.5
*28.25	19.5	18.0	42.25	39.5	36.5	56.25	79.0	73.5	70.25	148.5	138.0
*28.50	20.0	18.5	42.50	40.0	37.0	56.50	80.0	74.5	70.50	149.5	139.0
*28.75	20.5	19.0	42.75	40.5	37.5	56.75	81.0	75.5	70.75	151.0	140.5
*29.00	20.5	19.0	43.00	41.0	38.0	57.00	82.0	76.5	71.00	152.0	141.5
*29.25	21.0	19.5	43.25	42.0	39.0	57.25	83.0	77.0	71.25	153.5	143.0
*29.50	21.5	20.0	43.50	43.0	40.0	57.50	84.0	78.0	71.50	154.5	143.5
*29.75	22.0	20.5	43.75	43.5	40.5	57.75	84.5	78.5	71.75	156.0	145.0
*30.00	22.0	20.5	44.00	44.0	41.0	58.00	85.0	79.0	72.00	157.0	146.0
*30.25	22.0	20.5	44.25	44.5	41.5	58.25	86.0	80.0	72.25	158.5	147.5
*30.50	22.5	21.0	44.50	45.0	42.0	58.50	87.0	81.0	72.50	160.0	149.0
*30.75	23.0	21.5	44.75	45.5	42.5	58.75	88.0	82.0	72.75	161.5	150.0
*31.00	23.0	21.5	45.00	46.0	43.0	59.00	89.0	83.0	73.00	163.0	151.5
*31.25	23.5	22.0	45.25	46.5	43.5	59.25	90.5	84.0	73.25	164.5	153.0
*31.50	24.0	22.5	45.50	47.0	43.5	59.50	91.5	85.0	73.50	166.0	154.5
*31.75	24.5	23.0	45.75	47.5	44.0	59.75	93.0	86.5	73.75	167.5	156.0
*32.00	24.5	23.0	46.00	48.0	44.5	60.00	94.0	87.5	74.00	169.0	157.0
*32.25	25.0	23.5	46.25	48.5	45.0	60.25	95.5	89.0			
*32.50	25.5	23.5	46.50	49.0	45.5	60.50	96.5	90.0			
*32.75	26.0	24.0	46.75	49.5	46.0	60.75	98.0	91.0			
*33.00	26.0	24.0	47.00	50.0	46.5	61.00	99.0	92.0			
*33.25	26.0	24.0	47.25	51.0	47.5	61.25	100.5	93.5			
*33.50	26.5	24.5	47.50	52.0	48.5	61.50	101.5	94.5			
*33.75	27.0	25.0	47.75	52.5	49.0	61.75	103.0	96.0			

* Without clothing.

Table II

Table of Average Weights of Children at Various Heights Also Showing Weights 7% Underweight for Height

Girls

Height inches	Aver. weight for height lbs.	7% Underweight lbs.	Height inches	Aver. weight for height lbs.	7% Underweight lbs.	Height inches	Aver. weight for height lbs.	7% Underweight lbs.	Height inches	Aver. weight for height lbs.	7% Underweight lbs.
*20.00	8.0	7.5	*34.00	26.5	24.5	48.00	52.0	48.5	62.00	114.0	106.0
*20.25	8.0	7.5	*34.25	27.0	25.0	48.25	53.0	49.5	62.25	115.0	107.0
*20.50	8.5	8.0	*34.50	27.0	25.0	48.50	54.0	50.0	62.50	116.0	108.0
*20.75	9.0	8.5	*34.75	27.5	25.5	48.75	54.5	50.5	62.75	117.0	109.0
*21.00	9.0	8.5	35.00	29.0	27.0	49.00	55.0	51.0	63.00	118.0	109.5
*21.25	9.5	9.0	35.25	29.0	27.0	49.25	56.0	52.0	63.25	118.5	110.0
*21.50	10.0	9.5	35.50	29.5	27.5	49.50	57.0	53.0	63.50	119.0	110.5
*21.75	10.5	10.0	35.75	30.0	28.0	49.75	57.5	53.5	63.75	120.0	111.5
*22.00	10.5	10.0	36.00	30.0	28.0	50.00	58.0	54.0	64.00	121.0	112.5
*22.25	11.0	10.0	36.25	30.5	28.5	50.25	59.0	55.0	64.25	122.0	113.5
*22.50	11.5	11.0	36.50	31.0	29.0	50.50	60.0	56.0	64.50	123.0	114.5
*22.75	12.0	11.0	36.75	31.5	29.5	50.75	60.5	56.0	64.75	124.0	115.5
*23.00	12.0	11.0	37.00	31.5	29.5	51.00	61.0	56.5	65.00	125.0	116.0
*23.25	12.5	11.5	37.25	32.0	30.0	51.25	62.0	57.5	65.25	126.0	117.0
*23.50	13.0	12.0	37.50	32.5	30.0	51.50	63.0	58.5	65.50	127.0	118.0
*23.75	13.5	12.5	37.75	33.0	30.5	51.75	63.5	59.0	65.75	128.0	119.0
*24.00	13.5	12.5	38.00	33.0	30.5	52.00	64.0	59.5	66.00	129.0	120.0
*24.25	14.0	13.0	38.25	33.0	30.5	52.25	65.0	60.5	66.25	130.0	121.0
*24.50	14.5	13.5	38.50	33.5	31.0	52.50	66.0	61.5	66.50	131.0	122.0
*24.75	15.0	14.0	38.75	34.0	31.5	52.75	67.0	62.5	66.75	132.0	123.0
*25.00	15.0	14.0	39.00	34.0	31.5	53.00	68.0	63.0	67.00	133.0	123.5
*25.25	15.5	14.5	39.25	34.5	32.0	53.25	69.0	64.0	67.25	134.5	125.0
*25.50	16.0	15.0	39.50	35.0	32.5	53.50	70.0	65.0	67.50	135.5	126.0
*25.75	16.5	15.5	39.75	35.5	33.0	53.75	70.5	65.5	67.75	137.0	127.5
*26.00	16.5	15.5	40.00	36.0	33.5	54.00	71.0	66.0	68.00	138.0	128.5
*26.25	16.5	15.5	40.25	36.0	33.5	54.25	72.0	67.0	68.25	139.0	129.5
*26.50	17.0	16.0	40.50	36.5	34.0	54.50	73.0	68.0	68.50	140.0	130.0
*26.75	17.5	16.5	40.75	37.0	34.5	54.75	74.0	69.0	68.75	141.0	131.0
*27.00	17.5	16.5	41.00	37.0	34.5	55.00	75.0	69.5	69.00	142.0	132.0
*27.25	18.0	16.5	41.25	37.5	35.0	55.25	76.0	70.5	69.25	142.5	132.5
*27.50	18.5	17.0	41.50	38.0	35.5	55.50	77.0	71.5	69.50	143.0	133.0
*27.75	19.0	17.5	41.75	38.5	36.0	55.75	78.0	72.5	69.75	143.5	133.5
*28.00	19.0	17.5	42.00	39.0	36.5	56.00	79.0	73.5	70.00	144.0	134.0
*28.25	19.0	18.0	42.25	39.5	36.5	56.25	80.0	74.5	70.25	144.0	134.0
*28.50	19.5	18.0	42.50	40.0	37.0	56.50	81.5	76.0	70.50	144.5	134.5
*28.75	20.0	18.5	42.75	40.5	37.5	56.75	83.0	77.0	70.75	145.0	135.0
*29.00	20.0	18.5	43.00	41.0	38.0	57.00	84.0	78.0	71.00	145.0	135.0
*29.25	20.5	19.0	43.25	41.0	38.0	57.25	85.0	79.0			
*29.50	21.0	19.5	43.50	41.5	38.5	57.50	86.5	80.5			
*29.75	21.5	20.0	43.75	42.0	39.0	57.7.	88.0	82.0			
*30.00	21.5	20.0	44.00	42.0	39.0	58.00	89.0	83.0			
*30.25	21.5	20.0	44.25	43.0	40.0	58.25	90.5	84.0			
*30.50	22.0	20.5	44.50	44.0	41.0	58.50	92.0	85.5			
*30.75	22.5	21.0	44.75	44.5	41.5	58.75	93.5	87.0			
*31.00	22.5	21.0	45.00	45.0	42.0	59.00	95.0	88.5			
*31.25	23.0	21.5	45.25	45.5	42.5	59.25	96.5	90.0			
*31.50	23.5	22.0	45.50	46.0	43.0	59.50	98.0	91.0			
*31.75	24.0	22.5	45.75	46.5	43.5	59.75	99.5	92.5			
*32.00	24.0	22.5	46.00	47.0	43.5	60.00	101.0	93.5			
*32.25	24.0	22.5	46.25	48.0	44.5	60.25	103.0	96.0			
*32.50	24.5	23.0	46.50	49.0	45.5	60.50	105.0	98.0			
*32.75	25.0	23.5	46.75	49.5	46.0	60.75	107.0	99.5			
*33.00	25.0	23.5	47.00	50.0	46.5	61.00	108.0	100.5			
*33.25	25.5	23.5	47.25	50.5	47.0	61.25	109.5	101.5			
*33.50	26.0	24.0	47.50	51.0	47.5	61.50	111.0	103.0			
*33.75	26.5	24.5	47.74	51.5	48.0	61.75	112.5	104.5			

* Without clothing.

TABLE III

TABLE OF AVERAGE WEIGHT AND HEIGHT MEASUREMENTS AT VARIOUS AGES

Boys			Girls		
Age	Height in inches	Weight in pounds	Age	Height in inches	Weight in pounds
Birth	*20.5	*7.5	Birth	*20.5	*7.0
12 months	*29.5	*21.5	12 months	*29.0	*20.0
24 months	*33.5	*26.5	24 months	*33.0	*25.0
36 months	*36.5	*31.0	36 months	*36.0	*29.5
48 months	*39.0	*34.5	48 months	*39.0	*33.0
60 months	41.5	39.0	60 months	41.5	38.0
72 months	44.0	42.0	72 months	44.0	42.0
5½ to 6½ years	46.0	48.0	5½ to 6½ years	45.0	45.0
6½ to 7½ years	48.0	53.0	6½ to 7½ years	47.0	50.0
7½ to 8½ years	50.0	58.0	7½ to 8½ years	50.0	58.0
8½ to 9½ years	52.0	64.0	8½ to 9½ years	52.0	64.0
9½ to 10½ years	54.0	71.0	9½ to 10½ years	54.0	71.0
10½ to 11½ years	56.0	78.0	10½ to 11½ years	56.0	79.0
11½ to 12½ years	58.0	85.0	11½ to 12½ years	58.0	89.0
12½ to 13½ years	60.0	94.0	12½ to 13½ years	60.0	101.0
13½ to 14½ years	63.0	111.0	13½ to 14½ years	62.0	114.0
14½ to 15½ years	65.0	123.0	14½ to 15½ years	63.0	118.0
15½ to 16½ years	67.0	133.0	15½ to 16½ years	64.0	121.0
16½ to 17½ years	68.0	139.0	16½ to 17½ years	64.0	121.0
17½ to 18½ years	69.0	144.0	17½ to 18½ years	64.0	121.0

* Without clothing.

2%. Shoes, coats and sweaters for boys and shoes and sweaters for girls are not included.

The weights in these tables have been approximated to the nearest half pound.

It must never be forgotten that these are tables of averages, not of normals. It is not reasonable to expect children to conform exactly to these figures. Children are not all cast in the same mold. They normally differ in their height and, to a less extent, in their weight in the same way as do adults. Some are normally tall and slight in build; others are normally short and heavy in build. Inheritance plays a large part in their height and build, in the same way that it does in the color of their hair and eyes and the size of their feet and noses. The relation of height and weight to age is of relatively little importance, because that is dependent very largely on inheritance. The relation of weight to height is the important thing. This relation is practically the same at all ages.

It has been found that among children of the well-to-do the tendency is for them to be taller than their parents. It has also been found that, in general, their weight is lower than it should be for their height. The explanation appears to be, not that the race is becoming taller and slimer, but that the children of the well-to-do have more disturbances of nutrition than those financially less favorably situated. The apparent explanation is that the children of the well-to-do are so forced mentally, physically and nervously, that they are unable to maintain a proper state of nutrition. These handicaps more than counterbalance the less favorable hygienic surroundings of the poorer children.

TABLE IV

TABLE SHOWING INCREASES IN WEIGHT AT VARIOUS AGES

Boys

Ages	Year 52 weeks		20 weeks		Quarter 13 weeks		Week	
	Pounds	Ounces	Pounds	Ounces	Pounds	Ounces	Pounds	Ounces
36 months	3.5	56	1.4	22	.9	14	.07	1.1
48 months	3.5	56	1.4	22	.9	14	.07	1.1
60 months	4.0	64	1.6	24	1.0	16	.08	1.2
5½ to 6½ years	4.0	64	1.6	24	1.0	16	.08	1.2
6½ to 7½ years	5.0	80	2.0	30	1.3	20	.10	1.5
7½ to 8½ years	6.0	96	2.4	38	1.5	24	.12	1.9
8½ to 9½ years	6.0	96	2.4	38	1.5	24	.12	1.9
9½ to 10½ years	6.0	96	2.4	38	1.5	24	.12	1.9
10½ to 11½ years	7.0	112	2.8	42	1.8	28	.14	2.1
11½ to 12½ years	9.0	144	3.4	56	2.3	36	.17	2.8
12½ to 13½ years	11.0	176	4.2	68	2.8	44	.21	3.4
13½ to 14½ years	15.0	240	5.8	92	3.8	60	.29	4.6
14½ to 15½ years	11.0	176	4.2	68	2.8	44	.21	3.4
15½ to 16½ years	8.0	128	3.0	50	2.0	32	.15	2.5
16½ to 17½ years	4.0	64	1.6	24	1.0	16	.08	1.2
17½ to 18½ years	3.0	48	1.2	20	.8	12	.06	1.0

Girls

Ages	Year 52 weeks		20 weeks		Quarter 13 weeks		Week	
	Pounds	Ounces	Pounds	Ounces	Pounds	Ounces	Pounds	Ounces
36 months	3.5	56	1.4	22	.9	14	.07	1.1
48 months	3.5	56	1.4	22	.9	14	.07	1.1
60 months	4.0	64	1.6	24	1.0	16	.08	1.2
5½ to 6½ years	5.0	80	2.0	30	1.3	20	.10	1.5
6½ to 7½ years	5.0	80	2.0	30	1.3	20	.10	1.5
7½ to 8½ years	6.0	96	2.4	38	1.5	24	.12	1.9
8½ to 9½ years	7.0	112	2.8	42	1.8	28	.14	2.1
9½ to 10½ years	8.0	128	3.0	50	2.0	32	.15	2.5
10½ to 11½ years	10.0	160	3.8	62	2.5	40	.19	3.1
11½ to 12½ years	13.0	208	5.0	80	3.3	52	.25	4.0
12½ to 13½ years	10.0	160	3.8	62	2.5	40	.19	3.1
13½ to 14½ years	6.0	96	2.4	38	1.5	24	.12	1.9
14½ to 15½ years	4.0	64	1.6	24	1.0	16	.08	1.2
15½ to 16½ years	3.0	48	1.2	20	.8	12	.06	1.0
16½ to 17½ years	1.0	16	.4	6	.3	4	.02	.3

In general, it is probably true that, if the weight is 7% below or 20% above the average weight, there is something wrong with the child. I am inclined to believe, however, that there are certain exceptions to this rule and that the weight of children

may sometimes be outside of these limits without there being anything wrong with the child. If the child seems well in every other way, it is safe, I think, to disregard variations somewhat greater than these. This is especially true, if the parents are tall and thin or short and thick.

It is not uncommon to speak of a child as a little man. This expression conveys an entirely erroneous idea, because the relation of the different parts of the body to each other is different in the infant from that in the young child, that in the young child from that in the older child, and that in the older child from that in the adolescent. The following picture copied from Stratz's "Der Korper des Kindes" shows these variations.

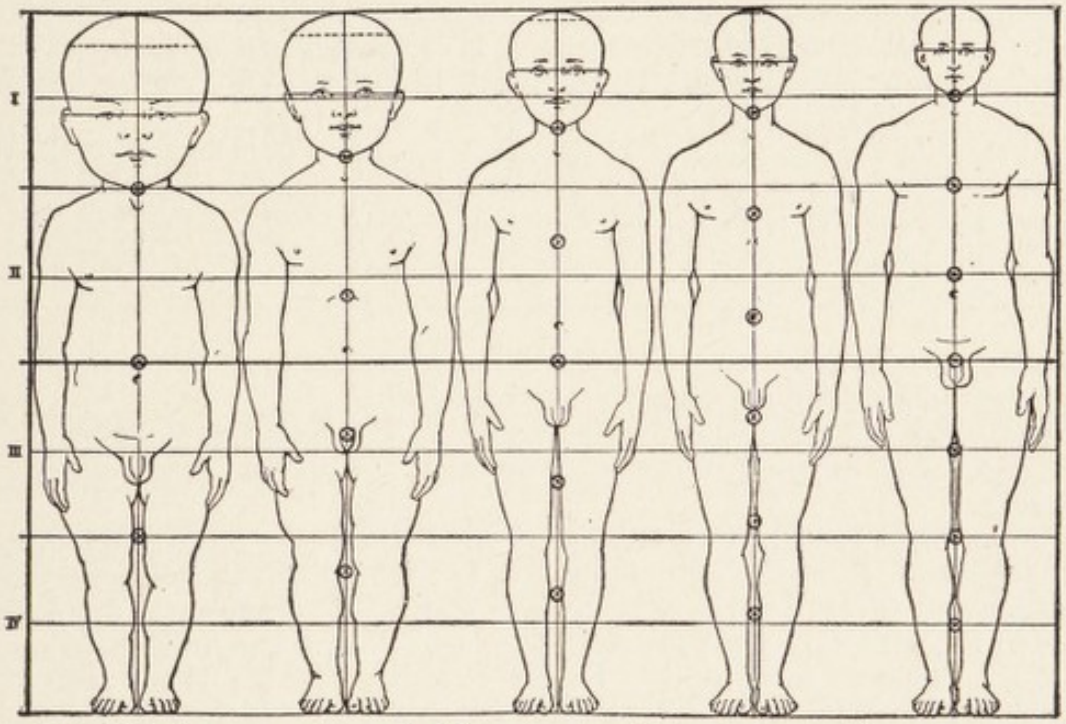

FIG. 37.—Relative proportions of different parts of the body at different ages. (After Stratz.)

Relative Growth of Extremities and Trunk.—It is well known that at birth the legs make up a much smaller proportion of the total length of the body than they do in the adult. There are, however, very few observations as to the relation between the length of the legs and that of the body at different ages. Grover's (Archives of Pediatrics. 1915, **32**, 473) figures show that the distance from the lowest point of the anterior superior spine of the ilium to the sole of the foot is approximately 42.5% of the total length at birth, 45.5% at one year, 48% at two years, 53.5% at five years, 56% at ten years and 57% at twelve years. His figures also show that the length of the arm, measured from high in the axilla to the tip of the middle finger, is approximately 34.5% of the total length at birth, 35% at one year, 36% at two years, 37.5% at five years, 38.5% at ten years and 40% at twelve years.

Head.—The head is relatively large at birth, its circumference being greater than that of the chest. It increases in size very rapidly during the first year. The rate of growth then becomes progressively slower and is very slow after five years. Excepting in rare instances, such as marked hydrocephalus, the absolute size of the head is of less importance in determining whether the head is of normal size or not than the relation

between that of the head and chest. The chest grows faster than the head and surpasses it in size sometime during the third year. The following table, copied from Holt (Diseases of Infancy and Childhood, 1906, p. 20), shows the relations between the head and chest at different ages.

TABLE V

Age	Head				Chest			
	Boys		Girls		Boys		Girls	
	Inches	Cm.	Inches	Cm.	Inches	Cm.	Inches	Cm.
Birth	13.9	35.5	13.5	34.5	13.4	34.2	13.0	33.2
6 months	17.0	43.5	16.6	42.2	16.5	42.0	16.1	41.0
1 year	18.0	45.9	17.6	44.6	18.0	45.9	17.4	44.4
2 years	18.9	48.2	18.6	47.2	19.0	48.4	18.5	47.0
3 years	19.3	49.0	19.0	48.4	20.1	51.1	19.8	50.5
4 years	19.7	50.3	19.5	49.6	20.7	52.8	20.5	52.2
5 years	20.5	52.2	20.2	51.3	21.5	54.8	21.0	53.5
10 years	21.0	53.5	20.7	52.8	25.8	65.6	24.7	63.0
15 years	21.8	55.5	21.5	54.8	30.0	76.6	30.3	76.8

Babies are usually able to hold up the head alone, if the back is supported, when they are eight or ten weeks old.

Fontanelles.—The posterior fontanelle is between one quarter and three eighths of an inch in diameter at birth. This fontanelle closes at six weeks.

The anterior fontanelle is smaller at birth than it is a few days later, when the head has come into shape. It is then approximately one inch in length and seven eighths of an inch in width. It apparently increases somewhat in size with the growth of the head during the first six to nine months. There is some doubt, however, as to whether there really is an absolute increase in size. It then gradually diminishes in size and closes at about eighteen months. The level of the anterior fontanelle is that of the surrounding bones or a little below it. Bulging of the fontanelle means an increase in the intracranial pressure. Depression of the fontanelle means a decrease in the intracranial pressure.

Sutures.—Separation of the cranial bones after birth is abnormal, except that the frontal suture may be open in its upper part for a few days or weeks. Mobility usually persists for six months and sometimes for nine months, at which time bony union is usually fairly firm. Overlapping of the bones at the sutures is very common in early infancy as the result of malnutrition. Imperfect ossification of the bones of the skull at birth is a manifestation of delayed or imperfect development and is not due to rickets or syphilis.

Superficial Veins of Skull.—The superficial veins of the skull are usually visible, if the hair is not too thick.

Hair.—The first hair is sooner or later replaced by a new growth. It sometimes begins to come out in the first few weeks and sometimes is retained for several months. The new hair may come in quickly or slowly, so that some babies always have a considerable amount of hair, while others are bald for a long time.

Face.—The face is relatively small and the cranium relatively large at birth, the relation at that time being about one to eight, while at five years it is one to four and in the adult one to two. If the part of

the head below the orbital arches is designated as the face and that above them as the cranium, the relation of the face to the cranium at birth is approximately one to one, and in the adult two to one. The shortness of the face is due principally to the rudimentary condition of the jaws and teeth. The face is relatively much broader in relation to its length at birth than in the adult, the relation of the breadth to the length at birth being as ten to four and in the adult as nine to eight.

Nose.—The nose is relatively small in infancy and early childhood and the bridge rudimentary and relatively wide.

Teeth.—Infants are sometimes born with teeth. The first tooth usually appears at six or seven months. The teeth usually erupt in groups with considerable regularity. There are so many normal variations, however, that it is difficult to lay down any hard and fast rules. In a general way the first, or temporary teeth, erupt as follows:

2 middle lower incisors	6– 8 mos.
4 upper incisors	8–10 mos.
2 lateral lower incisors 4 anterior molars	12–15 mos.
4 canines	18–20 mos.
4 posterior molars	24–30 mos.
20	

Delay or irregularity in cutting the teeth may be due to rickets or may be merely an individual or inherited peculiarity. It is not wise to attribute delay in dentition to rickets, unless there are other signs of the disease present. Disturbances of nutrition, other than rickets, seldom delay dentition. They often result, however, in an imperfect development of the enamel and a tendency to early decay. Syphilis never produces any characteristic changes in the first teeth, its action being the same as that of other disturbances of nutrition. The first of the second, or permanent, teeth are the so-called "six-year old" molars, which appear at this time behind the posterior molars of the first dentition. The permanent teeth then begin to replace the temporary teeth, the bicuspids taking the place of the temporary molars, after which the permanent molars erupt posteriorly. The permanent teeth erupt in a general way as follows, the lower teeth usually preceding the upper:

4 first molars	6 years
4 middle incisors	7 years
4 lateral incisors	8 years
4 first bicuspids	9 years
4 second bicuspids	10 years
4 canines	12–13 years
4 second molars	12–15 years
4 third molars	17–25 years
32	

The permanent teeth often show, in the same way as the temporary, an imperfect development of the enamel and a tendency to early decay, if the nutrition has been disturbed during early childhood. They also show the typical lesions of syphilis, the so-called "Hutchinson teeth."

Projection of the upper teeth, like crowding together of the upper teeth, is usually the result of imperfect nasal breathing, ordinarily due to adenoids, and not to thumb-sucking as was formerly supposed.

Eyes.—The eye is anatomically developed at birth. Vision is, however, probably very feeble. A strong light evidently causes discomfort during the first few weeks. The baby does not usually fix it eyes until it is at least six weeks old, and coördination is not well developed until three months or later, at which time it seems to recognize objects. It is, therefore, of little use to attempt to test the accommodation in young infants. The pupils react to light almost immediately after birth, and this test can, therefore, be used at once. It is best performed by bringing a light from above the head downward in front of the eyes.

The function of the lachrymal glands is not developed at birth. Tears are shed, as a rule, at about three months.

Neck.—The neck is relatively short during infancy, because of the large size of the head and its tendency to fall forward, the high position of the sternum and the large amount of fat tissue.

Spine.—The spine is largely cartilaginous at birth, ossification not being complete until the thirtieth year. It is, therefore, extremely flexible during infancy and early childhood. The infant is usually not able to sit up alone until it is about eight months old. The lumbar curve is less marked in the infant when sitting than in the child and adult. When infants are forced to sit up before they are able, or when infants and young children are feeble from any cause, they usually sit with a marked general kyphosis, most marked in the lower dorsal region, the so-called "curve of weakness." This disappears when they lie down. It is important not to mistake this condition for the kyphosis due to disease of the spine, which is localized and does not disappear on lying down.

It is very difficult to count the spinous processes in the infant and young child, although it is easy to do so in older children. The first dorsal spine, not the seventh cervical, is usually the most prominent in infancy. Failure to appreciate this fact is likely to lead to error. The spine of the fourth lumbar vertebra is at the level of the highest point of the crest of the ilium at all ages. This fact is of importance in relation to the operation of lumbar puncture. In this connection it is important to remember that at birth the spinal cord extends to the third, and after one year to the second, lumbar vertebra.

The development of the chest and of the various organs of the body is taken up under Diseases of the Chest and of these organs.

Development of Faculties.—*Smell.*—It is probable that the sense of smell is present in a rudimentary condition in the newly born. It develops slowly, however, and the ability to detect fine differences in odors is not acquired until late in childhood.

Taste.—The sense of taste is well developed at birth.

Hearing.—Infants hear little or nothing during the first few days of life, probably because of the swelling of the mucous membrane of the tympanum and the absence of air in the middle ear. The hearing rapidly improves, however, and in a short time becomes very acute.

Sight.—The eye is anatomically developed at birth. Vision is, however, probably very feeble. A strong light evidently causes discomfort during the first few weeks. The baby does not usually focus its eyes until it is at least six weeks old and coördination is not well developed until three months or later, at which time it seems to recognize objects.

Smiling and Noticing.—In a general way, the baby smiles at from four to five weeks and laughs at from five to six months. It begins to notice objects at from six to eight weeks and probably knows its mother or nurse from other people when about three months old. It shows signs

of fear at six months, or even younger, and shows plainly its likes and dislikes at a year.

Talking.—The baby enunciates single words at from ten to twelve months and forms short sentences by the middle or end of the second year. It makes purposeful gestures at eighteen months.

Control of the Sphincters.—The baby should control its sphincters by two years. This point is of comparatively little value, however, as the development of the control of the sphincters depends very largely on the training of the individual baby.

Sitting up and Walking.—The average, normal baby holds up its head alone at three months. It is usually able to sit up without support when it is seven or eight months old. The average baby can stand with support at about one year and without support at about fourteen months. It walks alone about a month later. Some babies stand and walk earlier, however, and others, that are apparently normal in every way, do not until some months later. Some babies go through the creeping stage before they begin to walk or stand, some do not. Some babies never creep, but get about by very peculiar methods of their own, such as hitching along in a sitting position.

FOOD REQUIREMENTS OF INFANTS AND CHILDREN

These requirements must cover the needs for the basal metabolism, the loss in the excreta, the needs for growth and those for muscular activity.

The basal metabolism has been fairly well worked out by Talbot and Benedict. The normal, new-born infant has a basal metabolism of forty-four calories per kilogram, or twenty calories per pound, of body weight and the older infant fifty-five to sixty calories per kilogram, or twenty-five to twenty-seven calories per pound, of body weight. Fat babies have a lower basal metabolism relatively than thin babies, because of the larger proportion of fat tissue, which is inactive. Emaciated and atrophic babies have a higher basal metabolism than normal babies.

After infancy the basal metabolism, whether referred to age or weight, varies somewhat between boys and girls, but it is so nearly the same in the two sexes that for practical purposes these differences do not need to be taken into consideration. Based on Benedict's findings, the average basal requirements are very roughly as follows:

TABLE VI

BASAL REQUIREMENTS AT VARIOUS AGES

Year	2	3	4	5	6	7	8	9	10	11	12	13	14	15
Calories per kilo	55	50	45	42	40	40	38	37	36	36	36	35	34	33
Calories per pound	25	23	20	20	18	18	17	17	16	16	16	16	15	15
Total	600	675	700	750	800	850	900	950	1000	1050	1100	1125	1200	1250

The loss of food value in the excreta is about 10% of that of the food taken. In determining the caloric needs, this factor should properly be calculated, therefore, after all the others have been fixed.

The amount of food required for growth is said to average from 10% to 12% of the total metabolic need. It is much greater, however, during the first two years, especially during the first six months, and at the time of the prepubertal acceleration of growth. How much greater, it is very

difficult to state. It is also very difficult to estimate the relation of the need for growth to that for the basal metabolism.

A large amount of food is required to cover the needs for muscular activity. These needs may vary from 10% to 100% of the needs for the basal metabolism, according to the age, sex and habits of the individual child.

In calculating the caloric needs of the infant and child it is necessary, therefore, to add to the needs for the basal metabolism those for growth and exercise, both of which must be estimated, and then to add 10% of this total for loss in the excreta. It is possible to approximate the amount to be added for growth. It is impossible to make even a reasonable guess at the amount needed for muscular activity. All the tables that are given as to the caloric needs of children are, therefore, merely approximate and represent largely what the authors think children ought to have. The figures which are obtained by estimating the caloric value of the food taken by supposedly well children ought to give more practical results. They can give the minimum, but they cannot give the maximum, because many children that have the price eat far more than they need. Much of the food taken by them passes through them without being utilized and much is wasted in the effort to digest and take care of that which is not needed. The figures given for the boys at St. Paul's School, for example, which are so often quoted, show merely a disgraceful state of affairs and not the caloric needs of children of that age. (F. C. Gebhardt, Boston Medical & Surgical Journal, 1917, **76,** 17.)

Caloric Needs of Babies.—The normal, new-born infant requires approximately sixty-two calories per kilogram, or twenty-eight calories per pound, of body weight. The needs rise rapidly to about one hundred per kilo, or forty-five per pound, during the second and third weeks, reaching the maximum of one hundred and twenty per kilo, or fifty-five per pound, at about six weeks. They gradually fall to about one hundred per kilo, or forty-five per pound, at the end of the first year. During the second year they gradually fall to about ninety calories per kilo, or forty calories per pound.

Caloric Needs of Children.—There is much difference of opinion as to what the caloric needs of children really are and, consequently, many different tables of their caloric needs. It is safe to state that no one really knows how many calories per unit of body weight a child of a given age really needs. Consequently, all of the tables which are given are merely approximate and the expression of individual opinions. It is sometimes said that doubling the basal metabolism will give the total number of calories necessary for growth. Doubling these figures, however, gives much lower totals after five years than are apparently usually taken by normal, average children.

Holt and Fales, from a study of the intake of a series of apparently normal children, come to the conclusion that the caloric needs gradually fall from the end of the first year to about eighty per kilogram, or thirty-six per pound, for boys at six years and that they remain at practically the same point until the sixteenth year. They follow much the same course in girls and fall to about seventy-six per kilogram, or thirty-four per pound, at six years, rising to eighty per kilogram, or thirty-six per pound, from the eleventh to the thirteenth year, and then steadily fall to forty-two per kilogram, or nineteen per pound, the average for an adult woman of moderate activity. They give the following table of caloric needs:

TABLE VII

Boys		Girls	
5 years	1500 calories	5 years	1400 calories
8 years	2000 calories	8 years	1800 calories
12 years	2600 calories	12 years	2900 calories
15 years	4000 calories	14 years	3300 calories

They say that children of both sexes, during adolescence, need one thousand more calories daily than do men and women of moderate activity.

I have taken the liberty of preparing a table of my own based on the tables of others and my own impressions as to what children of various ages usually take. This table is simply one of averages and is only approximate. I am inclined to think that many children can thrive in every way on a smaller number of calories per day. This table is calculated on the average weight without clothes of children of the given ages. It must never be forgotten that children who are underweight require more calories per unit of weight than those who are of average weight and that children who are overweight require fewer calories per unit of weight. It must also never be forgotten that active children require far more calories than do those who are quiet. This table is as follows:

TABLE VIII

CALORIC NEEDS OF CHILDREN

Years	Calories per kilo	Calories per pound	Total calories	
			Boys	Girls
2	90	40	1050	1000
5	80	36	1400	1300
8	80	36	2000	2000
12	75	34	2750	2950
15	70	32	3750	3700

It is necessary, of course, in order to know how many calories a child is taking or how to lay out a diet to contain a given number of calories, to know something as to the caloric value of the various foods. There are many books which give this information more or less accurately. The following table, which I prepared myself from Atwater's tables, is the one I am in the habit of using. It is not complete, but contains most of the articles which children should eat and may be used as a basis for estimating the value of others made from them.

Relative Proportions of the Food Elements.—Even less is known as to the proper relation of the food elements in the diet to each other than as to the necessary number of calories which it should contain. The average composition of human milk shows that the most suitable relations for the infant are presumably approximately 50% of fat, 40% of carbohydrate and 10% of protein. There are no correct data as to the most suitable relations for children. Holt and Fales found that well children, on an ordinary American diet, took about 35% of their calories in the form of fat, 50% in the form of carbohydrates and 15% in the form of protein. It is surprising how near these relations are to those in human milk. They think that these are the optimum proportions. Very likely they are. The fact that children took them

Table IX

Table of Food Values

	Calories	Grams		
		F.	C.	P.
Whole milk, 1 quart	670	38	43	34
Skimmed milk, 1 quart	400	10	43	35
Fat free milk, 1 quart	320	.....	43	35
Heavy cream, 32%, 1 pint	1580	154	22	14
Gravity cream, 16%, 1 pint	860	77	22	14
Buttermilk, 1 quart	360	5	43	35
Whey, 1 quart	260	5	43	9
Beef juice, 1 ounce	10	*	....	2
Crackers, 1 ounce[1]	120	3	20	3
Bread, 1 slice[2]	75	*	15	3
Zwiebach, 1 slice[3]	120	3	20	3
Shredded wheat biscuit	105	*	22	3
Rolled oats (cooked), 1 tablespoonful[4]	35	*	6.5	1.5
Cream of wheat, Ralston and similar cereals (cooked), 1 tablespoonful[4]	40	.....	8.5	1.5
Potato, size of large egg	70	.....	15	2
Macaroni (cooked), 1 tablespoonful[4]	32	*	5	1
Egg — Whole	72	5	....	7
Egg — Yolk	60	5	....	4
Egg — White	12	.....	....	3
Meat / Fish (cooked), 1 ounce[5]	60	3	....	7
Bacon, 1 slice = ½ ounce	90	9	....	1.5
Butter, 1¼ inches cube = 1 ounce	225	24		
Olive oil, 1 tablespoonful	125	14		
American cheese, 1¼ inches cube = 1 ounce	130	10.5	....	8.5
Cream cheese, 1⅛ inches cube = 1 ounce	130	10.5	....	8.5
Sugar — Cane, 1 rounded teaspoonful	25	.....	6	
Sugar — Milk, 1 rounded tablespoonful	60	.....	15	
Green peas / Lima beans (cooked), 1 tablespoonful[4]	40	.....	7	3
Carrots / Squash / Turnip / Beets / Onions (cooked), 1 tablespoonful[4]	30	.....	6	1
Orange, medium sized	50	.....	13	
Apple, medium sized	70	.....	17	
Banana	115	1	24	2
Prunes, 4, without sugar	30		7	

Clear soups and broths made without rice or barley have practically no nutritive value.

The nutritive value of the "fodder" vegetables, such as spinach, string beans, asparagus, lettuce, celery, cauliflower, cabbage, egg plant, tomatoes and cucumbers, is so slight that they may be disregarded.

Pears and peaches have about the same value as apples of the same size.

[1] Crackers vary so much in size that they must be weighed to determine how many it takes to weigh an ounce.

[2] Bread, 1 slice four inches square and three-eighths thick = 1 ounce.

[3] Zwiebach, 1 slice = large slice.

[4] A tablespoonful means as in ordinary serving, not level.

[5] The lean of a lamb chop weighs about an ounce; so does a piece of meat about 1–1–4 inches cube or a thin slice of beef.

* These foods contain from one-quarter to one-half gram of fat in each of the quantities given.

of their own volition does not prove, however, that they are. Nevertheless, they are probably the best data that we have and presumably represent a fairly well-balanced diet.

Protein Needs.—It is evident that, as protein is the only one of the food elements from which old tissues can be replaced and new tissues built, no diet can be adequate which does not contain sufficient protein to cover the needs for maintenance and growth. It is also evident that, as infants and children are growing, they need a relatively larger amount of protein in proportion to their weight than adults and that, as growth is most rapid in infancy and at the time of the prepubertal acceleration, relatively more is needed at those times.

The protein need in infancy is generally believed to be between 1.50 and 2.00 grams per kilo of body weight. Langstein's figures, which are the most accurate thus far obtained, seem to show that the protein need is not as high as 1.50. It is probably safe to say, therefore, that 1.50 grams of protein per kilo of body weight will amply cover the minimum protein need of infants.

Our knowledge of the protein needs of children is very limited. Holt and Fales found that healthy children on an ordinary mixed diet took from forty-four grams daily in the second year to one hundred and thirty grams daily in the fifteenth year. The amount of protein per kilo of body weight taken by these children averaged about four grams at one year. It diminished to 2.6 grams at six years and remained at about this value until the end of growth. These figures show that these amounts of protein are sufficient to cover the protein needs of children of these ages. They do not prove, however, that such large amounts of protein are necessary. An analysis of the available data which I made some years ago seemed to show that the average protein needs of children of four years were 3.5 grams per kilo, of eight years 2.5 grams per kilo and of twelve years two grams per kilo. The average of 2.6 grams found by Holt and Fales is not very different from these figures. It is probable that the minimum protein needs are somewhat less than these average protein needs, perhaps, even as low as three fifths. It is not sufficient, however, to consider simply the amount of protein taken, because according to their amino-acid content, the relative value of the different proteins for growth is very different. The amino-acid content of human milk is, for example, more suitable for the infant for growth than that of cow's milk protein. It is at least 15% more efficient. The amino-acid composition of the protein of cow's milk is, however, more suitable for the growth of children than that of any other protein. Vegetable proteins, as a class, are distinctly less suitable for growth than animal proteins. It is not safe to depend on them entirely for growth, although they may be adequate for maintenance. The children studied by Holt and Fales took about two thirds of their protein in the form of animal protein and one third in the form of vegetable protein.

It is well to remember in this connection that the protein constituents of the diet and of the tissues are built up of nineteen amino-acid radicals of known composition. The only amino-acid radical which is synthesizable by animal tissues is glycocoll. All the others must be present preformed in the diet in order to permit the accretion of living tissue. Certain of the amino-acid radicals are insufficient not only for growth, but also for maintenance. Others, while sufficient for maintenance, are insufficient for growth. No combination of proteins is, for example, sufficient that does not contain both lysin and tryptophane.

While protein is necessary for the replacement of old and the building up of new tissue, it should not be used as a source of energy. It is not economical and throws an unnecessary strain on the organs of elimination.

Fats.—Some fat is necessary in the diet to supply the fat-soluble vitamins. How much is required is not known, but probably very little. Fat is also important in relation to the metabolism of the salts, especially of calcium. It is not known, however, how much is necessary. There is evidently enough in good breast milk. Fat probably facilitates the digestion and utilization of protein. Again, it is not known how much is necessary. Fat serves as a source of energy. Carbohydrates may be substituted for fat for this purpose, but there is much less danger of intestinal disturbance, if fat is used to supply part of the needs for energy.

The breast-fed infant gets five or six grams of fat per kilo of body weight daily, if the milk contains 4% of fat, and at least four grams per kilo, if it contains 3%. It probably needs as much as this. At any rate, it thrives on it. Nothing is really known as to how much fat children need in their daily food. Holt and Fales found that well children on a mixed diet took on the average over three grams of fat per kilo daily when under six years of age and about three grams per kilo during the remainder of the growth period. As the children were thriving it is evident that this amount of fat was enough. It does not show, however, that they might not have done well on smaller amounts or better on larger amounts. It must be remembered that the caloric value of a gram of fat is 9.3, while that of a gram of carbohydrate and protein is only 4.1. The easiest way to push up the number of calories in the diet, therefore, is to increase the amount of fat.

Carbohydrates.—Carbohydrates serve as a source of energy. A certain amount is necessary to complete the oxidization of the fats. Otherwise the ketone acids are formed, which may lead to acid intoxication. If there is not too much fat, however, the carbohydrate derived from the breaking down of protein may take the place of carbohydrates in the food. Carbohydrates are a better source of energy than fat, when there are sudden or unusual demands to cover increased activity, as it is more economical physiologically.

The breast-fed infant gets at least ten grams of carbohydrate per kilo of body weight daily. It probably needs it. At any rate, it thrives on it. It seems advisable, therefore, to give artificially-fed infants at least as much.

Holt and Fales found that well children on an ordinary mixed diet took on the average ten grams of carbohydrate per kilo daily. Fifty-one per cent. of this carbohydrate was sugar and 49% starch. They conclude that it is rational to allow for a child of average activity about twelve grams of carbohydrate per kilo at one year, decreasing the amount to about ten grams per kilo at six years and maintaining it at this value throughout the remainder of the growth period.

The present tendency among the laity is to give an undue proportion of the food requirement in the form of carbohydrates, partly because it is cheaper and partly because of the fondness which most children have for sugar and sweets. The food value of the sugars and the starches is the same. Starches are, however, much less likely to disturb the digestion and set up fermentation in the intestines than the sugars. Furthermore, sweets are very likely to spoil the appetite and, therefore, interfere with

the taking of the proper amount of food. It is advisable, therefore, not to give sugar or sweets to children. Moreover, children that take large amounts of carbohydrates are likely to be fat and flabby and to have a diminished resistance to infection.

Mineral Salts.—The mineral salts are also necessary components of the diet. They are essential, not only for growth of the bones, but also of the other tissues. The normal processes of metabolism cannot go on without them. They do not, of course, serve as sources of energy. The need of infants and children for the inorganic salts is greater than that of adults, partly because they are growing and partly because of their more active metabolism. Both human and cow's milk contain a sufficient amount of the mineral salts, except iron, to meet the needs of the infant. The child that drinks milk regularly, as it should, will always get a sufficient amount of salts, except of iron. The child that is taking an ordinary, mixed, American diet will always get enough of the mineral salts, iron included. For practical purposes, therefore, it is unnecessary in every day work to think of the mineral salts, when children are being rationally fed. Although the intake of the mineral salts may be sufficient, they may not be absorbed in sufficient amounts, if there is diarrhea or some combination of the other food elements which prevents their absorption.

Water.—A considerable amount of water is also necessary for the proper carrying on of the metabolic processes of the body. It is also needed to facilitate the elimination of the products of metabolism. Milk contains nearly 88% of water, showing how important water is for the infant. It is known that children also require considerable amounts of water. How much water they need is not accurately known. It is known, however, that most children do not take enough water and that it is almost impossible to give too much water.

Vitamins.—It is not sufficient for proper nutrition to supply adequate amounts of fats, carbohydrates, proteins, mineral salts and water. Various accessory foodstuffs, or vitamins, must also be present in the diet or disturbances of nutrition develop. These substances are at present undefined.

Three classes of these accessory foodstuffs are at present recognized. A fat soluble, or vitamin A; a water soluble, or vitamin B; and the antiscorbutic, or vitamin C.

The fat soluble A vitamin is most abundant in milk, butter, the yolks of eggs and the green leaves of vegetables. It is abundant in cod liver oil and in the fat of meats. It is not present in lard, unless the pigs have been eating a large amount of green vegetables. There is none of it in the fats obtained from vegetables, such as olive oil and cotton-seed oil. This vitamin is practically unaffected by cooking.

The water soluble B vitamin is abundant in meat, eggs and milk, in the seeds of plants, such as beans and peas, in the cereal grains, if they are not milled, and in many vegetables and fruits. This vitamin is weakened to a certain extent by the temperatures ordinarily reached in cooking and destroyed by higher temperatures. It is very abundant in yeast.

The antiscorbutic vitamin C is present in many vegetables and fruits, also, to a certain extent, in milk and meat. It may be absent, however, in milk, if the cows have not had green fodder. It is nearly or entirely destroyed at the temperatures ordinarily used in cooking.

The following table, copied in a modified form from the report of the joint committee of the Lister Institute and Medical Research Com-

mittee, shows the distribution of these vitamins in the commoner foodstuffs.

TABLE X

Classes of foodstuff	Fat soluble A	Water soluble B	Anti-scorbutic C
Fats and oils.			
Butter	+++	0	
Cream	++	0	
Cold liver oil	+++	0	
Mutton fat	++		
Beef fat or suet	++		
Peanut or arachis oil	+		
Lard	0		
Olive oil	0		
Cotton-seed oil	0		
Hardened fats, animal or vegetable origin	0		
Margarine prepared from animal fat	Value in proportion to amount of animal fat contained		
Margarine from vegetable fats or lard	0		
Nut butters	+		
Meat, fish, etc.			
Lean meat (beef, mutton, etc.)	+	+	+
Liver	++	++	+
Fish, white	0	Very slight if any	
Fish, fat (salmon, herring, etc.)	++	Very slight if any	
Tinned meats	?	Very slight	0
Milk, cheese, etc.			
Milk, cow's whole, raw	++	+	+
Milk, cow's skim, raw	+	+	+
Milk, cow's dried whole	Less than ++	..	Less than +
Milk, cow's boiled whole	Undetermined	+	Less than +
Milk, condensed, sweetened	+	+	Less than +
Cheese, whole milk	+		
Cheese, skim	0		
Eggs.			
Fresh	++	+++	?0
Dried	++	+++	?0
Cereals, pulses, etc.			
Wheat, maize, rice whole grain	+	+	0
Wheat, maize, germ	++	+++	0
Wheat, maize, bran	0	++	0
White wheaten flour, pure cornflour, polished rice, etc.	0	0	0
Dried peas, lentils, etc.	..	++	0
Soy beans, haricot beans	+	++	0
Germinated pulses or cereals	+	++	++
Vegetables and fruits.			
Cabbage, fresh	++	+	+++
Cabbage, fresh cooked	....	+	+
Swede, raw expressed juice	....	..	+++
Lettuce	++	+	
Spinach (dried)	++	+	
Carrots, fresh raw	+	+	+
Potatoes, raw	+	+	
Potatoes, cooked	..	..	+
Onions, cooked	..	..	+ (At least)
Lemon juice, fresh	..	..	+++
Orange juice, fresh	..	..	+++
Apples	..	..	+
Bananas	+	+	Very slight
Tomatoes (canned)	+	++	++
Nuts	+	++	
Miscellaneous.			
Yeast, dried	..	+++	
Yeast, extract and autolysed	?	+++	0
Meat extract	0	0	0

It is evident, after looking over this table, that, when infants are taking raw milk and children taking a reasonable amount of milk and eating an ordinary American diet, it is not necessary to think about the vitamins. The possible lack of the antiscorbutic vitamin in the diet of an infant can easily be corrected by the addition of orange juice or tomato juice. A deficiency of any of the vitamins in the diet of a child will occur only under the most unusual conditions.

PHYSIOLOGY OF DIGESTION

Mouth.—Diastatic ferments are present in the parotid and submaxillary glands of the fetus. Saliva is secreted during the first day of life and has the power of converting starch into sugar as soon as it is secreted. The amylolytic power increases progressively from birth to one year. It is then nearly as strong as in the child. In the child the secretion of saliva depends very largely on whether the food is liquid or solid and how much it has to be chewed. It has been shown, however, that in the infant, contrary to the usual rule, the salivary glands are actively stimulated by milk. Nevertheless, the saliva probably plays but little part in digestion in infancy. How long the diastatic action of the saliva continues in the stomach depends on the buffer value of the food and the establishment of acidity.

Stomach.—The stomach is nearly vertical in the fetus. At birth it lies somewhat obliquely and by two years has almost reached the transverse position. The fundus, which is about one fifth of the stomach in the fetus, increases in size more rapidly than the stomach as a whole. It is not accurately known when the adult relation of one third is reached. There is no characteristic, normal shape for the stomach in the infant. Various types are described. It probably makes little or no difference to the baby what type of stomach it has. The shape apparently depends more on the amount of gas in the stomach than on the quantity or quality of the food.

Gastric Capacity.—The anatomic capacity of the stomach, determined post mortem, is of very little practical importance, because during life milk begins to pass from the stomach into the duodenum almost immediately after it is taken and continues to pass during the time that the meal is being taken. The anatomic capacity, in a general way, is a little more than an ounce at birth, an ounce and a half at two weeks, two ounces at four weeks, three and one half ounces at two months, four and one half at three months and five and one half to six ounces at six months.

Duration of Stomach Digestion.—There is much difference of opinion as to the duration of stomach digestion during infancy. In a general way, it is safe to say that the duration of stomach digestion in the breast-fed baby is between one and one half and two hours and in the artificially-fed baby three hours. These figures are of not as much importance as many people think them to be, however, because the duration of stomach digestion varies materially according to the amount and character of the food taken. It is obvious that it takes longer for a large meal to leave the stomach than for a small one. It is also known that liquids leave the stomach faster than solids. Carbohydrates leave the stomach most quickly. Proteins leave more slowly and the length of time they stay in the stomach depends on their character. Fats leave the stomach last. The duration of the stay of the food in the stomach depends very largely, therefore, on the relative proportions of the different food elements. Furthermore, Ladd has shown that when a baby takes food, the remainder of the last meal often passes at once from the stomach. Another point to be remembered is that, except at night, it is very improbable that anybody's stomach is empty when food is taken. That it is not fatal for babies to take food before their stomach is empty is shown by the fact that babies that are constantly at the breast grow up and that some of the babies that are fed at two-hour intervals live.

Gastric Motility.—The physiology of gastric motility is essentially the same in the infant and child as in the adult. Hunger contractions

are present at birth. The emptying time of the stomach varies inversely with the concentration of the food. The pyloric reflex is present in infancy. When the material in the antrum of the pylorus is acid, the valve opens; when it is alkaline, it remains shut. On the duodenal side of the pyloric valve, an alkaline reaction allows the valve to open and an acid reaction causes it to close. Strongly alkaline food, therefore, delays the opening of the pylorus, while strongly acid food, after it passes into the duodenum, also delays the opening. Free hydrochloric acid is not necessary for pyloric opening in the infant. Considerable doubt has recently arisen, however, as to the importance of this pyloric reflex. The stomach empties much more rapidly when the infant is on the right side than when it is on the left. It empties comparatively slowly when the baby is supine.

Secretions of the Stomach.—It appears from Taylor's observations that there is no appetite or psychic secretion of gastric juice in the infant. Pepsin is always present and active at birth. It is present in the gastric glands as pepsinogen, which is coverted by hydrochloric acid into pepsin. The amount secreted increases from birth until the end of the third month, after which it remains constant. The amount of pepsin produced corresponds to age rather than to weight. There may be no pepsin in severe, chronic disturbances of nutrition. Breast-fed babies secrete less pepsin than artificially-fed babies of the same age. Rennin is almost invariably present at birth. It is in the form of a proferment in the gastric mucous membrane, which is inactive until it has come into contact with hydrochloric acid. Rennin causes the coagulation of milk. A fat-splitting ferment is also present at birth and increases with the age of the infant. It is probable, however, that relatively little fat is split in the stomach. Moreover, the gastric lipase is destroyed by a concentration of 0.2% of free hydrochloric acid. Hydrochloric acid is secreted from birth. The amount secreted depends largely upon the need for it. The more casein there is in the food, the more hydrochloric acid is needed. The greater the buffer value, the more is needed, and the later will free hydrochloric acid appear. It may, indeed, never appear. It sometimes never appears even in the breast-fed. Lactic acid is often present as the result of the breaking down of sugar because of the delay in the appearance of free hydrochloric acid as the result of a high buffer value in the food.

The addition of alkalies to the food neutralizes hydrochloric acid and thus delays the coagulation of milk by rennin. As a result of this delay, it is believed that a portion of the milk is allowed to pass into the duodenum before coagulation takes place. It is possible that this belief may not be true. The addition of sodium citrate to milk changes calcium caseinate into sodium caseinate. Sodium caseinate is changed by rennin to sodium paracaseinate, which is soluble, while calcium paracaseinate, which is formed from calcium caseinate, is insoluble. Coagulation by rennin is thus prevented.

Absorption in the Stomach.—The absorption from the stomach is of relatively little importance. Peptones and sugars may be absorbed, if the concentration is 5%, and salts, if the concentration is 3%. It is said that absorption is more rapid in children up to four years of age than later. Water is not absorbed from the stomach, but passes directly into the duodenum.

Pancreas.—The pancreas increases very rapidly in size during the first three or four months and its fermentative activity increases proportionately. The increase in weight and activity is slower from this time

on. The secretion of the intestinal mucous membrane, which is present at birth, stimulates the production of the pancreatic ferments. It is carried by the blood to the pancreas. All the pancreatic ferments are present at birth; trypsin, which splits proteins, amylopsin, which changes starch into sugar, and steapsin, which splits fat into fatty acids and glycerin. All these ferments are less abundant and active in early infancy than later. Trypsin is secreted into the intestine as trypsinogen, but is changed to trypsin by enterokinase. It is most active in a weakly alkaline or neutral medium, but its action is not entirely inhibited by a weakly acid medium. Trypsin carries the digestion of the proteins from albumoses and peptones to peptides and amino-acids. The fat-splitting ferment, lipase or steapsin, is active in acid, alkaline and neutral surroundings. It is present in the pancreatic juice as a proenzyme which is changed by the bile into steapsin. Amylopsin is not present in as large amounts at birth as it is a few weeks later.

Intestines.—The ferments of the small intestines are all present and active at birth. They are erepsin, enterokinase, invertin, lactase and maltase. The three latter convert the corresponding sugars into monosaccharides. The secretion of enterokinase is stimulated by the pancreatic juice. Erepsin aids the trypsin of the pancreas in carrying on the splitting of the proteins into amino-acids.

Absorption in the Small Intestine.—It is known that in infants and young children the amino-acids are absorbed through the intestinal wall by the blood, not only of the portal but also of the peripheral circulation. It is not known whether the earlier products formed in the digestion of proteins are absorbed or not. The absorption of carbohydrates and of fats is the same in early life as in adults. It is possible that a part of the carbohydrates may be absorbed as disaccharides, but the greater part is undoubtedly in the form of monosaccharides. Carbohydrates are absorbed only by the blood capillaries. The proportion of the carbohydrates absorbed is very high. Fats pass into both the blood and lymph vessels. The maximum of fat absorption in infancy is between two and three hours after a meal. A very large proportion of the fat in the food—90% to 98%—is absorbed in both artificially and breast-fed infants. Salts are absorbed, but may be excreted into the intestines again.

Absorption in the Large Intestine.—In the older child and adult there is very little absorption from the large intestine. It is probable, however, that absorption is greater in infancy and early childhood.

Summary.—It is evident, when these data are considered, that salivary digestion is of very little importance in the infant and that gastric digestion is of much less importance than intestinal digestion. In fact, it is possible for a considerable proportion of the food to reach the intestines without having been acted upon at all by the saliva and gastric secretions. There is no absorption in the mouth and very little in the stomach. Almost the whole of it takes place in the small intestine, although, in early infancy, there may be a certain amount in the large intestine. It is evident, therefore, how much a diarrhea, whatever its cause, may interfere with both the digestion and absorption of food.

Digestion And Metabolism of the Various Food Elements

The digestive and metabolic processes are, in general, essentially the same throughout life. There are, however, certain peculiarities in infancy and early childhood which are worth bearing in mind.

Fat.—The absorption of fat by healthy babies is very good, whether they are taking human or cow's milk. It is almost always over 90% and may be as high as 98% of the fat taken in the food. About 10% of the fat ingested is absorbed in the upper part of the small intestine. The absorption is usually nearly complete at the ileocecal valve, although sometimes considerable amounts may be absorbed in the large intestine. The fat in the stools comes from the food, not from the intestinal secretions. There is a comparatively large amount of fat in the stools during the first days of life, but this decreases from 50% of the dried stool to between 14 and 25% in older infants. Under normal conditions at least 75% of the fat in the stools is in the form of fatty acids nd soaps. "Soap stools" show that fat absorption is below normal. The loss of fat in "soap stools," however, is much less than that in diarrhea. When there is a large amount of soaps in the stools, there is also a loss of mineral salts, chiefly calcium and magnesium. If fat absorption is normal, a high fat intake does not change the mineral composition of the stool. In chronic malnutrition, an increase of fat in the diet raises the output of salts in the stools.

Carbohydrates.—The power of digesting starch is almost always present both in the fetus and in the newly-born. The amount of the ferment increases with the age of the infant, the increase being very rapid during the first weeks of life. It can digest twice as much at eight months as at birth and at twelve months as much as a three-year-old child.

The carbohydrates must be broken down into the monosaccharides before they can be absorbed. A large part of the digestion and absorption of the carbohydrates takes place in the upper part of the small intestine, although a little may take place in the large intestine. There is little or no sugar in the stools under normal conditions. It may be found in the stools when there is diarrhea. "Sugar fever" is not due to sugar, but to salts. The limits of assimilation of the different sugars vary. Roughly, they are for lactose 3.1 to 3.6 grams per kilogram, for grape sugar 5 grams per kilogram and for maltose at least 7.7 grams per kilogram. The assimilation limits of cane sugar are about the same as those of milk sugar. Carbohydrates make the digestion of proteins more complete and favor their retention. They have little or no effect on the retention of ash or the absorption of fats. They may increase the retention of sodium and water. An excess of sugar increases the acidity of the intestinal tract and increases peristalsis, which washes the irritating food out of the bowels. Large amounts of fat, protein and ash are, however, carried out at the same time.

Proteins.—As a rule, less nitrogen is taken in the food of breast-fed babies than in that of artificially-fed babies. When approximately the same amounts are ingested, however, there is less fecal nitrogen in the artificially-fed than in the breast-fed. An increase of nitrogen in the food of babies fed both normally and artificially, their needs being equal, increases the nitrogen in the feces. Nevertheless, the nitrogen in the feces comes principally from the intestinal secretions and the intestinal bacteria. If the caloric value of the food is and remains insufficient, there is an increased retention of nitrogen when the amount of protein in the food is increased. Babies, unlike adults, are able to retain nitrogen, even when they are not receiving the required number of food calories. The amount of fat in the food influences the nitrogen metabolism but little under normal conditions. When babies do not digest fat well, an increase in the fat in the food may, however, cause a negative

nitrogen balance. Carbohydrates, however, have a marked property of sparing nitrogen. All sugars have the same action. The retention of nitrogen per kilogram of body weight decreases with increase in age.

Water and Salts.—The body of the fetus contains a very large proportion of water, the proportion diminishing as the fetus grows older. The body of the new-born infant is relatively richer in water and fat, and poorer in nitrogen and ash, than the body of the child or the adult. The mineral salts play a very important rôle in digestion and metabolism. It is very difficult to understand just what they do, because they are not only absorbed by the intestines but also may be reëxcreted into the digestive tract. I do not feel competent to discuss them. Human milk contains, with the exception of iron, much less of the mineral salts than cow's milk. On the other hand, more of the salts in human milk are in organic combination than in cow's milk. It is supposed, therefore, that they are more easily utilizable. Babies fed on cow's milk mixtures almost always get much more salts than breast-fed babies, but absorb less of them. Roughly, the breast-fed baby absorbs about 80% of the ash in the food and retains between 40 and 50%, while the baby fed on cow's milk absorbs from 50 to 75% and retains a little over 40%. Diarrhea causes a great loss of ash in the stools. Holt and his coworkers found that more than 80% of the ash in the food was lost in severe diarrhea. There was a proportionately greater loss of sodium and potassium than of calcium. The amount of iron in both human and cow's milk is insufficient for the needs of an infant. Anemia would develop at once if it was not for the supply present in the liver. It is probable that the iron in human milk is more easily retained than that in the milk of other animals. The retention of magnesium, sodium, potassium and phosphorus is better on human than on cow's milk. It is probable that the fact that three quarters of the phosphorus in human milk is in organic combination as against one quarter in cow's milk accounts for the better absorption and retention of phosphorus by the breast-fed baby.

BACTERIOLOGY OF THE DIGESTIVE TRACT

Mouth.—The baby's mouth is sterile at birth. It becomes infected during birth from the mother's vagina, or soon after birth from the air or from things which are put into it. It is evident that the flora must be made up of the organisms introduced. When food is taken the flora is increased by those organisms which happen to be in the food. It is again evident that the variety of organisms will be greater when the food taken is cow's milk than when it is human milk. The flora becomes more complicated after the eruption of the teeth, other organisms, such as the leptothrix and fusiform bacteria, being then able to gain a foothold, which they could not do before. It is probable, however, that the bacteria present in the mouth are of very lit le importance, because relatively few of them are swallowed and of those which are most are probably quickly destroyed in the stomach.

Stomach.—The flora of the stomach depends on that of the mouth and on the nature of the organisms introduced in the food. The number of bacteria which enter the stomach depends chiefly on the purity of the food. It is evident that fewer bacteria get into the stomach in breast milk than in cow's milk. It is also evident that fewer bacteria enter in pasteurized and boiled milk than in raw milk. As pasteurization destroys most of the lactic acid forming bacteria, lactic acid fermentation in the

stomach ought to be less common when milk is pasteurized than when it is not. Butyric acid fermentation may occur when the milk is pasteurized, however, as pasteurization does not destroy spores. It cannot when milk is boiled, because boiling also destroys spores. Free hydrochloric acid is able to destroy bacteria in the stomach. The amount of free hydrochloric acid present and the time of its appearance depend, however, on the character of the food, especially on its buffer value. It is very possible, therefore, that, when the food has a high buffer value, there may be no free hydrochloric acid in the stomach and, in consequence, no destruction of bacteria by it. The number of bacteria in the stomach also depends on the length of time that the food stays in the stomach. It is evident that when the food leaves the stomach quickly, fewer bacteria have had time to develop than when it leaves it slowly. In general, only those organisms which grow upon sugars and fats thrive in the stomach. The part which they play is, moreover, as a rule, unimportant.

Duodenum.—The flora of the duodenum depends to a considerable extent upon that of the stomach. The number of organisms is relatively small compared with that in the lower part of the small intestine and the large intestine. The duodenum may be practically free from bacteria during the latter part of the intervals between feedings.

Jejunum and Ileum.—The number of bacteria remains relatively small in the upper portion of this tract, but increases very rapidly toward the lower part of the ileum. The flora depends very largely on the character of the food. It is much simpler in the breast-fed than in the artificially-fed. The predominating organisms are the B. bifidus, the Mic. ovalis, the B. coli, the B. lactis aërogenes, and the B. acidophilus. The B. bifidus is much the most numerous. It is said that the B. lactis aërogenes appears high up, the Mic. ovalis in the lower jejunum and upper ileum and the B. coli and B. acidophilus low down. It is very doubtful, however, whether this is so and of very little importance whether it is or not. In the artificially-fed the B. bifidus is relatively less and the B. coli and the B. acidophilus relatively more numerous.

Large Intestine.—The flora of the large intestine is essentially the same as that of the lower portion of the small intestine and depends very largely on the nature of the food. In breast-fed babies the B. bifidus predominates. Other organisms are cocci, the B. lactis aërogenes, the B. coli and the B. acidophilus. Other acid-forming organisms, such as the butyric acid bacillus and the gas bacillus, are occasionally present. In babies fed on cow's milk no one type of organism predominates. The B. coli and the B. lactis aërogenes are likely to be more abundant than the B. bifidus. The organisms which thrive on proteins are usually more numerous. As a rule, a breast-fed baby's stool is Gram positive, while that of a baby fed on cow's milk is Gram negative.

Stools.—The meconium is sterile at birth, but infection quickly occurs through the anus and a little later from the mouth. Meconium is, however, a poor culture medium, probably because of its low water content, and, therefore, usually contains relatively few organisms. The bacteriology of the stools after food has begun to come through is necessarily the same as that of the large intestine. Roughly, about 25% of the normal dried stool is made up of bacteria. Most of them are dead. When there is indigestion, as much as 60% of the stool may be made up of bacteria.

When a more varied diet than milk begins to be taken, the character of the flora changes, varying with the nature of the food. As soon as the

child begins to take a diet similar to that of the adult, the bacterial conditions in the digestive tract are the same as in the adult.

General Principles.—It is far more important to know and understand what the bacteria in the intestinal tract do than it is to know what they are. It is also easier to understand what they do, if it is remembered that in this connection bacteria may be divided into three great classes; those which grow and thrive on proteins—proteolytic; those which grow and thrive on carbohydrates—fermentative; and those which can grow and thrive on either class of foodstuffs—facultative. It must also be remembered that the facultative bacteria, when both proteins and carbohydrates are present, always grow on the carbohydrates in preference to the proteins. Furthermore, it must be remembered that the products of the decomposition of proteins by bacteria are toxic, while those due to the breaking down of carbohydrates by bacteria are nontoxic. It makes it easier to understand the bacteriology of the intestinal tract, if it is borne in mind that the intestines are simply a container of culture medium for the bacteria in them and act as an incubator. The culture medium which they contain is, moreover, derived almost entirely from the food. The situation is further clarified, if the terms fermentation and putrefaction are properly used. Fermentation means the changes produced in non-nitrogenous nutritive substances, usually carbohydrates, and putrefaction means the changes produced in nitrogenous nutritive substances, that is, proteins and protein derivatives. The products of the breaking down of carbohydrates are acid and those of proteins alkaline. The products of the breaking down of fats are also acid.

The predominant organism in the intestinal flora of the breast-fed infant is the B. bifidus. The organisms associated with it are the Mic. ovalis, the B. lactis aërogenes and the B. coli. The B. acidophilus is also often present, but in small numbers. All of these organisms are either of the types which thrive on carbohydrates or facultative. The reason that the flora is of this type is that human milk provides a culture medium largely carbohydrate in character. Furthermore, the carbohydrate being lactose, it is relatively slowly broken down. The medium remains favorable to fermentation throughout the whole intestinal tract. Fermentation being the predominant condition, the contents of the intestines, at any rate of the small and almost always of the large, are acid. The stools are also acid in reaction. It is probable that the products of the breaking down of fat also play a part in the acid reaction of the breast-fed baby's stool. When infants are fed on cow's milk mixtures to which milk sugar has been added, the intestinal bacterial flora is essentially the same as in the breast-fed baby. When other forms of sugar are used, they are more quickly absorbed and the maintenance of the normal fermentative flora is less constant. When, however, infants are fed on cow's milk, whether whole or diluted, to which little or no sugar has been added, the bacterial flora necessarily changes toward the proteolytic because of the change in the culture medium, the proteins being nearly or quite as abundant in it as the carbohydrates. The obligate fermentative bacteria are replaced to a certain extent by proteolytic types, and the facultative bacteria, which can thrive on either type of medium, become relatively more abundant. The proportion of organisms of the B. bifidus and B. lactis aërogenes types, therefore, diminishes, while organisms of the B. coli type, which are facultative, increase in number and organisms like the B. mesentericus and B. proteus increase.

When other foods, usually proteins and starches, are added to the diet, the relations of the organisms which make up the flora are less constant and there is a more definite stratification of the flora, corresponding to different levels of the tract. Organisms like the Mic. ovalis and the B. lactis aërogenes are most abundant in the upper portion of the small intestine. The end products of the breaking down of the starches being rapidly absorbed, there is little for such organisms to grow on in the lower portions of the intestine. The bacteria of the normal nursling flora are, moreover, unable to break up starches. Lower down in the intestinal tract the flora is made up, therefore, largely of facultative organisms, such as the bacillus coli, and of the organisms which thrive on proteins. The reaction of the intestinal contents changes from one consistently acid to one which, in the lower portions, may be either acid, neutral or alkaline, according to the nature of the intestinal contents, which, of course, depends on the character of the food. The reaction of the stools is, of course, the same as that of the contents of the large intestine. It is, or should be, evident that, as the intestinal flora depends on the character of the culture medium in the intestines and the character of the culture medium on the character of the food, the intestinal flora can be changed by changing the character of the food. The most important point clinically in relation to intestinal bacteriology is whether the processes going on in the intestines are chiefly fermentative or chiefly proteolytic, that is, putrefactive, in type. It makes no difference to the clinician what the bacteria which make up the flora are called. It is what they are doing that counts. The other practical point for the clinician is that the nature of the processes in the intestines can be changed by changing the character of the food.

The products of the activity of the fermentative, or lactic-acid forming, organisms are inimical to the development of proteolytic organisms and also to the growth of organisms of the gas bacillus type. It is probable that this fact protects the young infant to a certain extent against infection by pathogenic organisms, many of which are of the proteolytic type. This fact may also be taken advantage of in the treatment of certain of the diseases of the intestinal tract which are caused by pathogenic organisms of the proteolytic type.

THE STOOLS IN INFANCY AND EARLY CHILDHOOD

I have found, and still find, that the examination of the stools is of great assistance in determining the nature of disturbances of digestion in infancy and early childhood. To me, it affords information which I can not get as easily, quickly or accurately in any other way. There are, however, a number of pediatrists who believe that the examination of the stools affords no information of any value and that nothing can be told from the stools as to the nature of a digestive disturbance or its etiology. There are, I think, several reasons for this belief. One is that certain other pediatrists have laid altogether too much emphasis upon the value of the examination of the stools, have relied too much upon their findings and have failed to appreciate the value of other methods of diagnosis. Their methods and conclusions have prejudiced others against the proper use of the examination of the stools. Another reason is that certain pediatrists have been unwilling to examine the stools carefully enough and for a long enough time to find out for themselves not only how useful the examination of the stools is, but also its limitations. Disbelieving in it, they have condemned it without a fair trial or even without any trial.

It is, of course, not reasonable to believe that a positive diagnosis as to the nature and etiology of all disturbances of digestion can be made simply from an examination of the stools without taking other factors into consideration. The amount and composition of the food, the age of the patient, and the other symptoms must all be taken into account. If these other factors are taken into consideration and the results of the examination of the stools reasonably interpreted and estimated at their proper relative value, I am confident that everyone will find the examination of the stools of as much assistance as I do.

The character of the stools depends primarily on the composition of the food. It is modified by the digestive powers of the individual infant and by the amount and rapidity of absorption of the products of digestion. The amount of absorption depends to a considerable extent on the rapidity with which the intestinal contents pass through the intestinal tract. The character of the stools also depends on the nature of the intestinal bacterial flora. This in turn is dependent, to a large extent, on the nature of the food. The influence which the bacteria exert depends largely on the digestive power of the infant and the rapidity with which the products of digestion are absorbed. The more feeble the digestive power and the slower the absorption, the greater is the affect of the bacteria. It is very difficult, therefore, and often impossible, to draw definite conclusions from the examination of the stools as to just what is going on in the intestines. It is usually possible, however, to determine whether any given food element is properly digested and absorbed or not and in many of the disturbances of digestion to tell what elment is at fault. The presence of an improperly digested food element or of an excess of one of the food elements does not necessarily show that this is the element primarily at fault, although it usually is. An excess of fat in the stool may, for example, simply mean that there is more fat in the food than can be taken care of and that it has simply passed through undigested and unabsorbed. An excess of fat in the stools may again be due simply to its being passed through the intestines before there has been time for it to be absorbed, because of excessive fermentation and peristalsis resulting from an excess of sugar. An excess of any or all of the food elements in the stools may be due simply to excessive peristalsis from causes entirely outside of the digestive tract preventing proper absorption.

Meconium.—The meconium is dark, brownish-green in color. That which is first passed in semisolid, as the result of partial drying out in the large intestine. The remainder is more viscid. It is composed of mucus, bile, intestinal secretions and cells mixed with vernix caseosa, epithelial cells and hairs swallowed with the amniotic fluid. The meconium stools are replaced after from two to four days by stools composed of bile-tinged mucoid intestinal secretions. They are usually dark green, but may be dark brown or brownish-yellow, according to whether the bile pigment is in the form of bilirubin or biliverdin. The change to the normal fecal stool occurs gradually during the next two or three days.

The Starvation Stool.—The starvation stool is composed of bile, the intestinal secretions and bacteria. It is small and brownish or brownish-green in color and looks much like meconium. It may be either constipated or loose. It usually has a stale odor like that of paste, but is sometimes foul. The reaction is usually alkaline. It often contains bile-stained mucus.

The Stools of Breast-fed Infants.—The breast-fed infant has three or four stools daily during the first few weeks or months of life and from

one to three stools daily as long as it is on breast milk and the supply is sufficient. In the beginning the stools have the consistency of thick, pea soup and are of a characteristic golden-yellow color. When the number of stools diminishes, the consistency becomes more salve-like. The odor is slightly sour or aromatic and the reaction slightly acid. The golden-yellow color is due to bilirubin, which, on account of the short time which it remains in the intestine, the relatively low protein content of the milk and the low reducing power of the infant's intestine, passes unchanged through the intestinal tract. The odor is due to a combination of lactic and fatty acids. The acid reaction is due chiefly to the high percentage of milk sugar in human milk, but the relative excess of fat over protein in the milk probably also plays a part.

Breast-fed babies that are apparently thriving in every way often have a considerable number of loose, brownish movements, which not infrequently contain mucus. In such cases the breast milk is usually found to contain a high percentage of proteins. It is also not uncommon for apparently healthy breast-fed babies to have an excessive number of loose, green stools, which often contain mucus and many small, soft curds. When this happens, there is, presumably, usually excessive fermentation in the intestines. In certain cases, however, the milk contains a high percentage of proteins and is normal in other ways. It is not advisable to pay much attention to abnormal movements in the breast-fed baby, if it is thriving and appears well, because many breast-fed babies often go weeks and months without having a normal stool and yet thrive perfectly. In some instances they never have a normal stool. If a breast-fed baby is doing well in other ways, it should never be weaned simply because the stools are abnormal.

The Stools of Infants Fed on Cow's Milk.—*Whole Milk and Whole Milk Mixtures.*—When babies are fed on cow's milk mixtures prepared with milk sugar and in which the relations of the different food elements are essentially the same as in human milk, the characteristics of the stools are, in general, the same as those of the stools of breast-fed babies. They are likely, however, if thriving, to have fewer stools in the twenty-four hours. These are firmer in consistency. In fact, slight constipation is not uncommon for the first few months and is not pathologic. The color of the stools is a lighter yellow, because part of the bilirubin has remained long enough in the intestines to be converted into hydrobilirubin. When babies are fed on whole cow's milk or dilutions of cow's milk without the addition of sugar, the percentage of protein is about the same as that of the fat and not much below that of the sugar. The tendency is then for the intestinal flora to change from the fermentative to the proteolytic. In consequence, the reaction becomes alkaline and the odor modified toward the fecal or cheesy. When milk sugar is added to whole milk dilutions these changes are often prevented by the presence of the sugar.

Skimmed Milk Mixtures.—When babies are fed on skimmed milk or on mixtures containing very low percentages of fat and high percentages of protein, the stools have a slightly brownish-yellow color, a slightly cheesy or foul odor and a strongly alkaline reaction. The change in the color is due to the change of bilirubin into hydrobilirubin. The cheesy or foul odor and the strongly alkaline reaction are due to a preponderance of protein in relation to sugar and fat and the consequent predominance of a proteolytic flora in the intestines. The stools, when spread out, usually have a peculiar, smooth, salve-like appearance. This appearance

is often attributed by those who are not familiar with the characteristic skimmed milk stool to an excess of fat.

Whey and Whey Mixtures.—When infants are fed on whey mixtures, low in fat, the characteristics of the stools are essentially the same as those from skimmed milk, except that they are usually browner.

Buttermilk and Buttermilk Mixtures.—The stools of infants fed on buttermilk and buttermilk mixtures have a peculiar, shiny, salve-like appearance. They are grayish-brown or olive green in color, alkaline in reaction and have a very characteristic acrid odor. The stools of babies to whose food lactic acid has been added often have a similar appearance. They are not infrequently very constipated.

Starch Mixtures.—When starch is added to cow's milk mixtures, the color usually becomes slightly brownish. If there is an abundance of sugar in the mixture, the addition of starch does not change the reaction or the odor. If, however, there is but little sugar in the mixture, the addition of starch tends to change the reaction from alkaline to acid and the odor from foul or cheesy to acid. It is generally believed that barley starch is constipating and oatmeal starch laxative. There is, however, no justification for this belief, different starches acting differently in different babies. When starch flours are added to milk, the small brownish specks from the husks which they contain are often passed unchanged in the stools. They are sometimes mistaken for intestinal sand or dirt.

Maltose-dextrins Mixtures.—The addition of the various combinations of the dextrins and maltose to cow's milk mixtures changes the color to a definite brown, the intensity of the brown varying with the amount of these sugars added. The reaction and the odor of the stools is acid. When the proportion of the dextrins is greater than that of the maltose, these sugars are often constipating. When the proportion of maltose is greater than that of the dextrins, they are likely to be laxative. When these combinations of the dextrins and maltose or the malted foods are given without milk, the stools are dark brown, sticky, strongly acid in reaction and have a characteristic odor.

Beef Juice and Broth.—When beef juice or broth is added to the infant's diet, the color of the stools is changed to brown. It is not uncommon, when babies are having beef juice, for one portion of the stool to be dark brown and another to be bright yellow, the dividing line often being sharp. The dark portion, of course, represents the meal at which the beef juice was taken and the light portion one at which no beef juice was taken. The tendency of beef juice and broth, which are proteins, is to change the reaction of the stools to alkaline and the odor to fecal.

The Reaction of the Stools.—The reaction of the normal stool depends very largely on the relation between the carbohydrates and protein in the food. If there is a relative excess of carbohydrates in the food, the normal fermentative flora is maintained and the reaction is acid. If there is a relative excess of protein in the food, a proteolytic flora develops and the reaction is alkaline. It is probable, however, that the amount of fat in the food also has some affect on the reaction. At any rate, when there is a relatively large amount of fat in the food, the reaction is likely to be acid. When there is an excessive amount of carbohydrates, there may be excessive fermentation of the carbohydrates as the result of bacterial action and the acidity of the stools is markedly increased. If the stools are very acid, they irritate the buttocks. Excessively alkaline stools, however, also cause irritation of the buttocks.

When there is excessive fermentation, the stools are frothy. Frothy stools are sometimes, however, due to gases formed during the decomposition of proteins. Clinically, it is sufficient to test the reaction of the stools by placing wet red or blue litmus paper on a fresh surface of the stool. It is important to get a fresh surface, in order that the reaction may not be changed by that of the urine. The reaction of the normal stool is of comparatively little clinical importance. The reaction of the abnormal stool is, however, of great importance. If I was limited to one test in the examination of an abnormal, loose stool, I should prefer the reaction to any other, because it shows whether fermentation or putrefaction is going on in the intestines.

Odor of the Stools.—The odor of the stools depends very largely on the composition of the food. The fats give the odor of butyric or lactic acid to the stools. The carbohydrates, under normal conditions have but little to do with the odor. If not utilized, they give the odors of lactic, acetic or succinic acids. The proteins give cheesy odors of various sorts, sometimes those of skatol, indol and phenol.

The odor of the normal stool and the influence of variations in the diet upon it have already been mentioned. The stools of fat indigestion may have a strong odor of butyric acid, those of protein indigestion various cheesy or putrefactive odors as the result of the decomposition of the protein by bacteria. When several elements of the food are improperly digested the odor is a combination of those resulting from the decomposition of the various elements. The stools of cholera infantum are almost odorless. Stools composed almost entirely of mucus have a peculiar aromatic odor resembling that of wet hay. When there are deep ulcerative or gangrenous processes in the intestine, the stools have a putrefactive or gangrenous odor.

Color of the Stools.—The real color of the stool, unless it is loose, cannot be determined from the outside, as it may be changed by drying and exposure to the air. The stool must be broken up or smoothed out and the inside examined. The normal variations in the color of the stools, dependent on the composition of the food, have already been described. Abnormal variations are not at all uncommon. Some of them are as follows:

Green.—The most common abnormal color is green. It may vary in shade from a light grass-green to a dark spinach-green. In a general way the darker the shade of green, the greater is its significance. It is of no importance when a stool is otherwise normal, if it is light grass-green in color. It is not abnormal for a stool to change from yellow to light green after it is passed. The green color is not characteristic of any special type of disease. It is almost always due to the change of bilirubin to biliverdin. The cause of this change is not always the same, but it is probably most often excessive fermentation. A green color is sometimes due to the action of the B. pyocyaneus. If it is due to this organism, the addition of nitric acid decolorizes the stool. If it is due to biliverdin, the addition of nitric acid gives the characteristic colors of Gmelin's test.

Gray.—The gray stool is usually due to a combination of the absence of bile and the presence of fat, which is generally in the form of soap. There may be bile in the stools, in the form of the colorless leucohydrobilirubin, however, even when they are gray. The presence or absence of bile can be determined with the corrosive sublimate test, which is reasonably accurate. In this test a small, finely divided, portion of the stool is shaken up in a saturated solution of corrosive sublimate and allowed to

stand for several hours. The solution is colored pink or dull red, if there is bile present. When the stools are gray at birth or become so within a few days after birth, the cause is usually some congenital malformation of the bile ducts. An atresia or obstruction in the intestines below the entrance of the common duct has the same affect. When the stools become gray during infancy and childhood, if there is also bile in the urine and jaundice, the difficulty is usually acute infectious jaundice.

Other Colors.—Other more unusual colors are white, black and blue. *White* stools are made up chiefly of fat in the form of soaps. They may be soft and creamy, but more often are hard and dry, resembling the stools of a dog that has been eating bones.

Black stools are ordinarily due to bismuth, but sometimes to iron or charcoal. The black color is sometimes due to changed blood. In rare instances the stools are *slatey-blue* in color. This color is due to some change in the bile pigments and is of no more importance than the green color.

A *pink* stain on the diapers about a stool is not uncommon and is of little or no pathologic significance. It is due to some unknown change in the bile pigment. It may be mistaken for blood stains or those from uric acid.

Abnormal Constituents.—*Curds.*—The most common abnormal constituents of the stools are curds. There are two kinds of curds, casein curds and fat curds. Casein curds vary in size from that of a bean to that of a pecan nut. They are firm, tough and difficult to break up. When placed in water they sink. They are usually white, but sometimes light yellow in color. When placed in formalin, they become as hard as rocks. They are insoluble in ether. Fat curds are small and vary in size from that of a pinhead to that of a pea. They are usually white, but may be stained either yellow or green. They are soft and easily broken up. When put in water, they tend to float. They are not affected by formalin and are soluble, to a considerable extent, in ether, after acidification and heating.

Mucus.—There is always microscopically some mucus in all stools. It is never visible macroscopically in normal stools, but can often be seen in the abnormal. It merely signifies an increased secretion of the mucous glands of the intestines as the result of irritation of some sort. It does not denote any special type of disease. When the mucus is thoroughly mixed with the feces, it usually originates in the small intestine. When the stool is clay colored, it comes from the duodenum. When it is on the outside of a constipated stool, it comes from the rectum. When stools are composed mostly or entirely of mucus and blood, there is either a severe inflammation of the large intestine or an intussusception. Undigested starch may be mistaken for mucus. Starch can be distinguished from mucus by the addition of some preparation of iodine. This stains starch blue and does not affect mucus. It must not be forgotten that there may be starch on the diaper. The solution of iodine must not be too strong.

Blood.—Blood on the outside of a constipated stool indicates a fissure or crack of the anus. Blood mixed with mucus means either severe inflammation of the large intestine or intussusception. In infancy, blood almost never comes from hemorrhoids. In rare instances, hemorrhage is due to an intestinal polyp. It may be a sign of purpura. Hemorrhage from the bowels during the first few days of life is usually a symptom of hemorrhagic disease of the new-born.

Pus.—In rare instances, pus in the stools comes from a fistula or from an abscess which has broken through into the intestines. In the vast majority of instances, however, pus means a severe inflammation of the large intestine. It does not usually appear except in connection with mucus and blood, and generally not until after they have been present for some time. If the patient survives, it usually persists well into convalescence. It can be found with a microscope in almost every case of inflammation of the colon. It is, however, not of much importance, unless it can be seen macroscopically.

Membrane.—Membrane indicates a very severe inflammation of the large intestine. It is seldom seen, because the patients usually die before membrane appears in the stools.

Undigested masses of food are often present, more often, however, in the stools of older children than in those of babies or young children. Foreign bodies, which have been swallowed, are sometimes present, and occasionally worms.

Microscopic Examination of the Stools.—In the great majority of instances the macroscopic examination of the stools affords data sufficiently reliable for clinical work. This is especially true, if the observer has had considerable experience in the microscopic examination of the stools, so that he is able to appreciate from the macroscopic examination what can probably be found by the microscopic. The macroscopic examination alone, however, may lead to erroneous conclusions, especially as regards the amount of fat and undigested starch. Stools which contain an excess of fat and starch sometimes appear perfectly normal in gross, and microscopic examination will alone prevent mistakes. It is advisable, therefore, except in the plainest cases, to examine the stool microscopically as well as macroscopically. The microscopic examination is a simple one and gives sufficiently accurate information for everyday use. Considerable experience is necessary, however, in order to recognize the normal variations in the microscopic picture. It must never be forgotten that the stools normally contain a certain amount of fat in some form or other, that the amount of fat necessarily depends not only on the amount of fat in the food but also on its relation to the other food elements, and that what seems to be an excessive amount of fat is of no importance, if the baby is thriving and shows no clinical evidences of indigestion. The stools do not normally, however, show undigested starch.

If the feces are hard, they should be rubbed up with a little water. It is important, however, not to dilute them too much. It is also important to examine a portion of the stool which is representative of the stool as a whole.

A small portion of the stool is spread out very thin on a slide and covered with a cover-glass. It is then examined for the presence of undigested tissues or pathologic elements, such as blood, pus and the eggs of parasites.

A second portion is spread out thin in the same way and stained with Lugol's solution (iodine 2, potassium iodide 4, distilled water 100) and examined for starch after a cover-glass has been placed over it. Starch granules stain blue or violet. Broken-up starch granules also stain blue or violet. The presence of whole starch granules is of importance. The presence of a small number of broken-up granules is of much less importance and does not justify a diagnosis of starch indigestion. It is also important to be certain that the stools are not contaminated with starch

from baby powders. Certain microörganisms also stain blue. These are known as iodophilic bacteria. They are associated with faulty carbohydrate digestion. When they are found together with undigested starch, they indicate a more serious condition than when starch granules are found alone. When they are found without starch, they are suggestive of a beginning disturbance in the digestion of the carbohydrates. When they persist after starch has disappeared from the stools, they show that the tendency to trouble with the digestion of starches is still present.

Another portion of the stool, which should not be diluted with water, is spread thin on a slide and stained with a saturated alcoholic solution of Sudan III, a cover-glass being placed over it. Neutral fat drops and fatty acid crystals are stained orange-red. Soap crystals are not stained by Sudan III. A drop of glacial acetic acid is then allowed to run under the cover glass. The mixture is then heated until it begins to simmer. It should not be boiled, because, if it is, the fat drops are driven to the edge of the cover glass and lost. The addition of acetic acid and boiling changes the soaps into fatty acids, which appear as drops, stained as before. Any increase in the amount of fat, after the addition of acetic acid and boiling, indicates the presence of a corresponding amount of soaps. If the specimen is allowed to cool, the fatty acids formed from the soaps crystallize. The crystals usually, however, retain the red stain.

This method of staining with Sudan III distinguishes between neutral fat and fatty acids on the one hand and soaps on the other, but does not show the relative proportions of neutral fat and fatty acids. This point is, however, of little practical importance in the examination of the stools of babies and children. The relative proportions of neutral fat and fatty acids, can, however, be determined by staining with Carbol-fuchsin, the stain being prepared as for tubercle bacilli and then diluted one half with 95% alcohol. Carbol-fuchsin does not stain neutral fat, but stains fatty acids a brilliant red and soaps a dull red. It is possible, therefore, by using these two stains in conjunction to determine accurately enough for clinical purposes the relative proportions of neutral fat and fatty acids and soaps in the stool. An excess of neutral fat indicates that the digestion of fat is not carried on normally; an excess of fatty acids and soaps, that the digestion is normal, but absorption is abnormal. It must be remembered that the mineral oils are stained by Sudan III. Chemical examination of the stools shows that this method is, in certain instances, not very accurate and cannot be relied upon implicitly, therefore, in doubtful cases. In general, however, it is satisfactory for every-day use.

The Bacteriologic Examination of the Stools.—Our knowledge of the bacteriology of the disturbances of digestion and of the various inflammatory diseases of the intestines is so limited at present that no conclusions of clinical importance can be drawn from the microscopic examination of the stools, the only exception being perhaps, the appearance of large numbers of iodophilic bacteria, which, as already stated, points to a disturbance of the digestion of the carbohydrates. In general, Gram-positive bacteria predominate in an acid and Gram-negative in an alkaline stool. The determination of the reaction of the stool gives, however, as valuable information as finding either a Gram-positive or a Gram-negative flora. In inflammatory conditions of the intestines, the bacteriological examination of the stools for dysentery bacilli and other pathologic organisms may be of great importance.

BREAST FEEDING

Chemistry and Biology of Human Milk

Colostrum.—The milk which is secreted during the first few days postpartum is materially different from that which is secreted after lactation is fully established. It is known as colostrum. It is deep lemon yellow in color, presumably because of coloring matter in the fat drops. Its specific gravity ranges from 1.028 to 1.072, the average being about 1.040. It is strongly alkaline in reaction. Its composition is not constant. In a general way, it contains a little less fat and lactose and considerably more protein and salts than the fully developed milk. Roughly, its composition is fat 3.25%, lactose 5.25%, protein 3% and salts 0.40%. The composition of the fat is not quite the same as that of normal milk, as it contains less of the volatile fatty acids. There is also more cholesterin and lecithin than in normal milk. Because of the smaller percentage of sugar, it is not as sweet as the later milk. It is coagulated into solid masses by heating, because of the globulin which it contains.

Colostrum Corpuscles.—Characteristic of colostrum are the colostrum corpuscles, which are granular bodies, four or five times as large as the leucocytes, nucleated and full of fat droplets. They are quite numerous at first, but disappear as lactation is established. They are large leucocytes whose cell membranes are completely filled with fat droplets, and, like other leucocytes, they have ameboid motion. They are, likewise, phagocytic. They recur in the milk whenever lactation is interrupted and when the breasts are not completely emptied. When the breasts are not completely emptied, the protein and sugar are reabsorbed, while the fat is taken up by the colostrum corpuscles.

Human Milk.—Human milk has the same appearance as cow's milk, except that when it is cooled, small white flakes are apt to stick to the side of the bottle. These flakes disappear when the milk is warmed. It has no odor. The taste is sweet and, to the adult, sickish. The color varies from a bluish white to a rich creamy yellow. The amount of fat does not, however, correspond to the appearance. The milk is sometimes very yellow. The yellow color is due to carotin, which is absorbed from ingested vegetables. It has no pathologic significance. It is said that the milk is sometimes greenish, when the mother has been eating liver. I have never seen this. The specific gravity averages between 1.030 and 1.032. It may, however, be as low as 1.020 or as high as 1.036. The reaction is amphoteric, that is, it is acid to phenolphthalein and alkaline to litmus. The explanation of the double reaction is that the milk contains both monophosphates, which are weakly acid, and diphosphates, which react as a base. The average composition of human milk is usually stated to be fat, 4%; lactose, 7%; protein, 1.50%; salts, 0.20%.

Fat.—There is normally considerable variation in the percentage of fat, although it tends to be quite constant in the milk of each individual. The percentage of fat is smallest at the beginning and largest at the end of a single nursing, that in the first milk drawn varying between 1 and 3%, and that in the last milk between 6 and 10%. The amount of fat in the milk may vary materially from nursing to nursing, although the average content through the day is constant. In comparison with cow's milk the fat of human milk is relatively poor in volatile fatty acids. The fat in human milk is in a very fine emulsion, the globules averaging much smaller than in cow's milk, although individual globules may be larger.

Lactose.—The sugar in human milk is milk sugar, that is, lactose. It is essentially the same in human milk as in that of other animals. There is, on the average, 7% of lactose in human milk. It varies less in its quantity than any of the other elements. The extremes are about 4.25% and 11%.

Protein.—Human milk contains two groups of albuminous bodies: *casein*, which is unsoluble in water, and *lactalbumin* and *globulin*, which are soluble in water. There is much difference of opinion as to the relative proportions of casein, lactalbumin and globulin, and residual nitrogen. It is probable that these proportions vary. In general, however, the proportions are approximately, casein, 41%, lactalbumin and globulin, 44 to 39%, residual nitrogen, 15 to 20%. The amount of protein in human milk varies from day to day and in different nursings in the same day. In general, however, there is a gradual diminution in the percentage of protein during lactation.

The casein of human milk is precipitated less easily by acids and salts than that of cow's milk and it does not coagulate uniformly after the addition of rennin. The clot formed is not as large and coarse as that from the casein of cow's milk, but is looser and flocculent. Human milk does not coagulate with rennin alone, but does when the milk is acidified.

Salts.—The average amount of ash in human milk is a little over 0.20%. Like the protein it gradually diminishes a little during the course of lactation. The percentage of salts is higher at the beginning of a single nursing than at the end. The average percentage of *calcium* is between 0.042 and 0.044. The daily variations amount to as much as 0.02%. The calcium content decreases with the progress of lactation. The amount of calcium in the milk cannot be increased by giving calcium salts to the mother.

The amount of *phosphorus* in human milk varies considerably. It depends largely on the amount of casein in the milk. On the average, it probably contains about 0.461 P_2O_5 grams per litre. About three quarters of the phosphorus in human milk is in organic combination, and only one quarter in cow's milk. There is, however, a much larger amount of phosphorus in cow's milk than in human milk, because of the larger proportion of casein which it contains.

There is much difference of opinion as to the amount of *iron* in human milk, the figures of different observers varying between 1.215 and 7.21 mg. per litre. The probable explanation of these variations is that the iron content depends on the general condition, being higher in the healthy and lower in the feeble and debilitated. The amount of iron does not decrease during lactation.

The other salts in the milk are of less importance. Potassium salts are much more abundant than sodium.

The caloric value of a litre of human milk is usually given as 782. It is evident, however, that this can be nothing more than an average figure and that the calories must necessarily vary with the composition of the milk.

Ferments or Enzymes.—A considerable number of ferments are present in human milk. Some act on proteins, others on carbohydrates and fats. It is not likely, however, that these ferments are of much importance in digestion. There are other ferments, such as the salol-splitting and the oxidases, which have the property of splitting up or reducing various substances. It is difficult to see of what use they are to the baby. These ferments are affected by heat. The temperature required to destroy

them varies. No one knows whether these various ferments are of any use or not. Consequently, no one knows whether it does any harm to destroy them. It seems idle, therefore, to bring them into discussions as to the comparative suitability of raw and heated milk in infant feeding.

Transmission of Toxins, Antitoxins, Immunity and Drugs through Human Milk.—Toxins, antitoxins and antibodies may be present in human milk and transferred through the milk to the nursing infant. Agglutinins and various bactericidal substances are also transmitted in the same way. It is also said that infants may be sensitized toward various poisons and albumins through the milk.

Potassium iodide, salicylates, antipyrin, mercury, arsenic, the bromides and hexamethylenamin may be excreted in the milk, and occasionally drugs which are soluble in fat, and acetanilid. They are present, however, only in traces and do not have any affect on the infant. It is possible that morphin and atropin may also be excreted in human milk, as they are in that of animals. They have never been found, however, in human milk. Alcohol is present in human milk in very minute amounts, if large amounts are ingested, but not when small amounts are taken. Salvarsan is excreted in the milk. The amount excreted varies markedly. It may be sufficient in some cases to do good and in others to cause death, while in others there is not enough to have any affect. No attempt should be made to treat syphilitic babies in this way, therefore, and it should be remembered that when salvarsan is given it may be a source of danger to the nursing baby.

Recognition of Human Milk.—There are a number of characteristic reactions which serve to distinguish human milk from that of other animals. The following are the simplest: A 1% aqueous solution of neutral red turns human milk yellow and cow's milk purple (Moro's reaction). The addition of one drop of a 0.25% aqueous solution of neutral blue sulphate to two or three c.cm. of milk turns human milk violet-blue and cow's milk greenish blue (Bauer's reaction). When equal amounts of a 2% aqueous solution of nitrate of silver and milk are mixed, shaken, and quickly boiled for three minutes, human milk changes to a coffee brown or brownish violet color, while cow's milk does not change (Tugendreich's reaction).

Influence of Nervous Conditions and Menstruation on Milk.—Clinically, such things as fright, anger, grief and excitement may stop the secretion of milk or change its composition. No one with a wide clinical experience can doubt the effect of these emotions on the secretion of milk. It is more difficult to know its effect on the composition of the milk. Clinically, there is no doubt that babies occasionally become suddenly ill after the mother has experienced some violent emotion. There are practically no data, however, as to the changes in the composition of the milk under such conditions, except a few given by Rotch. (Pediatrics. 1901, p. 144.)

Grulee and Caldwell found that beginning with the first day of menstruation, there was an increase in the amount of breast milk which lasted from one day to two weeks. The amount then diminished, reaching its lowest point four to seven days before the next menstruation. There seems to be a tendency toward a diminution in the percentage of fat and an increase in that of the protein during menstruation. These variations are no greater, however, than are often seen at other times and are probably within the normal physiologic limits.

Bacteriology.—Human milk often contains micro-organisms, even when there are no pathologic conditions in the breasts. The organism most commonly found is the staphylococcus aureus. It is probable that these organisms enter the milk ducts from the outside. Even when there are bacteria in the foremilk, they are usually absent in that which follows. Under normal conditions, the bacteria in human milk do no harm to the babies taking the milk. They are not of pathologic significance for the healthy infant.

It is said that typhoid bacilli have been found in the milk of women ill with typhoid. It is possible that other organisms may pass in this way. The spirochaeta pallida are not transmitted through the milk.

GENERAL CONSIDERATIONS AND TECHNIC

There is no doubt that the natural food for the human infant is human milk, and that it is a baby's birthright to be nursed by its own mother. There is also no doubt that breast-fed babies are more likely to live than the artificially-fed and that, as a class, they are healthier, more vigorous and more resistant to disease. It is hardly necessary to give figures to prove how much smaller the mortality is in the breast-fed than in the artificially-fed. General figures are, that less than 10% of infantile deaths are in breast-fed babies, that more than 90% of infantile deaths from the diarrheal diseases are in the artificially-fed and that the chances of a breast-fed baby to live through the first year are at least six times as good as those of a bottle-fed baby. It is impossible, of course, to give even approximate figures as to the relative morbidity in the breast-fed and the bottle-fed. It is also impossible to show in figures how much stronger, more vigorous and more resistant breast-fed babies are than the artificially-fed, no matter how evident this is.

The Comparative Frequency of Breast and Artificial Feeding.—The proportion of babies that are breast-fed depends much more on the custom in the given locality and in the given class than on the ability of women of different nationalities and classes to nurse their babies. There is no doubt that, with very few exceptions, all women are able to nurse their infants either wholly or in part. Most of them are able to nurse them entirely, at any rate for several months. There are many reasons why women do not nurse their babies. These reasons vary somewhat according to the social position of the mother. The main reason, however, why women do not nurse their babies is that they do not appreciate the importance of nursing. They do not realize how much smaller the morbidity and mortality is among the breast-fed than among the artificially-fed. They do not appreciate that, even from a selfish point of view, it is really much easier and much cheaper to nurse a baby than it is to feed it artificially. One of the reasons why they do not appreciate these things is that they are not properly advised by physicians, nurses and midwives, many of whom are unfortunately as ignorant regarding these matters as the laity. The chief reason why wealthy and fashionable women do not nurse their babies is that they are not willing to sacrifice their own pleasure and convenience. Other reasons are that their friends do not nurse their babies, even when they condescend to have them, and that their husbands do not wish them to because they do not want them tied down in any way. The chief reason why women of the poorer classes do not nurse their babies, is, as a rule, because they have to go out to work and cannot take their babies with

them. They also are more ignorant regarding the importance of breast feeding and are more influenced by the advertisements of the proprietary foods.

Contraindications to Breast Feeding.—There are very few real contraindications to breast feeding. The most important of these is active pulmonary tuberculosis. A woman with this disease should not nurse her baby, because of the great danger of infecting the baby. It is usually inadvisable for a woman with healed tuberculosis, especially if pulmonary, or with closed tuberculosis, to nurse her baby, if she is foolish enough to have one, because of the danger of starting up or increasing the activity of the lesion. The chance of infection of the infant through the presence of tubercle bacilli in the milk is negligible. The danger lies in the sputum or in the discharges from open sinuses. Syphilis is not a contraindication to a woman nursing her own baby unless it was contracted after the birth of the baby, because, if she has active syphilis, the baby has been infected before birth, and, if the baby has syphilis, the mother also always has it. A syphilitic woman ought not, however, to nurse the baby of another woman. Insanity, feeble-mindedness, and epilepsy are contraindications to nursing only in that the mother may injure her infant. It is allowable, if the mother can be kept under close observation whenever she nurses her baby. Women suffering from serious chronic diseases should not nurse their babies. This is partly because the strain of nursing is almost certain to do them serious harm and partly because their milk is usually of poor quality. The same rule applies, but to a less extent, to very delicate and feeble women. They are, however, often able to nurse their babies for a few weeks, long enough to give them a good start. It is usually inadvisable for women that have had severe hemorrhages, who are septic or who have nephritis to nurse their babies. Babies should not be put to the breast of women with puerperal eclampsia before they have entirely recovered from the illness, because of the danger of serious or fatal symptoms in the baby as the result of anaphylaxis.

There are no contraindications to breast feeding on the part of the infant. Sometimes babies are unable to nurse because of deformities of the lips and mouth. Other premature and congenitally feeble babies are not strong enough to nurse. These babies should be fed, however, on breast milk, and are less able to be deprived of it than normal babies. The milk should be expressed from the breast or drawn with a breast-pump and fed to the baby with a dropper, spoon or Breck feeder or through a tube.

Nursing Given Up Unnecessarily.—One of the reasons why nursing is given up unnecessarily is because it is not appreciated how late the milk may be in appearing. The milk appears in two ways. Sometimes it increases slowly and steadily, while at other times the secretion is very small for a time and then suddenly increases. This sudden increase is often spoken of as the "running-in" of the milk. If the "running-in" of the milk is delayed, the supply of milk is often thought to be insufficient and nursing is given up on this account. Dluski found that in three hundred and twenty-six primiparæ the "running-in" occurred in the second twenty-four hours in about 3%, in the third twenty-four hours in about 35%, in the fourth twenty-four hours in a little over 50%, and in the fifth twenty-four hours in about 13%, while once it occurred in the sixth twenty-four hours. The best way to hasten the appearance of the milk is to empty the breasts as completely as possible. The best way to

do this, if the mother's own baby is not strong and vigorous and does not empty the breasts, is by putting an older and stronger infant to the breast. The older infant not only sucks more strongly, but does not get as tired as the young baby, who often refuses to nurse after a few minutes, if the milk does not flow easily.

It is often taken for granted that because the supply of milk is insufficient while the mother is in bed it will continue to be insufficient after she gets up, and nursing is given up on this account. The supply of milk, however, is very likely to increase after the mother gets up and about and begins to lead her usual life.

It is often believed that it is not worth while for a mother to nurse her baby unless she can nurse it for a considerable time and that it is not only inadvisable, but dangerous, for a mother to nurse her baby in part and give it artificial food in part. Both of these beliefs are, of course, erroneous. There is no time in a baby's life when it is more important for it to have breast milk than during the first few weeks. There is no time in a baby's life at which its digestion is so easily upset and, if upset, so hard to correct, as during the first few days and weeks. The longer it gets breast milk the better start it gets and the easier it is to change to an artificial food later, if it becomes necessary. It is, of course, foolish to think that there is any danger in mixing human milk and artificial food. The digestion of the breast milk is certainly not made any more difficult by the presence of the artificial food and it seems as if the digestion of the artificial food was made easier by the presence of the breast milk. Clinically, at any rate, even a little breast milk makes it easier for a baby to thrive on an artificial food. The explanation probably is that the baby is better able to utilize the proteins of human milk to build tissues than it is those of an artificial food.

Nursing is often not attempted because the nipples are retracted and unsatisfactory. It is also often given up on account of poor nipples or cracked nipples. Nursing should not be given up for these reasons, because the nipples can usually be drawn out or the baby can be taught to nurse with a nipple shield. Furthermore, cracked nipples will almost invariably heal, if time and trouble enough are taken. Even if the nipples cannot be drawn out or the cracks healed, the milk can be expressed and fed to the baby, so that it is not deprived of its natural food.

Not infrequently nursing is not attempted because it is feared that the strain of nursing will be too much for the mother's health. It is true that in rare instances nursing is too great a strain and that it does pull some women down considerably. Even if it does, however, a mother ought to be not only willing, but glad, to sacrifice herself for a time in order to give her baby a good start. A few weeks or months of nursing often makes a great deal of difference to a baby. Furthermore, it should always be remembered that nursing is a physiologic, not a pathologic, condition. Many women are better and stronger while nursing than at any other time. Nursing a baby ought not be to looked upon, as it often is, as a disease to be avoided at any cost.

Many women either think themselves or are thought by their friends to be too nervous to nurse their babies. If they really are, they are too nervous to have babies or to bring them up after the nursing period is passed. No woman will admit this. No woman should give up nursing, therefore, because she is nervous. It is often said that the milk of nervous women is bad. Sometimes it is, sometimes it is not. At any rate, it should be tried. In many instances, the milk of women who are

supposed to be nervous is very good. If it does not agree with the baby, nursing can then be given up.

Finally, no woman who has not tried to nurse her baby has done her duty by it. If she tried, but failed, she has done her best. If she has not tried, she should consider herself responsible for any disturbances of digestion which may develop in her artificially-fed baby.

Feeding during the Early Days and Weeks.—The baby should be put to the breast from six to twelve hours after birth, according to the condition of the mother and the strength of the baby. The object of putting the baby to the breast at this time is not to give the baby food, but to stimulate the breasts to secretion. It should be put to the breast every six hours during the next twenty-four hours and ever four hours during the succeeding twenty-four hours. When the milk appears, the intervals may be shortened. There is much difference of opinion as to what the intervals should then be. Advocates of all intervals from two hours to four hours can be found. Each one believes that he is right and that all the others are wrong. If all of them did not get good results, they would not all believe that they were right. It seems evident, therefore, that babies thrive on both long and short intervals and that the interval chosen is, in general, rather for convenience and because of local custom than because it is the best. I am inclined to think that it is unnecessary to feed new-born babies at so short an interval as two hours and inadvisable to feed them at so long an interval as four hours. It seems to me better to begin with two and one half or three hour intervals, according to circumstances, and, if the shorter interval is chosen, to get on to three hour intervals in a few weeks.

The average amount of colostrum obtained during the first twenty-four hours is between 4 and 6 c.cm., and during the second twenty-four hours, 90 c.cm. In the majority of instances the milk comes in rapidly on the third and fourth days, but may not until a day or two later. It is evident from the small amount of colostrum secreted during the first two or three days that the baby is not intended by nature to get much food during this time. Further proof of this fact is that the initial loss of weight is not prevented by giving extra food from the beginning. It is advisable, however, to give babies water freely during the first few days in order to prevent dehydration and dehydration fever and to flush out the kidneys. At least one or two drachms should be given every two hours and more if the baby can be made to take it. Many authorities believe that it is wise to give a solution of milk sugar and water at this time in order to favor the development of the normal fermentative flora. No one knows, however, whether giving the sugar and water does this or not, while others believe that it does harm. Those who think that it does harm advocate a mixture of saccharin and water instead of sugar and water. The saccharin being entirely inert, there is no advantage in saccharin and water over plain water, however, unless babies take it more willingly.

Most babies begin to act as if they are hungry after the first two days. It must be remembered, however, that crying does not necessarily mean hunger. It may as well mean thirst. Furthermore, babies of this age normally cry more or less. No one knows why they do, but it is certainly not from hunger. If the breast milk has not begun to come in on the third day, it is usually advisable to begin to feed them. It is not necessary, however, as it is evident from experience that a baby will not only not starve to death, but will apparently not be injured, if it has little

or nothing to eat for four or five days. It is most important, however, when beginning to feed a baby, not to give it too much food or too strong a food, because there is no time in a baby's life when it is so easy to disturb its digestion or so hard to cure a disturbance of digestion, if one is set up, as in the first few days of life. If a new-born baby is put on the breast of a woman whose milk is thoroughly established, it is very likely to take too much and be upset. In such instances, therefore, the duration of the nursing must be short at first. In fact, it is often wiser to give a baby diluted breast milk from a bottle for one or two days than to put it at once on a fully established breast. It is said that a baby is better able to digest breast milk, if it has had colostrum for a few days first. There is no proof, however, as to whether this is so or not.

If it is necessary to give a baby an artificial food instead of breast milk, it is very important to start with a weak mixture. It is more necessary to be careful about the exact composition of the mixture at this time than at any other. It is all wrong to think that anything will do for a few days and that a more carefully worked out mixture can be given later, if the breast milk does not develop. In the beginning small amounts of a very weak mixture should be given in order not to disturb the digestion. It is easy to strengthen the mixture and to give more of it later. It is hard to straighten out the digestion, if it has been upset by a strong mixture or too much food. The mixture should be low in fat and proteins and relatively high in sugar. It is also advisable to give a part of the proteins in the form of the whey proteins. A reasonable mixture is fat 1%, milk sugar 5%, whey proteins 0.25%, casein 0.25%. This mixture may be quickly strengthened, perhaps in the first twenty-four hours, to fat 1.50%, milk sugar 6%, whey proteins 0.50%, casein 0.25%, and then in another twenty-four hours to fat 2%, sugar 6%, whey proteins 0.75%, casein 0.25%. When it is not easy or feasible to give a baby a whey mixture, an ordinary mixture with the same percentages of protein may be given, the digestion of the casein being facilitated by the addition of lime water or citrate of soda or by boiling. Two drachms (10 c.cm.) is enough at first. This can be quickly increased to one half and then to one ounce (15 or 30 c.cm.).

The colostrum is supposed to have a laxative action, but it is not known whether it really has or not. If the bowels have not moved well at birth or within the first twenty-four hours, it is advisable, because of the danger of the absorption of toxic products from decomposition of the meconium (see Intestinal Toxemia of the Newborn), to give a teaspoonful of castor oil in order to clean them out thoroughly.

Intervals between Nursings.—There is much difference of opinion as to the proper intervals between nursings. The tendency during the last few years has been to lengthen the intervals materially. I am inclined to think that the short interval of two hours in use some years ago was too short and that the long interval of four hours now advised by many physicians is too long. I do not think that it is advisable, in most instances, to have the interval longer than three hours. There is also much difference of opinion as to how soon night feedings should be stopped. I do not think it is advisable to feed babies between 9 p.m. and 6 a.m. after they are one or two months old, but ordinarily do not stop the evening feeding before they are four months old and often not until they are six months old. If the four hour interval is used, it is necessary to keep on with the evening feeding longer. The four hour interval is, of course, easier for the mother, as it gives her more time to get out and

do other things between nursings. I doubt, however, if it is as good for the baby and feel strongly that a mother should consider her baby's good before her own convenience. It is often easier for women who do not have to take the whole care of their babies to give them one artificial feeding daily. This gives them more time to go out and often enables them to nurse their babies when they otherwise would not be able. When a woman is taking all the care of her own baby and doing her own work, it is easier for her to nurse it entirely than to give it one bottle daily. Another advantage in giving one artificial feeding a day is that the baby becomes accustomed to taking the bottle and weaning is, therefore, much easier, when it becomes necessary. More than that, it is known what artificial food agrees with the baby.

Regularity of Nursing.—Whatever intervals between nursings are adopted, the baby should be nursed regularly at these intervals. Many breast-fed babies do well, of course, that are fed at any and all times both day and night. Nevertheless, babies that are fed regularly are more likely to do well and less likely to be overfed. Furthermore, it is much easier to take care of them. Babies quickly accomodate themselves to regularity in feeding and do not expect to be fed at other times. It is especially important not to nurse a baby at short intervals during the night. In the first place a baby should never sleep with the mother. If it does, it should not be allowed to nurse.

If a baby is not already awake when it is time for it to nurse, it should be waked. If the intervals are regular and of sufficient length, it is very seldom necessary to wake a baby up. It is true that babies on the breast will thrive, even if they are not waked at the proper time, just as they will thrive on breast milk under all sorts of adverse conditions. It is much easier and better, however, to wake them at the proper time.

Duration of Single Nursings.—If there is plenty of milk and a baby is well and vigorous, it will usually nurse for about twenty minutes. Many normal babies, however, get all that they want in ten or fifteen minutes. The time taken varies with the suction power of the baby, the amount of milk in the breasts and the ease with which the milk flows. It is physiologic fact that the milk flows most freely at the beginning of a nursing and that the amount which a baby gets diminishes progressively during the nursing. It gets more than one half of the total amount of the meal in the first five minutes, more than one quarter in the next five minutes, and comparatively little after this. If a baby nurses more than thirty minutes, there is something wrong. It may be that the supply of milk is insufficient or that the baby is too feeble to nurse vigorously and continuously. A baby should not drop off to sleep while nursing. If it does, it means that the supply of milk is insufficient, that it is not hungry, or that it is sick in some way. Although the supply of milk is greatest at the beginning of a nursing, the strength of the milk increases progressively throughout the nursing, the total solids being greater at the end than at the beginning of a single nursing.

If the supply of milk is sufficient, a baby should be put to the breasts alternately, because in this way the breasts are more thoroughly emptied and the production of milk is encouraged. If both are given at the same time, neither is thoroughly emptied and the production of milk is discouraged. There is, moreover, a tendency to reversion to the colostrum stage. If the supply of milk is insufficient, both breasts should be given at each nursing. Alternate breasts should be given at the beginning of each nursing, however, in order that one breast, at least, may be thor-

oughly emptied each time and the supply of milk, in this way, kept up. If they are not given alternately, neither is emptied and the supply of milk is not kept up.

The Amount of Food Taken at a Single Nursing.—The amount of food taken at a single nursing varies materially from nursing to nursing, although the amount taken in twenty-four hours varies but little from day to day, it being understood, of course, that the amount taken in twenty-four hours increases as the baby grows older. The amount taken at a single nursing is, of course, greater on the average, when the intervals between nursings are long than when they are short. If a baby has taken a large meal at one nursing, it naturally will take less at the next. Less milk is taken, if it is rich, than when it is not. Babies, like adults, are also not as hungry at some times as at others. The amount taken at a feeding bears no relation to the anatomic size of the stomach, much more almost always being taken than the stomach can hold. The reason that a baby can take more than its stomach can hold is that the milk passes directly into the duodenum during the nursing.

Technic of Nursing.—The three most important points are that the mother is in a comfortable position while nursing, that the baby is held in such a way that it can completely relax and that the breast is held away from its nose so that it can breathe easily. When the mother lies down to nurse her baby, she should lie on her side, hold the baby in the crook of her arm and with her other hand keep the breast from obstructing its nose. When she sits up to nurse the baby, she should sit in a low chair and rest the arm with which she holds the baby on her knee or on the arm of the chair. A footstool often makes nursing much easier. The more placid and the less nervous and excited the mother is while nursing her baby, the easier it is both for her and for the baby. If she is excited and nervous, she does not relax, the baby is not comfortable and is often so disturbed that it will not nurse.

Difficulty in nursing may be the fault of either the mother or the child. If the trouble is with the baby, it may be some deformity of the lips or mouth, nasal obstruction from adenoids or some other cause, which mechanically interferes with nursing, or weakness. Babies that have had the bottle are often unwilling, when first put to the breast, to take it, because they are unaccustomed to it. Babies that are partly bottle and partly breast-fed often after a time refuse the breast, because it is more trouble for them to get the milk from the breast than from the bottle.

Retracted or small nipples are the most common cause of difficulty in nursing, on the part of the mother. Occasionally the nipples are so large that the baby cannot take them easily. Cracked nipples also interfere materially with nursing. In other instances the mother does not know how to hold the baby to make it comfortable. In others she is nervous and excited and disturbs the baby. In still others the room is noisy and full of people, so that the baby's attention is distracted.

It is not advisable to operate on deformities of the mouth and lips at once. A harelip ought not to be operated upon before the baby is three or four weeks old, and a cleft palate not until it is at least six months old. If possible, the operation should be delayed until the eighteenth month. Breast feeding should not be given up on account of these deformities, however, even if the babies are unable to nurse. They should be given the breast milk in some other way. The methods to be used in feeding are described in the section on these deformities. Adnoids should be removed at once, if they cause interference with nursing,

no matter how young the baby may be. Feeble babies must be fed either wholly or in part in the same ways as those with deformities of the mouth and lips. Babies that are unwilling to take the breast can usually be starved to it. Putting sugar on the nipples, pressing some of the milk into their mouths at the beginning of nursing or the use of a nipple shield will sometimes induce them to take hold. Older babies will sometimes nurse in the dark or when blindfolded, when they will not otherwise.

Care of the Nipples.—Much can be done during pregnancy to bring out retracted nipples by manipulation, the careful application of a breast-pump and sucking. The nipples should be carefully washed and cleaned during the later days of pregnancy in order to remove the excess of epithelium and to clear the openings of the ducts. They should be washed before and after each nursing with sterile water or with a saturated solution of boric acid and thoroughly, but carefully, dried with a soft cloth or absorbent cotton. It is advisable to protect them with a cloth moistened with albolene or boric acid ointment between the nursings. If they are tender, they should be washed with a 50% solution of alcohol. If the nipples become cracked, the baby should be taken off the breast temporarily. A nipple shield may be tried and, if satisfactory, continued. As a rule, however, it causes more pain and does more harm than the baby does without it. The breast should be thoroughly emptied by expression and the milk given to the baby. Breast-pumps are unsatisfactory and are likely to keep the crack open. Rest, cleanliness, and a simple ointment, like boric acid ointment, are usually sufficient to heal the cracks. In some instances, however, it is necessary to touch the cracks with a 1% or 2% solution of nitrate of silver or even with a nitrate of silver stick.

If the breasts become full or tender, they should be supported by a breast binder and kept empty, preferably by expression. This is a far better procedure than massage and a breast-pump. If the milk contains no pus corpuscles, there is probably no risk to the baby, if the nursing is continued.

Breast-pumps and Nipple Shields.—The ordinary breast-pump, known as the English breast-pump, is usually satisfactory. There are other breast-pumps which the mother works by her own suction and by which the milk is collected in the nursing bottle in which it is given. These pumps are rather better than the English pumps. In general, however, it is inadvisable to use a breast-pump. It is impossible to empty the breast completely with them and when they are used, the breasts always finally dry up. *Manual expression* of the milk is much better.

The glass nipple shields with a rubber nipple are the best. A baby will often nurse better from a shield, if it is first filled with milk. If the nipples are small or retracted and cannot be enlarged or brought out and the baby takes a nipple shield well, they may be used. In general, however, it is inadvisable to use nipple shields when the nipples are cracked. It is far better to stop nursing temporarily, express the milk and feed it in some other way.

Care of Baby's Mouth.—It is important to keep a baby's mouth clean, because of the possible danger of infection of the nipples and breasts from it, if it is dirty. The surest and easiest way to get it inflamed is to wash it. If it is not washed, it almost never becomes either inflamed or infected. A swallow of water after nursing is all that is necessary.

Abnormal Breast Milk.—There is no doubt that human milk is the best food for infants. There is also no doubt that most human milk is

good milk and that most babies will thrive on any human milk. Nevertheless, it is also true that not all human milk is good milk. Some milks will not agree with any baby. Other milks will agree with one baby and not with another. Furthermore, the digestive powers of babies are not all the same. One baby can digest and thrive on a milk which does not suit another and what suits the second baby does not suit the first baby. It is impossible to determine from an analysis of a milk whether it will or will not agree with a given baby. This can only be told by trying it. Babies often thrive on a milk which, from its analysis, seems most unsuitable. The same baby will often thrive on different types of milk. While it is impossible to determine from an analysis of the milk whether it will or will not agree with the baby, it is, however, often possible, if a milk is not agreeing with a baby, to tell from the analysis why it does not. If a milk does not agree with a baby, the most common abnormality in the milk is an excessive amount of protein. The next most common abnormality is an excess of fat. There is almost never an excess of sugar.

Analysis of Breast Milk.—Great care must be taken in interpreting the results of an analysis of human milk, because the composition of the milk of the same woman varies, not only from day to day and from nursing to nursing, but also at different periods of the same nursing. An analysis is without value, therefore, unless all the milk is taken from the breast, or at least samples from the beginning, the middle and the end of the nursing. Even if the milk is properly taken, the results of a single analysis may also be misleading, because of the variations from day to day and nursing to nursing. Positive conclusions are only justified when the results of several examination are similar.

Types of Abnormal Milk.—Pediatrists in general pay less attention to the composition of human milk than they did a number of years ago. Many of them are, I fear, unacquainted with the different types of abnormality which occur in human milk and with the measures which can be taken to remedy them. Three general types of abnormality can be recognized. First, that in which all the elements are too high; second, that in which the fat and sugar are low and the proteins high; and finally, that in which the fat and sugar are very low and the proteins very high. The first type is most often found in indolent women of the wealthy classes who, having a good appetite and a good digestion, eat too much and too rich food, and do nothing. This type of milk is not seen very often, because women of this sort are usually too lazy to nurse their babies. An example of such a milk is one containing 5% of fat, 7.50% of sugar, and 2.60% of proteins. It is easy to correct this type of milk, if the mother will control her appetite, eat simple food and exercise. Unfortunately, however, it is usually very difficult to induce such women to change their habits.

The second type is most often found in women of the poorer classes who are compelled to work hard and do not have enough to eat. It is really a starvation milk. A characteristic milk of this type is one containing 1.75% of fat, 4.50% of sugar, and 2.50% of proteins. It is also easy to improve this type of milk by giving sufficient food and diminishing the work. The difficulty lies in getting the food and finding somebody else to do the work, when there is no money to pay for them.

The third type is usually found in the high-strung, overeducated and highly civilized women of the large cities, but may be found in neurotic women of any class or community. A sample analysis of this

type of milk is fat, 1%; sugar, 4%; proteins 3.75%. It is practically out of the question to improve this type of milk, because it is impossible to change the nervous make-up of women of this type.

The Normal Breast-fed Infant.—It hardly seems necessary to describe the normal breast-fed infant. It should gain from six to eight ounces a week during the first five months and from four to six ounces a week during the rest of the first year. Smaller, but regular, gains are not, however, necessarily abnormal. It should double its birth weight in the first five months and treble it at a year or a little later. It should not vomit, unless it is disturbed or shaken up soon after a feeding. It should have from two to four smooth, orange-yellow stools of the consistency of thick, pea soup daily during the first few months and from one to three similar stools of somewhat greater consistency during the rest of the first year. It should not cry, unless hungry or uncomfortable from some external cause. Its flesh should be hard and firm and its lips, cheeks and nails pink. It should sleep from twenty to twenty-two hours out of the twenty-four during the first two months and about sixteen hours a day during the latter half of the year. It should be happy when awake and active when given the opportunity.

The Abnormal Breast-fed Infant.—When a breast-fed baby, that is not gaining properly, has one or more normal stools daily and is not vomiting, it is almost certain that the failure to gain is not due to any defect in the quantity or quality of the milk. If there was a deficiency in the quantity, the baby would not have one or more normal stools daily. If the quality was abnormal, the baby would show signs of indigestion or would not have one or more normal stools daily. The source of the trouble must be sought elsewhere. It will then be found that the baby is being improperly handled in some way or that it has some disease. It may be that it is excited too much, that it doesn't get enough sleep, that it does not get enough fresh air or that it is not kept warm enough. Hidden tuberculosis, pyelitis and an insufficient supply of air as the result of adenoids are frequent causes of failure to gain.

When a breast-fed baby is not thriving as it should, and there is no cause outside of the food, the trouble may be in either the quantity or the quality of the milk. An excessive amount of breast milk seldom causes any trouble, because nature almost invariably diminishes the supply to just enough to satisfy the demand. Moreover, if there is an excessive amount of breast milk and the baby takes it, it usually simply vomits up the excess and has no further disturbance from it. If the baby does get too much breast milk and is disturbed by it, it is usually because it is nursed too often. When the intervals between the nursings are properly regulated, the difficulty usually stops at once. The symptoms are, of course, discomfort, vomiting, crying and an excessive number of stools of diminished consistency, containing small, soft curds. Sometimes they may be green and contain mucus. The baby usually continues to gain, but not as rapidly as it should.

If a baby is not gaining in weight, there are no symptoms of disturbance of the digestion and it is constipated, the food is deficient in quantity and quality or both. If the supply of food is sufficient to allay the pangs of hunger, the baby will not appear hungry, even if the food is entirely inadequate to enable it to gain. The only way to determine how much breast milk a baby is getting is to weigh it before and after each nursing for twenty-four hours. It is better to do this for two or three days than for one. It is useless to weigh the baby before and after only one or

two nursings, because the amount taken at single nursings varies so much. It is not difficult to determine the amount of milk in this way, because an ounce of milk weighs practically an ounce avoirdupois. It is not necessary to undress the baby before it is weighed. If the bowels have moved during the nursing, the diapers should not be changed before it is weighed. It is sometimes easier to weigh the mother before and after each nursing than it is to weigh the baby. What she loses in weight is, of course, the amount of milk which the baby has taken. In no other way can the amount of breast milk be accurately determined. It is impossible to judge anything as to the amount of milk secreted from the size of the breasts. Many large breasts contain but little glandular tissue and secrete but little milk, while many small breasts secrete considerable amounts of milk. It is impossible to determine the amount of milk by using a breast-pump, because the baby often gets much milk when the breast-pump gets but little. More can be told by expressing the milk, but even in this way not as much milk is obtained as by the baby. When a baby wakes up and cries for a time before each nursing period, the probability is that the supply of milk is insufficient. When a baby lets go of the nipple during the feeding and cries with anger or when it grabs the nipple, bites it and shakes it, the chances are that it is not getting much milk.

The quality of the milk can only be determined by chemical analysis. Great care must be taken, however, in estimating the value of such analyses, because of the difference in the composition of the milk at different nursings and the differences in the milk taken at various parts of the same nursing. Unless the milk of a whole day is taken, erroneous conclusions are very likely to be drawn. In general, however, when a baby is not gaining, has no disturbance of digestion and is constipated, if there is a sufficient quantity of milk, the difficulty is either that the milk is weak in all its constituents or that the percentage of fat is very low, while the other elements are approximately normal.

When there are symptoms of a disturbance of the digestion, such as colic, vomiting and abnormal stools, in addition to failure to gain properly, and there is not an excessive amount of milk, the difficulty is usually in the quality of the milk. The abnormality is most often an excess of fat or proteins, usually of proteins, rarely of sugar. An excess of fat often causes vomiting. It is shown most often, however, by the presence of small, soft curds in the stools. The typical soap stool is unusual in the breast-fed baby. When there is an excess of proteins, the stools are likely to be watery, more or less brownish in color, and contain mucus. They are often greenish and frequently contain mucus, whether the abnormality is an excess of fat or of proteins. The typical green, acid, irritating stool due to excessive fermentation from an excess of sugar is, however, seldom seen. The only way in which the abnormality in the composition of the milk can be definitely determined, however, is by an analysis of the breast milk, due regard being paid to the possibility of error in the analysis, unless it is properly done.

Modification of Breast Milk.—A number of years ago it was thought that much could be done to modify the character of breast milk. The general feeling at present is that little or nothing can be done to modify it. It seems to me that, as usual, the truth lies between the two extremes. I am confident that not only the quantity, but also the quality of breast milk, can be changed by proper methods and treatment.

It should be remembered in the first place that lactation is, or should be, a physiologic, not a pathologic, process. A nursing woman is not ill

because she is nursing. She is simply doing what she is intended to do. She should, therefore, lead the same sort of life when she is nursing that she does when she is not nursing, provided her manner of living is a normal one. She should be careful, however, not to overdo or to get overfatigued, as there is undoubtedly a certain amount of additional strain in nursing. Many women, however, are better and stronger when they are nursing than at any other time. Her diet should be the same as that to which she is accustomed when she is not nursing. One writer has summed up the situation by saying that she should eat whatever agrees with her husband. This statement is probably a little exaggerated, but there is no reason why she should not eat anything which does not disturb her digestion. She should, as when not nursing, avoid articles of food which do not agree with her. There is very little in the old theory that a woman should not eat acid fruits or vegetables with strong flavors on the ground that they are likely to disturb the baby's digestion. Nevertheless, it is true that sometimes when a given woman eats a certain article of food her baby is upset, and that it is upset whenever she eats it. It is impossible, however, to know in advance what this article of food will be. The thing which causes trouble in one baby, when the mother eats it, does not cause any trouble in another baby when eaten by another mother, and something else which did not disturb the baby of the first mother will upset the baby of the second mother. The nursing woman should, therefore, eat a reasonable general diet, avoiding articles of food which she knows are likely to disturb her digestion. If her baby's digestion is upset, she should think over what she has eaten in order to find out if there has been anything new or unusual in her diet. If there was and the baby is upset when she eats it again, she should cut this article of food out.

Modification of Quantity of Breast Milk.—In this connection it must never be forgotten that nature tends to accommodate the supply of breast milk to the demand. If little is taken, little is produced; if more is taken, more is produced. It is often possible, for example, for a woman to nurse two, or even three, babies at one time. If this woman had only one baby to nurse, there would not be more than enough milk for that baby. There is, of course, however, a limit to the amount of milk which an individual woman can produce and this limit may be either high or low. It is, however, almost always higher than is usually supposed. It must also never be forgotten that the best stimulant to the secretion of milk is the thorough emptying of the breasts. Nothing can take the place of it. There is nothing which empties the breasts so completely as a hungry baby. A breast-pump does not thoroughly empty the breasts and eventually dries them up. The breasts can, however, be well emptied by manual expression. The thumb and forefinger should be placed, one on either side of the nipple, just outside or a short distance beyond the border of the areola. Pressure should then be exerted downward and forward. It is not necessary to use force or cause pain. It is useless to go far back on the breast. The old-fashioned massage did more harm than good. This procedure is similar to that carried out in milking a cow, the object being to empty the reservoirs in the ampullae, into which the excretory ducts from each lobe empty, not to push the milk from the lobes into the ampullae. If they are empty, the milk flows in and fills them again. The milk should be expressed at the same hours at which a baby would nurse. If the baby is feeble and cannot empty the breasts itself, nursing should be followed by expression. It is said that

lactation can be reëstablished by regular nursing or expression, even three or four months after it has ceased, and that but little difficulty is experienced after three or four weeks. Judging from my own experience, this point of view is decidedly optimistic.

The next best stimulants to the secretion of milk are a liberal general diet and a normal life. Increasing the quantity of liquid in the diet increases the quantity of the milk to a certain extent. It is useless, however, for a woman to take more than a quart of extra liquid daily. A pint will probably do as well. More than this disturbs her digestion, spoils her appetite for other food or makes her grow fat. This extra liquid should not be too rich. It should be taken chiefly for its action as a liquid, not as a food. Milk and cocoa shells are probably the best drinks. Chocolate, egg-noggs and rich drinks of this sort should not be taken. They destroy the appetite for solid food and disturb the digestion. Gruels are said to have a certain action as galactagogues. I confess that I am skeptical. Malt liquors are also said to have a specific action. This is also doubtful. It is better not to use them, because they are very likely to disturb the digestion and almost invariably fatten the mother. There is no danger to the baby from their alcoholic content. There are no drugs which, taken internally or applied externally, can increase the flow of milk to any appreciable extent.

It is seldom necessary to diminish the quantity of milk. If the breasts are not thoroughly emptied, nature quickly diminishes the amount secreted and fits the supply to the demand. A diminution in the amount of food and liquid taken will also usually quickly diminish the amount of milk secreted. External applications almost never do any good and should never be used. If it is necessary to reduce the amount of milk quickly, the bowels may be freely opened.

Modification of Quality of the Breast Milk.—When the total solids of the milk are all high as the result of overeating and lack of exercise, they can be reduced by regulating the diet and increasing the exercise. When they are low as the result of starvation and malnutrition, they can be increased by proper food and care. They can also be influenced, to a certain extent, by varying the intervals between nursings. Lengthening the intervals diminishes the total solids, shortening the intervals increases them.

Fat.—The amount of protein in the food has no effect upon the amount of fat in the milk. If the mother is underfed, an increase in the fat in the food will temporarily cause an increase in the fat in the milk. If she is not underfed, an increase in the fat in the food does not increase the fat in the milk. An excessive amount of fat in the milk is most often due to too large an amount of food in general and can be diminished best by cutting down the food as a whole. An insufficient amount of fat in the milk is usually due to malnutrition. It can be best increased by improving the general condition, by proper food and regulation of the life.

When breast milk is low in fat, but otherwise of good quality it is easy to increase the percentage of fat by giving the baby cream at the time of the nursing. If, for example, a baby is taking four ounces of breast milk containing 2% of fat, the fat may be raised to nearly 4% by giving one half ounce of gravity cream (16% fat) or one quarter ounce of 32% cream with the breast milk. The cream may be given before, after or during the nursing. It is best to give it during the nursing. It may be given with a spoon or a dropper. Sometimes a

baby takes it better, if it is given from a dropper introduced into the mouth beside the nipple while the baby is nursing.

Sugar.—It is impossible to influence the percentage of sugar alone in milk in any way. It varies somewhat, however, as do the other elements, with the general condition of the mother.

Protein.—Hoobler found that the percentage of protein in breast milk can be changed to a certain extent by variations in the diet. It can be increased by increasing the proportion of protein in the food in relation to that of the combined fat and carbohydrate and by increasing the proportion of animal to vegetable protein. It can be diminished by giving a diet containing a relatively large amount of fat and carbohydrate in proportion to protein and by giving most of the protein in the form of vegetable protein. He found that there should be at least one part of food protein and six parts of food carbohydrate and fat for the best production of milk. He also found that the protein of milk is the most economical, available and affective form of protein for the production of protein in human milk. A vegetarian diet, that is, one made up of fruits, cereals and vegetables, does not give sufficient available protein and causes a severe drain on the tissues of the mother. The addition of nuts to the diet helps, however, to supply the deficit.

The most common cause of an excessive amount of protein in human milk is nervousness. If the mother's nervous condition can be overcome, the percentage of protein will diminish. The amount of protein can also be diminished by exercise, but, if the exercise is excessive and causes overfatigue, it will be increased. Overfatigue is also, therefore, one of the causes of high protein in human milk. If this is the case, rest diminishes the amount of protein.

Mixed Feeding.—When a woman does not have enough milk to satisfy her baby and it is impossible to increase the supply sufficiently by the measures already detailed, the baby should not be weaned, but should be given enough artificial food in addition to the breast milk to make up the deficit. As a general rule, it is much better to give some of the artificial food at each feeding, after the baby has emptied the breasts, than to substitute artificial feedings for breast feedings. The amount to be given can best be determined by weighing the baby before and after every feeding until the average amount which the baby gets from the breast has been determined. It is usually satisfactory, however, to give the baby as much as it wants of the artificial food after each nursing. If, however, the supply of breast milk is almost sufficient, the baby may be given one, or possibly two, feedings of the artificial food in place of the same number of breast feedings. No more than two nursings should ever be omitted, however, because, if they are, the supply of milk will certainly diminish further because of lack of stimulation of the breasts.

In deciding what artificial food to give a baby no attempt should be made to imitate the composition of the mother's milk. The composition of the artificial food should be decided on general principles, due consideration being paid to the age and apparent digestive capacity of the baby. An analysis of the breast milk is, however, sometimes of assistance in determining the composition of the artificial food, because, if there is a marked deficiency of one element in the breast milk, this deficiency can be made up for by having this element relatively high in the artificial food.

Weaning.—A baby should never be taken off the breast unless it is absolutely necessary. It should never be weaned because there is not

enough milk to satisfy it. The breast feeding should be continued and the deficiency made up with an artificial food. A baby should never be weaned simply because it has the colic, vomits or has abnormal stools, until everything has been done to improve the quality of the breast milk. Even if the quality cannot be improved, it should not be weaned, if it is gaining and thriving in other ways. It is almost certain to do worse on an artificial food than on the breast milk. It is especially important not to wean babies in the early weeks of life on account of evidences of indigestion, because the breast milk often improves and all the symptoms disappear as soon as the mother is well and strong again and back on her usual routine. A baby ought not be weaned hastily on account of cracked nipples, because these can almost always be cured with a little time and patience.

The appearance of menstruation is not a cause for weaning. As a matter of fact, more women menstruate during lactation than do not. Furthermore, the changes which take place in the milk at this time are no greater than those which occur at many other times during lactation. In most instances, the baby shows no evidences of disturbance of the digestion during the menstruation. If it does, they are usually nothing more than a little colic and a few green stools. If they are more marked, the baby can be taken off the breast for a few days and fed on an artificial food, being put back on the breast after the menstrual period is over. The breasts should, of course, be kept active during this time by expression of the milk.

Pregnancy is, in my opinion, an indication for weaning. There are undoubtedly some women that are able to nurse continuously, having a baby on the breast constantly. However this may be, it is impossible for the average woman to satisfactorily nourish three individuals at one time—herself, a baby on the breast and another *in utero*. Some one is almost sure to suffer, most often the baby on the breast. While it is inadvisable for a woman to nurse her baby when she is pregnant, the discovery of pregnancy does not mean that the baby on the breast should be weaned at once. If it is not well at the time, the nursing should be continued until it is in good condition again. At any rate, it should never be weaned quickly. Pregnancy does not make the milk poisonous for the baby. It simply in time diminishes it in quantity and weakens it. Weaning because of pregnancy is advisable, therefore, simply because it is to the advantage of all concerned.

Acute disease in the mother may or may not be an indication for weaning. If the mother is seriously ill, the milk usually diminishes or disappears, so that there is no question but what the baby must be fed in some other way. If the mother is not seriously ill, it is usually advisable to keep the baby on the breast. If the mother has a contagious disease and is not seriously ill, it is usually safe to keep a young baby on the breast, because young babies are, to a large extent, immune to infection with these diseases. If the baby is more than six months old, its immunity is less and it is wiser to keep it away from the mother, unless it has already been thoroughly exposed to the disease before its nature was realized. Whatever the disease, if the mother recovers, it is advisable to attempt to reëstablish the secretion of breast milk by putting the baby to the breast and by expression.

The development of a chronic disease in the mother is usually an indication for weaning. How soon the baby should be weaned must be decided, however, on the conditions in the individual case, that is, on the

severity of the disease in the mother and the general condition and need of the baby. In some instances, although the baby is thriving, the strain of nursing exhausts and pulls down the mother. In such cases, the decision as to weaning must likewise depend on the conditions in the individual instance. The condition of the mother, on the one hand, must be weighed against the needs of the baby on the other, always bearing in mind that it is a mother's duty to sacrifice herself, at any rate up to a certain point, for her baby. The older the baby, the less dependent it is upon breast milk. Weaning is justifiable, therefore, for slighter disturbances of the mother's health after the first few months than it would be earlier.

Nowadays it is seldom necessary to decide when to wean a baby, because the breast milk gives out before the time at which the baby would naturally be weaned. In such instances, however, the milk usually diminishes slowly and weaning goes on gradually without any disturbance. If the supply of milk continues sufficient in quantity and quality, it is advisable to wean a baby when it is about ten months old. It is not advisable for it to nurse more than a year, as babies seldom thrive on breast milk alone for more than a year, and usually not as long. If a baby is doing well on the breast and reaches the proper age for weaning in the summer, it is usually better to continue the nursing through the summer and wean it in the autumn than to take it off the breast in the summer. It is better, however, to wean it in the summer, even if it is then ten or twelve months old, than to wean it in the spring so that it will not have to be weaned in the summer, as used to be done. The reason that it used to be considered so dangerous to wean a baby in the summer was that the milk supply was then so poor that it was very difficult to get a suitable food for the baby. Now that pure milk can be obtained, the chief danger in putting a baby on an artificial food in the summer has been removed, so that is better to give the baby the advantage of a few months more breast milk than to wean it in the spring. Furthermore, the older a baby is when it is weaned, the better able it is to take care of an artificial food.

Babies should always be weaned slowly, if this is possible. It is much easier for the mother and the baby is much less likely to be made ill by the change to artificial food than when it is weaned quickly. If it is made ill, it can easily be put back on the breast again. If it is weaned suddenly and the milk goes, as it usually does, it cannot get anything from the breasts at first, although it may later. Weaning is much easier, if the baby has been in the habit of taking one bottle a day from the beginning of nursing. If a baby has had one or more bottle feedings daily, there is usually no trouble in weaning it gradually and neither mother nor child is disturbed. Furthermore, it is known what artificial food agrees with the baby. If the baby has never had anything but the breast, it is often difficult to make it take the bottle. Sometimes it will refuse everything except the breast and cannot be made to take an artificial food in any way. I have seen babies who had never had anything but the breast and who had been weaned quickly that would have starved to death, if they had not been fed through a tube. When a baby has to be weaned suddenly, whatever the reason, it is best to separate the baby from its mother. Whether it is or not, some other person must give it its food, because it expects to get breast milk and nothing else from its mother, while, at any rate, it does not expect to get breast milk from anyone else. A little baby should be given a bottle. An older baby should be fed from a glass

or with a spoon. If a baby refuses to take its food after reasonable urging, it should be allowed to go hungry until the next feeding time. On no condition should it be fed except at the regular hours. Most babies get hungry enough to eat after one or two days and take what is offered to them. Occasionally, however, a baby has to be forced to take food or even fed through a tube to save its life.

When a baby has been taking one artificial feeding daily, there is no difficulty in deciding what food to give it. The same food is continued, the number of artificial feedings simply being increased. When a baby that has been entirely breast-fed can be weaned slowly, it is safe to give it a fairly strong food. If it is more than nine months old, it is usually safe to start it on dilutions of whole cow's milk, to which starch may be added. When a baby is weaned suddenly, it should always be given a weaker mixture than a baby of its age would naturally take, in order to avoid, if possible, setting up a disturbance of the digestion by too strong a food. The strength of the mixture must necessarily depend very largely on the age and general condition of the individual infant. The general principles of artificial feeding should be followed in selecting the food.

WET NURSES

There is no doubt that the most suitable food for an infant that is so unfortunate as not to be nursed by its own mother is the milk of another woman. As a matter of fact, the milk of some other woman is not infrequently better for a baby than that of its own mother, if she is nervous and feeble and the stranger placid and strong. Owing to the improvement in the milk supply and the advances which have been made in the knowledge of artificial feeding, the need of wet nurses is not as great as it was years ago. Most normal babies can now be fed satisfactorily without breast milk. Nevertheless, there are many feeble and debilitated babies, premature babies and babies that have had serious disturbances of the digestion that will die or, at any rate, not thrive properly for a long time unless they have human milk. No artificial food can take the place of it for such babies. I have made it a rule for many years not only never to allow a baby to die, but never to allow a baby to get into a condition in which I fear that it may die of disturbances of nutrition or of diseases of the digestive tract without getting it a wet nurse, or at least breast milk, provided they can be procured. If the parents are unwilling to get a wet nurse or breast milk, I refuse to take any further responsibility and resign from the case.

It is true that wet nurses are often not an unmixed blessing in a family. They realize how important they are and not infrequently take advantage of it. They are, however, very much like other people—good, bad and indifferent—and, like other people, how they conduct themselves depends very largely on how they are treated. Even if they do cause trouble in the household, a family should be willing to put up with a great deal of annoyance and inconvenience for the sake of saving their baby's life. Domestic troubles are not to be compared with the anxiety dependent on the severe illness of a baby or with the grief which follows its loss.

Every mother dislikes to have another woman nurse her baby. She should, however, in the first place, appreciate the fact that, if she was fulfilling her duty to her baby, a wet nurse would not be necessary. In the second place, she should be not only willing, but glad, to sacrifice her own feelings for the good of her infant. There is, of course, no possi-

bility of the transference of mental, moral or physical characteristics from the nurse to the baby. If there was, it would be far better for many babies to have wet nurses than to be nursed by their own mothers. It makes no difference to the baby what is the color, race, creed, disposition or moral character of the nurse, provided her milk is of good quality and sufficient in quantity.

It is sometimes said that it is wrong to employ wet nurses, because it is immoral to deprive one baby of its natural nourishment and give it to another. This objection is not well-founded, because women do not go out as wet nurses for pleasure, but because they are compelled to support themselves and their babies. They can make better wages as wet nurses than they can in any other way and, if they board their babies out, they are able to board them in better places and to save money for the future. If, as is always advisable, they have their babies with them, they can take care of their babies, become fond of them, and are much less likely to go astray in the future. It is often an advantage to the foster baby for the nurse to have her own baby with her, because she is happier and more contented. Furthermore, in the beginning, the foster baby is often not strong enough to properly empty the breasts and the nurse's own baby keeps up the supply of milk for it. In many instances, the nurse is able to feed both the foster baby and her own. The wet nurse must always be made to understand, however, that she is hired to nurse the foster baby and that the foster baby has the first claim on her milk. Wet nurses are, of course, expensive. They are not nearly as expensive, however, as doctors and trained nurses, and after the wet nurse comes the trained nurse disappears and the doctor makes very few visits.

Qualifications of a Wet Nurse.—A wet nurse should be healthy and free from syphilis, tuberculosis and other chronic diseases. No woman should be accepted as a wet nurse without a complete physical examination by a competent physician and a negative Wassermann test. It is just as much a physician's duty, moreover, to be certain that the foster baby has not syphilis as it is that the wet nurse has not syphilis. It is just as wrong to give syphilis to a wet nurse as it is for a wet nurse to give syphilis to a baby. It is impossible to determine from the general appearance of a woman or from the size, shape or feeling of her breasts whether she has or has not a good supply of milk. The ease with which milk can be expressed from the breast is also unreliable as a guide, because a baby can often obtain much milk from a breast from which but little milk can be expressed, while in other instances, where there is little milk, it can all be easily expressed. The only way in which the quantity of milk which the breast is secreting can be positively determined is by weighing the wet nurse or her baby before and after each nursing for twenty-four hours. Next to this, is the appearance of her baby. If it is thriving, it is evident that it gets a sufficient supply of milk and that this milk is of good quality. Incidentally, it is of much importance to be sure that the baby which is brought for inspection is really the wet nurse's own baby. It is useless to analyze the milk to determine whether it will be suitable or not, partly because of the variations in the milk and partly because it is impossible to know in advance whether a given milk will or will not agree with a given baby. The composition of human milk being practically the same from the end of the colostrum stage until nearly the end of lactation, it is not necessary for the foster baby and the nurse's baby to be of the same age. It is not advisable, however, if the foster baby is young, to get a wet nurse whose baby is old, because her

milk is likely to give out before the need for it has ceased. Otherwise, there is no objection. It must also be remembered that when a young, feeble or premature baby is put to a breast which is thoroughly established, it will not be able to empty the breasts thoroughly and the milk will soon diminish or dry up. Many a good wet nurse has been spoiled in this way. It is a great advantage, therefore, under these conditions for the wet nurse to have her own baby with her to empty the breasts. If she has not, the breasts should be thoroughly emptied by expression. Furthermore, a young baby may be upset by getting too much milk from a well established breast. This can be avoided by weighing the baby during nursing and diminishing the duration of the nursing as necessary.

Management of Wet Nurses.—A wet nurse should be treated kindly and considerately. It must never be forgotten that she is in a very trying position. On the other hand, she should not be babied, petted and pampered. She should be given the sort of food to which she is accustomed and given the sort of work to do which she has been in the habit of doing. If a woman that has been in the habit of doing hard manual labor is allowed to sit about and do nothing and given large amounts of rich food, she is sure to become ill both mentally and physically. Her milk will suffer at the same time. On the other hand, if a woman that has been in the habit of leading a sedentary life and of having good food is made to do hard work and put on a coarse diet, she also will be upset and her milk will suffer. Attention must be paid to all of these details.

Methods of Procuring Wet Nurses.—A wet nurse can always be found, if she is sought for with sufficient energy and vigor. In Boston there is a Directory for Wet Nurses where a considerable proportion of the women who wish to be wet nurses go. They are examined there and the Wassermann test done. The quantity and quality of their milk are also determined. They are not sent out unless they are healthy and their milk good. When they are through with one case they return to the directory and wait for another. They take their babies with them. The directory takes a portion of their wages in order to support the institution. This is the best solution of the problem. It large cities wet nurses can usually be obtained at the various maternity homes. In smaller places they can usually be found through physicians and the various public health and district nurses. In smaller communities, while it may be harder to find a wet nurse locally, there is almost always some woman who has more milk than her baby needs and who is willing to help out in an emergency.

If it is impossible to secure a wet nurse or it is preferred not to have one in the home, it is often possible to collect the milk from one or more women and feed it to the baby with a bottle. In many ways this is easier and more satisfactory. The Directory for Wet Nurses in Boston will keep the wet nurse there, express her milk and have it ready for delivery daily. Incidently there is no harm in giving the mixed milk from several women to one baby. In feeding with cow's milk, herd milk is preferred for many reasons to that of a single cow. It seems reasonable to apply the same arguments to the feeding of babies on breast milk. The experiments at the Boston Floating Hospital have shown moreover, that breast milk may be kept for several days under suitable conditions, in the same way as cow's milk. Breast milk may also be frozen and kept for a long time, being thawed out and used as necessary. Breast milk may also be dried and kept in powdered form in the same way as cow's milk.

ARTIFICIAL FEEDING

CHEMISTRY AND BIOLOGY OF COW'S MILK

The colostrum of cow's milk is never used in infant feeding. Lactation is well established and the milk suitable for use after a week. The color of cow's milk is white, with a more or less yellow tinge, which varies according to the amount of fat, the size of the fat globules and the amount of coloring matter in the fat. Pure, fresh milk has practically no odor. If it has an odor, the odor is that of manure, not of milk. The taste of cow's milk is not as sweet as that of human milk. The specific gravity varies materially in different breeds of cattle and according to the amount of fat in the milk. It varies between 1.027 and 1.040. The reaction is amphoteric to litmus paper, but, like human milk, it gives an acid reaction to certain reagents and an alkaline to others. The acidity becomes less for a short time after milking, because of the loss of carbon dioxide. The reaction soon becomes acid to litmus paper, however, as the result of the production of lactic acid from the decomposition of milk sugar by bacteria. The average composition of cow's milk is usually given as fat 4%, milk sugar 4.50%, protein 3.50% and ash 0.70%. The caloric value of cow's milk necessarily varies with the amount of fat which it contains. The figure which is usually adopted as the average is 670 calories per litre.

Fat.—The percentage of fat varies decidedly in different breeds of cattle, averaging from about 3.25% in the milk of Holsteins and 3.75% in that of Ayrshires to 5.40% in Guernseys and 5.80% in Jerseys. The fat droplets are, on the average, larger than in human milk. The proportion of fatty acids is also larger than in human milk. It is larger also in the milk of Jerseys and Guernseys than in that of Holsteins and Ayrshires. The yellow color of the fat is due chiefly to carotin, but in part to xanthophyll, which are derived from the cow's food. The fat rises more quickly and more completely in the milk of Jerseys and Guernseys than in that of Holsteins and Ayrshires. Boiling milk interferes with or prevents the rising of the fat.

Lactose.—The sugar in cow's milk is lactose. The extreme variations are said to be 2.11% and 6.12%. The average is 4.60%.

Protein.—The total nitrogenous compounds vary, according to different authorities, between 3.20% and 3.80%. The proteins are of two classes, soluble and insoluble, that is, albumins and casein. The principal soluble proteins are lactalbumin and lactoglobulin. Lactalbumin is much more abundant than lactoglobulin. The important ones, in relation to infant feeding, are casein and lactalbumin. There is much difference of opinion as to the relative proportions of casein and albumin. The average, according to Van Slyke, is 3.6 parts of casein to 1 part of soluble protein.

Casein.—Casein has the properties of a weak acid and occurs in milk in combination with calcium in the form of calcium caseinate, probably as tricalcium caseinate. It is soluble in water. Casein is precipitated in fine flakes by very small amounts of dilute acids, but large amounts produce a compact curd. The solidification of milk, which takes place when it sours, is due to the action of lactic acid, formed from the sugar by bacterial action, on the casein. Casein is coagulated by rennin in the presence of soluble lime salts. Coagulation does not occur when the reaction is alkaline, but may when it is neutral or acid. The dilution of milk with water delays the coagulation of milk by rennin, because the

proportion of soluble calcium salts is decreased. The addition of calcium chloride or of a free acid to milk diluted with water hastens the time of coagulation and increases the amount of casein coagulated. The addition of foreign inert matter, like starch or sawdust, hastens rennin action. Rennin coagulates milk most completely at from 106° to 108° F. and less completely at temperatures above and below this point. Milk heated about 160° for a considerable length of time coagulates less rapidly than unheated milk. The coagulum of heated milk is more flocculent. Boiled milk is not coagulated normally, if at all, by rennin. The addition of the soluble salts of calcium, barium and strontium favor or hasten coagulation, while the salts of ammonium, sodium and potassium retard or inhibit coagulation. Rennin splits the casein molecule into two similar molecules of paracasein. Paracasein, like casein, possesses acid principles, but has only one half the combining power of casein. Calcium paracaseinate is less soluble than the calcium caseinate from which it is formed, and, therefore, when milk curdles, it is precipitated. Coagulation, therefore, is really the result of a change in solubility.

Sodium caseinate is very soluble in water, as are also the caseinates of the other alkalies. Rennin splits sodium caseinate into two molecules of sodium paracaseinate. Sodium paracaseinate is soluble. Coagulation, therefore, does not occur. This is the explanation of the reason why the addition of sodium citrate to milk makes the casein easier to digest. When sodium citrate is added to milk, the sodium replaces the calcium, changing calcium caseinate to sodium caseinate. The addition of rennin changes the sodium caseinate to sodium paracaseinate which is not coagulated. 1.7 grains of sodium citrate to one ounce of milk is sufficient to prevent coagulation.

The action of lime water in delaying or preventing the coagulation of casein is different. A basic calcium caseinate is formed which is not acted upon by rennin. In practice, the addition of lime water to milk may increase the alkalinity to such a point that the stomach will not secrete enough acid to make the stomach contents neutral or acid.

Lactalbumin.—The lactalbumin is present in the whey which remains after the coagulation of the casein. The whey also contains lactoglobulin and extractives. These are, however, small in amount and unimportant. Whey contains, roughly, from 0.90% to 1.00% of soluble proteins.

Salts.—The ash of cow's milk varies between 0.60% and 1.00%, the average being 0.70% or 0.75%. The relative proportions of the different salts in cow's milk are of little importance to the clinician in everyday work. They are more abundant in cow's milk than in human milk. There are more of them than can possibly be utilized by a baby. The proportion of phosphorus in organic combination is only about one third as much in cow's milk as in human milk. There is an insufficiency of iron for the infant's needs in cow's milk as well as in human milk.

Bacteriology.—Cow's milk, when freshly drawn, contains bacteria. These bacteria do not get into milk from the blood, but from the outside through the teat ducts. They are most numerous in the milk first drawn, but there are usually some in the milk which is drawn later. The vast majority of the bacteria in milk enter it, however, during or after milking. The number which milk contains depends upon the care taken at the time of milking and how it is guarded against contamination afterward. It hardly seems necessary to go into the details of the production of clean milk. It is now common knowledge that cows should be washed before they are milked, that the milker's hands should be clean, that clean

utensils should be used, that the small-top pail is better than the open milk pail and that the milk must be kept in sterilized containers, protected from exposure to the air, and cold.

The bacteria which are present in the milk in the udder are non-pathogenic. Those which enter the milk after it leaves the udder may be either pathogenic or non-pathogenic. In the vast majority of instances, however, they are non-pathogenic. The most common organisms are those which produce lactic acid from milk sugar. There are more than one hundred varieties of organisms which may cause lactic acid fermentation and the souring of milk. The commonest of these organisms which sour milk are, however, the B. lactis acidi and the streptococcus lacticus. Less common is the B. lactis aërogenes. Colon bacilli, which get into the milk chiefly in manure, are facultative bacteria and may also cause souring. Most of the organisms which sour milk are harmless to human beings. If sour milk is to be used as a food, however, it is far wiser to inoculate the milk with organisms known to be harmless than to use ordinary sour milk. As a rule these lactic acid organisms overgrow and crowd out other organisms of the putrefactive type. After the milk sugar has been used up by fermentative organisms, these organisms may flourish and produce putrefactive changes. Various yeasts also develop on milk which has soured.

Various discolorations of the milk may be produced by certain specific organisms. Certain other organisms may impart characteristic tastes to the milk. As a rule, however, peculiar tasting milk is due to the food which the cow has eaten rather than to bacterial action. Milk may also be slimy, stringy or soapy as the result of bacterial action, as in garget. All of these changes are relatively unimportant, however, as they are easily recognized and no one would be foolish enough to use milk which shows such changes. The most important of the organisms pathogenic for man which are found in cow's milk are the tubercle bacillus and the streptococcus of septic sore throat. Tubercle bacilli are probably almost never present in the milk in the udder of a tuberculous cow, unless the udder itself is diseased. They enter the milk in manure, dust and the pulmonary secretions of the cow. Contamination of the milk with human tubercle bacilli is always due, of course, to infection from the sputum or discharges of those who handle the milk. There is great difference of opinion as to the importance of tuberculous infection in early life through milk. This question has been discussed elsewhere. Whatever its importance, it can be stopped by the elimination of tuberculous cows and the pasteurization of all milk from cows which are not known to be free from tuberculosis. The streptococcus of septic sore throat enters the milk, of course, after it has come from the cow, in the discharges from the nose and throat of some person ill with this disease. The streptococcus associated with inflammation of the udder of the cow may or may not be harmful to man. The streptococci which are "normally" present in cow's milk are not pathogenic. The danger of infection from streptococci in the milk can be avoided by proper care in the handling of the milk, proper examination of the cows and pasteurization. Other pathogenic bacteria which may contaminate milk and cause epidemics are the typhoid bacillus, the diphtheria bacillus, the streptococcus of scarlet fever and the dysentery bacillus. These organisms must evidently enter the milk after it has been drawn, as the result of the carelessness of those who handle it. Other organisms which are sometimes found in milk are anthrax bacilli, actinomyces, the organism

which causes foot-and-mouth disease, the B. lacti morbi, which causes the "trembles" in cattle and is very fatal in man, and the B. abortus, which fortunately is not pathogenic to infants.

When milk stands, bacteria settle in part to the bottom and in part rise with the fat globules. In consequence, cream usually contains many more bacteria per c.cm. than the original milk. This is not true, however, of cream obtained by centrifugalization.

The growth of bacteria may be hindered by the addition of preservatives to the milk. When formaldehyd is present in milk, the addition of commercial sulphuric acid to equal parts of milk and water shows a purple line at the junction of the acid and the milk. Boric acid or borax can be detected by adding a drop or two of hydrochloric acid to a few drops of milk in a white dish and then several drops of a saturated alcoholic solution of turmeric. When the dish is heated for a few minutes, a pink or dark red color appears, if boric acid or borax are present. A dark blue-green color appears when the dish is cooled and a drop of ammonia is added. The addition of a few drops of a 1% solution of rosolic acid to equal parts of milk and alcohol gives a red rose color, if sodium bicarbonate is present.

Enzymes or Ferments.—There is some doubt as to whether the enzymes or ferments present in cow's milk are inherent or the result of bacterial activity. It is doubtful whether they have any influence on the digestibility of the milk. Probably, they have not. They are destroyed by pasteurization.

Pasteurized Milk.—There are three methods of commercial pasteurization in common use: the flash method, the holding method and pasteurization in the bottle. The flash method consists in momentarily heating the milk to a temperature of approximately 170° F. by allowing it to flow in a film over heated metal pipes or coils and then at once chilling it. This method has been repeatedly shown to be unreliable and ought not to be used. The holding method consists in heating the milk to between 140° F. and 155° F. and keeping it at approximately this temperature for from twenty minutes to an hour. At present it is believed that under commercial conditions pasteurization should be at 145° F. for at least thirty minutes. Pasteurization at this temperature and for this time in sealed bottles is preferable to pasteurization in bulk. No chemical changes can be detected in milk pasteurized at 145° F. It is probable that most of the enzymes in milk are destroyed at this temperature. This fact is of no importance, however, because there is no evidence to show that the enzymes in cow's milk are of any use to a baby. The fat soluble vitamin A and the water soluble vitamin B are not affected by pasteurization. The water soluble vitamin C is, however, destroyed. Pasteurization has no effect on the digestibility of the milk. It destroys non-spore-bearing microörganisms, including the tubercle bacillus, and bacterial toxins. It does not change the taste, odor, or color of the milk.

It must never be forgotten that the pasteurization of milk does not do away with the necessity of taking care of it and keeping it cold. It is just as important to keep pasteurized milk cold as it is to keep raw milk cold, because pasteurization simply diminishes the number of microörganisms. It does not destroy them entirely. It was supposed, at one time, that the pasteurization of milk favored the growth of proteolytic bacteria and that, in consequence, pasteurized milk putrefied instead of soured. It is now known, however, that pasteurized milk sours in the same way as raw milk and that the relations between the bacteria

are not changed. It is evident, therefore, that, unless it is possible to procure a clear milk for babies and take care of it properly, it is wiser to pasteurize it than to give it raw.

It is sometimes necessary to pasteurize milk at home. There are two good pasteurizers on the market: that sold by the Walker-Gordon Laboratory Co. and the one designed by Dr. R. G. Freeman of New York. It is not necessary, however, to have any special apparatus for the pasteurization of milk in the home, as any dish of sufficient size and depth will serve. Each feeding should be placed in a separate, clean, boiled bottle. The bottle should then be tightly stoppered with absorbent cotton and placed in a pail or dish of cold water, the water in the dish being at the level of the milk in the bottles. The dish should then be placed on the stove and heated until the thermometer suspended in the water reaches 145° F. The dish and its contents should then be taken off the stove and covered with a blanket. It should be allowed to stand for thirty minutes. The bottles should then be taken out, cooled quickly, preferably in running water, and kept in a cold place until used.

Boiled Milk.—When milk is boiled, a well marked scum, or pellicle, develops on the surface. It is due to the disassociation of the casein compounds as the result of drying. Changes in the taste and smell develop. Prolonged boiling changes the color toward brown because of the caramelization of the sugar. Boiling materially delays, or entirely prevents, the rising of cream. Numerous minor chemical changes take place in the milk. Some of the calcium and magnesium salts are precipitated, as is a part of the citric acid. There is a diminution in the amount of organic and an increase in that of inorganic phosphorus. There is also a certain amount of decomposition of the compounds of casein. It is doubtful, however, whether any of these changes make the milk materially less valuable for the infant. Coagulation of the casein by rennin is wholly or partly prevented. The action of pepsin and pancreatin is also interfered with. The curds produced by the action of acids and rennin are softer and more flocculent than those in raw milk. The soluble albumins are entirely precipitated. The ferments and the bactericidal power of the milk are destroyed by boiling. Non-spore-bearing organisms are destroyed by boiling for one or two minutes. The spores of peptonizing bacteria may, however, withstand boiling for an hour. Animal experiments show that all young animals do better when fed on the raw than on the boiled milk of their own species. On the other hand, they appear to thrive somewhat better, if the milk of a foreign species is given boiled instead of raw. What little experimental data there is goes to show that the utilization by babies of raw and boiled cow's milk is essentially the same. Boiling milk destroys vitamin C, does not injure B, and probably does not affect A. When milk is boiled in the home, it must be remembered that it must be really boiled in an open dish and that heating in a double boiler, even if the water is boiling in the lower part, does not really boil it.

Clinically, milk should be boiled for two reasons: to destroy bacteria and to make the digestion of the casein easier by preventing the formation of large, casein curds. It must never be forgotten that vitamin C is destroyed and that, in order to prevent the development of scurvy, some antiscorbutic must be given, if boiled milk is used continuously for more than a few weeks.

Frozen Milk.—Clinically, there is no doubt that babies and young children are often upset, sometimes seriously, by taking milk which has

been frozen. Every one knows, moreover, that the gross appearance of milk and cream which have been frozen and thawed is different from that of milk and cream which have not been frozen. It is known that the emulsion of the fat is more or less broken up by freezing. It is also known that the casein is affected in some way, presumably being changed to a more permanent compound. Nothing accurate, however, is known as to the chemical changes which take place. The action of bacteria after the milk is thawed is somewhat different than in milk which has not been frozen. Clinically, this difference is probably of no importance.

Goat's Milk.—Goat's milk is pure white in color. It has the taste and smell of the goat. It is probable, however, that it would not have this taste and smell, if it was taken as carefully and under the same conditions as cow's milk. The composition of goat's milk is very similar to that of cow's milk. Bosworth and Van Slyke give it as fat 3.80%, milk sugar 4.50%, and protein 3.10%. The fat globules are smaller in goat's milk than in cow's milk. Consequently, the cream rises slowly. It is difficult to separate it thoroughly, even with a separator. The casein coagulates more quickly with rennin than does that of cow's milk. It forms compact masses. It is probably, therefore, harder to digest than the casein of cow's milk. The composition of the salts is somewhat different from that in cow's milk, but no more like that in human milk.

Goat's milk is no more suitable for the feeding of infants than cow's milk. It is necessary to modify both of them in some way to make them suitable for babies. It is said that goats are less often infected with tuberculosis than cows. This may be so, as goats are less often herded together in stables and exposed to infection. This fact is of no importance, however, as no baby should ever be given milk which is not known to come from non-tuberculous cows or which, if it is not, has not been pasteurized.

PRINCIPLES OF ARTIFICIAL FEEDING

There are certain fundamental principles as to nutrition and feeding, which have already been mentioned, which must be kept constantly in mind. In the first place, there are certain food elements—fats, carbohydrates, proteins and salts. A food must contain one or more of these elements. If it does not, it is not a food. Nothing else can take the place of these elements. In order to thrive and gain a baby must get a sufficient amount of food, not in ounces or quarts, but in calories. The number of calories which it needs corresponds to its weight and age. It must not only get the proper amount of calories, but it must get a sufficient amount of protein to cover its nitrogenous needs. Furthermore, it is not sufficient for it to get the required amount of calories and proteins, but they must be in such a form that they can be digested and utilized. The salts must also not only be sufficient in amount, but in such a form that they can be made use of. Water is also necessary for the proper carrying on of the metabolic processes of the body. The food must also contain a sufficient amount of the vitamins. If it does not, the baby cannot thrive, no matter how satisfactory the food is in other ways. All of these fundamental principles must be kept in mind when feeding babies artificially. If a single one of them is forgotten, the result will be failure instead of success.

It would seem, offhand, as if an artificial food which contained the same food elements in the same relative proportions that they are in human milk would be a perfect food and answer as well as human milk.

Experience has shown, however, that, while some babies will thrive on a food of this composition, it is not suitable for all babies or for all ages. While babies thrive throughout the nursing period on human milk of uniform strength, they cannot take a food as strong as this in the early weeks and months and need a stronger food in the later months. Furthermore, no artificial food, although it may contain the same proportions and amounts of the different food elements, is the same as human milk. It is impossible to make an artificial food which is identical with human milk.

Both human milk and cow's milk contain 4% of fat. Human milk contains 7% of lactose and cow's milk only 4.75%, while cow's milk contains more than twice as much protein as human milk, 3.50% against 1.50%. Cow's milk, moreover, contains 0.70% of salts in comparison with 0.20% in human milk. Both are amphoteric in reaction when they leave the breasts. Cow's milk, however, is usually acid when it reaches the baby. Human milk is practically sterile as the baby takes it. Cow's milk, even under the best conditions, is far from sterile when the baby gets it. The emulsion of the fat is much finer in human milk than in cow's milk. The proportion of fatty acids is much higher in cow's milk than in human milk. A large proportion of the protein in human milk is in the form of whey protein, while a large proportion of the protein in cow's milk is in the form of casein. The buffer value of cow's milk is, therefore, much greater than that of human milk. The curd formed in the two milks by rennin is different. The enzymes of the two milks are different and each milk has a specific serum reaction. It is evident, therefore, that no matter how cow's milk is modified, it will still be different from human milk. It is true that the percentages of the different food elements can be made the same. It is also true that the size of the fat corpuscles can be made the same by homogenization. The composition of the fat can also be changed to resemble that in human milk. The differences in the proteins can be corrected by the use of whey. The composition of the salts can also be changed. Not all of these things can be done at the same time, however. The ferments can never be made the same and the specific serum reaction cannot be changed. If cow's milk is corrected in one way, it cannot be in another. If all these corrections and modifications could be made at once, the cow would certainly not recognize it as her own product and the baby would never mistake it for human milk.

In spite of the fact that it is not possible to make human milk from cow's milk, physicians are constantly attempting to do it. Those who criticise most harshly the calculation of the proportions of the different food elements in percentages are the worst offenders. All of them attempt to imitate human milk in one way or another, although they disclaim it. Certain of them devote all their attention to changing the character and the composition of the fat. Others are satisfied that they have made human milk when they imitate the relations of the salts. Still others think that they have accomplished their object when they have made the hydrogen-ion content of the two milks the same. They all apparently forget that in changing one part of the milk they have not changed the others and that they still have cow's milk instead of human milk as their product.

The composition of human milk does, however, teach us certain things as to the digestive capacity of infants and as to the general principles to be followed in the preparation of a food to meet this digestive capacity. Nature provides a dilute food, rich in fat and carbohydrates and rela-

tively low in protein, that is, approximately 50% of the caloric value of human milk is furnished by the fat, 40% by the sugar and 10% by the proteins. It seems reasonable to suppose that this type of food is the one best suited for the infant's digestive power and metabolic processes. If so, it is evident, therefore, that well babies should be given artificial foods, which are dilute and which contain relatively large amounts of fat and carbohydrates and relatively small amounts of protein. The object aimed at in giving babies such foods is not, however, to imitate the composition of human milk, but to follow the indications furnished by human milk as to their digestive capacity and metabolic processes. In my experience, well babies, on the whole, thrive better on foods of this character than on any others. The same principles cannot be applied, however, to the feeding of sick babies or to that of a certain number of well babies. When a baby does not thrive on foods of this type, they must be discarded at once and the composition of the food regulated to fit the digestive capacity of the individual baby. The digestive capacity must be determined by a careful study of the symptoms and stools in the individual instance. The composition of the food must then be changed to suit the individual baby. Success in the artificial feeding of infants can never be attained by following any hard and fast rules. Every baby is a problem by itself. The baby, not rules, must be followed in solving this problem.

It has become the fashion recently for many men, in discussing infant feeding, to state, or at least to imply, that it is a very simple matter and that all babies can be fed in the same way. Furthermore, they openly or covertly ridicule those who believe that it is not as simple as they think. I do not hesitate to admit that I consider the artificial feeding of babies a difficult problem. The average normal baby does well, of course, on any reasonable food. The reason that it does is not because the food is the best one for it, but because of its wide limits of tolerance. That is why so many physicians think that artificial feeding is so simple. The strong babies are able to get on in spite of the unsuitability of the food. The feeble, however, do not. It is safe to say that the physician who thinks that he is able to feed all babies in the same way and who has never had any trouble in feeding a baby on an artificial food either has been remarkably fortunate in the class of babies that have come into his hands or has had very few babies to feed. Any one who believes that he can successfully feed all babies on a single food or by the same method is certain, if he has many babies to feed, to find that he is mistaken.

The artificial food for a baby is best prepared from the milk of some animal, because the milk of animals contains the same food elements as human milk, does not contain any other elements and is intended for the growth and development of a young animal. It is not intended, however, for the growth and development of an infant, but for that of the young animal of that species. It is fitted, therefore, to the digestive capacity of that young animal and for its peculiar needs for growth and development, rather than to the digestive capacity and for the growth and needs of an infant. It is not entirely suitable, therefore, as a food for infants. Some babies can thrive on the undiluted or unmodified milk of an animal, but most babies do better when it is diluted or modified in some way to make it more suitable for their digestive capacity and needs.

The milk of the cow is the most suitable for the preparation of a baby's food. The composition of mare's milk is more like that of human milk than is that of cow's milk. It is, however, impossible to obtain,

at any rate in this country, a sufficient amount of mare's milk for infant feeding. Goat's milk is no more like human milk than is cow's milk. Its supposed advantages are discussed elsewhere. It has to be modified to fit the baby's needs in the same way as cow's milk. Furthermore, cow's milk can be obtained anywhere in sufficient quantities, while it is difficult to get large amounts of goat's milk.

It is not sufficient, however, simply to get cow's milk for a baby. To be satisfactory, it must be pure and clean. It is now possible to get certified milk in all the large cities. This is as pure and clean as it is possible for milk to be. In my opinion, it is safe to give certified milk raw. Furthermore, although I realize that it is impossible to bring forward any arguments to show that raw milk is preferable to pasteurized milk as a food for infants, except that the antiscorbutic vitamin is destroyed, I cannot help feeling that babies do somewhat better on raw than on pasteurized milk. I also realize that the destruction of the antiscorbutic vitamin is of no importance, because it is easy to make up for its loss by the administration of orange juice or some other antiscorbutic. Certified milk can be rendered even safer, of course, by pasteurization, if there is fear of bacterial infection. "Grade A" milk, which is also obtainable in all the large cities and is pasteurized, is a thoroughly satisfactory milk for babies. In the cities it is safer to pasteurize all milk, unless it is certified. It is often more difficult to get a suitable milk for babies in the country than in the cities. In the country, unless the cows are known to be free from tuberculosis, all milk, no matter how fresh it can be given to the baby, should be pasteurized or boiled. If the cows are free from tuberculosis, the milk taken under reasonably sanitary conditions and but little transportation required, it may be given raw. Otherwise, it should be pasteurized or boiled. It goes without saying that, no matter how pure or how fresh the milk or whether it is raw or pasteurized, it must be kept cool and guarded against bacterial infection after it reaches the consumer to make it a safe food for a baby. Bacterial infection of the milk is certain to occur, if the container is open or if it is carelessly handled, and bacteria are certain to multiply in it very rapidly, if it is not kept cold.

The milk of Ayrshires and Holsteins is more suitable for babies than that of Jerseys and Guernseys, because of its lesser fat content, the finer division of the fat and the lower proportion of volatile fatty acids. Some babies can take Jersey and Guernsey milk without being disturbed, others cannot. Not infrequently, babies that are upset by a given percentage of fat in the food, when it is prepared from Jersey or Guernsey milk, can take the same amount of fat without difficulty, when the food is prepared from Ayrshire or Holstein milk.

Other things being equal, it is better to use the mixed milk of a herd than the milk of one cow, because, if the milk of one cow only is used, and that cow becomes sick, the baby is almost certain to be made ill, while, if one or two cows in a herd are sick, their milk is so diluted by that of the others that the baby is usually not affected. On the other hand, it is evident that a baby is better off on the milk of one cow that is healthy and well taken care of and whose milk is properly looked after than on the mixed milk of a herd that is not properly fed and whose milk is not carefully taken or looked after.

Idiosyncrasy to Cow's Milk.—It is often said that certain babies cannot take cow's milk in any form without being made ill. In most instances the trouble is not with the cow's milk, but with the way in

which it is given. Almost all of these babies can take cow's milk without difficulty, if it is properly modified to fit their individual digestive capacities. In rare instances, however, there is a real anaphylaxis to the protein of cow's milk. If this is the case, even the smallest amount of milk causes symptoms and a positive reaction is almost invariably obtained when a skin test with cow casein is done. The usual cause of the sensitization is the giving of cow's milk during the first few days of life, when the intestines were permeable to the foreign protein. The sensitization apparently occasionally develops later when cow's milk is given at intervals of ten days or more instead of continuously, at a time when the intestines are abnormal and absorption is possible. Sensitization through breast milk is theoretically possible, but probably seldom happens. When a baby is sensitized to cow's milk, if it cannot get breast milk or is old enough to be weaned, goat's milk, either fresh or dried, may be used in place of cow's milk. It can be desensitized by giving very small amounts of milk and increasing the amount until immunity is obtained. This procedure is usually unnecessary, however, because the sensitiveness usually disappears during childhood.

Modified Milk.—If it is true, as I believe it is, that it is advisable to follow the indications apparently shown by nature as to the most suitable relations between the different food elements for the digestive capacity and metabolic processes of the normal infant, it is evident from the comparison of human and cow's milk that the relations between these elements in cow's milk must be changed in some way to correspond to those in human milk.

TABLE XI

	HUMAN MILK, PER CENT	COW'S MILK, PER CENT
Fat	4.00	4.00
Sugar	7.00	4.75
Protein	1.50	3.50
Salts	0.20	0.70

It is evident that, while the percentage of fat is the same in the two milks, the percentage of sugar is higher and that of the protein lower in human than in cow's milk. It is necessary, therefore, in order to have the general relations of the fat, sugar and protein to each other in foods prepared from cow's milk correspond to those in human milk and, therefore, to meet the indications given by nature as to the combination of the food elements most suitable for the average, well baby, to modify these relations in some way. Simple dilution of cow's milk does not change the relation of the food elements to each other in any way. Consequently, simple dilutions of whole milk do not provide a suitable food for the average well baby.

It is possible to prepare foods for babies from cow's milk, in which the relations between the different food elements are similar to those in human milk, by taking advantage of the fact that cream contains a relatively large amount of fat in comparison to the amount of sugar and protein, while skimmed milk contains a relatively small amount of fat and a relatively large amount of sugar and protein. When milk stands, the fat rises to the top, while the sugar and protein remain approximately evenly divided throughout the mixture. This is equally true, when milk is separated by machinery.

Cream is, technically, any milk which contains more than 4% of fat. The greater the percentage of fat, the less the percentage of sugar and

protein. For example: 10% cream, that is, cream containing 19% of fat, contains 4.45% of sugar and 3.27% of protein. 16% cream contains 4.20% of sugar and 3.05% of protein and 32% cream contains 3.40% of sugar and 2.50% of portein. It is evident that when cream is diluted, the relation between the fat and the protein will be similar to that in human milk. For example: a mixture of one part of 16% cream with three parts of water contains 4% of fat and about 0.75% of protein. It is also evident that when 16% cream is diluted in this way, the mixture contains only a little more than 1.00% of sugar, that is, a proportion of sugar much lower than that in human milk. It is very easy, however, to raise the percentage of milk sugar to any desired point by the addition of dry sugar. The method of modifying cow's milk in order to get the general relations between the different food elements present in human milk consists roughly, therefore, in diluting cream with water and adding dry sugar.

If the mixtures prepared on this general plan do not fit the individual baby, it is very easy to vary the relations and the amounts of the food elements. The amount of fat can be increased or diminished by varying the strength or the amount of the cream used. The amount of protein can be increased without changing that of the fat by the addition of skimmed or fat free milk, which contains a considerable amount of protein and very little fat. The amount of sugar can be varied by varying the amounts of sugar added. Other sugars may be used in place of milk sugar, if desired. Starch may be added to the food, if it seems advisable. If there is trouble with the digestion of casein, the amount of casein can be diminished by using a cream containing a high percentage of fat and the additional protein desired added in the form of whey.

The Use of Percentages.—I have found that the most satisfactory way of determining the composition of an infant's food is to think and calculate in percentages of the different food elements. This does not mean that I believe in any special combination of the food elements. It is merely a convenient method of calculation and a means of attaining relative accuracy in the preparation of the food. It neither presupposes nor implies anything as to what should be in the food or why it should be there. These points must be determined in other ways. It is evident, of course, that the mixtures, when prepared, do not contain exactly the percentages of the food elements which they are calculated to contain. It would be impossible for them to do so, because the cream and milk of which they are made are not constant in their composition. This is especially true when the mixtures are prepared at home. The percentages are, however, approximately correct and nearly enough so for practical purposes. As a matter of fact, most babies do not notice slight variations in the composition of the food. The variations in the composition of a mixture from day to day are, moreover, probably less than the daily variations in the composition of breast milk. In any event, the relative proportions of the various food elements are correct, even if the exact percentages are not. If the food does not agree, changes in the percentages of the different elements to meet the indications furnished by the symptoms will be accurate relatively to the original percentages, which is all that is necessary.

It is said that, while the statement of the percentages of the various food elements in a mixture shows the relation of these food elements to each other, it shows nothing as to the amounts of each element which the baby gets, which is the important thing, and that it is, therefore, more scientific to calculate the amounts of each element which the baby gets

than their relations to each other. Both of these statements are true. Nevertheless, clinically, when a baby is given enough to satisfy it of a mixture, the percentage composition of which is known, it will be found that the amount of the various food elements is sufficient. It is very easy, of course, to determine the number of grams of each element in the food by multiplying the amount of food in cubic centimeters by the percentage of this element in the mixture. For example: if a baby is taking a quart, 960 c.cm., of a mixture containing 3.00% of fat, it gets 28.8 grams of fat, which furnishes 268 calories. Incidentally, if it is desired to calculate the composition of a baby's food by giving it so many grams of each element per unit of weight daily, which seems to me unnecessarily complicated, it is useful to know that the average breast-fed infant taking a milk containing 4% of fat gets from five to six grams of fat per kilogram of body weight daily, and one taking a milk containing 3% of fat, a little more than four grams per kilogram daily. The breast-fed baby gets about ten grams of sugar per kilogram of body weight daily.

The Use of Calories.—The calorie referred to in infant feeding is the large calorie, that is, the amount of heat necessary to raise one kilogram of water 1° C. The caloric needs of babies have already been referred to. Briefly, they are, in the newborn, sixty-two calories per kilo or twenty-eight calories per pound; at three weeks, one hundred calories per kilo or forty-five calories per pound; at six weeks, one hundred and twenty calories per kilo or fifty-five calories per pound; at one year, one hundred calories per kilo or forty-five per pound; and, during the second year, ninety calories per kilo or forty calories per pound. Most young babies will hold their weight on from seventy to seventy-five calories per kilogram. Occasionally, a baby will gain regularly on this amount. Other babies, however, need as much as one hundred and forty calories per kilogram in order to gain. Babies that have been underfed or are convalescing from some illness need more calories than normal babies. Fat babies need fewer calories than the average baby, because they have less active tissue in proportion to their weight. On the other hand, a thin baby needs more calories than the average. Extremely emaciated babies often need as much as one hundred and sixty calories per kilogram. Active babies need more calories than quiet babies. That is, there is no hard and fast rule as to how many calories a given baby must have in order to thrive and gain.

While it is true that a baby must have a sufficient number of calories in order to thrive and gain, it seems to me irrational to base any scheme of feeding simply on the caloric needs and to be satisfied, if the food contains the required number of calories. To me it does not seem enough to say that a baby of a given weight must be given so many ounces of milk a day to cover its caloric needs, as if that was all that was necessary and it made no difference in what form the source of these calories was present in the food. Providing a sufficient number of calories in the food meets only one of the requirements. The food which provides these calories must be one which the individual infant can digest and utilize. It must never be forgotten that, no matter how high the caloric value of a food may be, it will be of no use to the baby, if it cannot digest it and utilize it. Crackers and cheese both have a high caloric value, but they are not suitable articles of diet for a three month's old baby. Sawdust has a high caloric value, but not even a calf can be fed satisfactorily on sawdust. A food may have a high caloric value and be suitable for the average baby and yet be of but little value to an individual baby. If, for example, a baby that has an

intolerance for fat is given a food whose caloric value is correct, but which is due largely to its fat content, the baby will not gain on it, but will be made sick. On the other hand, if the caloric value of the food is due to sugar and protein, it will be able to utilize them and gain. Chapin has also shown that, on account of the different powers of fat and carbohydrates as producers of metabolic water, they cannot be considered as interchangeable as regards favoring the growth of the organism. He has also called attention to the fact that the net caloric value of a food depends on the amount of energy required for its digestion and assimilation. Foods having the same gross caloric value may differ very materially, therefore, in their net caloric value. For example, the net caloric value of carbohydrates is much higher than that of proteins. It seems to me, therefore, that, as already stated, it is irrational to base any scheme or system of feeding on the caloric needs of babies or on the caloric values of the food. The fitting of the food to the digestive capacity of the individual infant is the most important thing. When the food agrees with the baby, it is not difficult to give enough of it to cover the caloric needs. The calculation of the caloric value of the food should, therefore, serve merely as a check. If, when a baby is not gaining, it is found that the caloric value of the food is insufficient, it is evident that this is the cause. If the caloric value is amply sufficient, it is evident that there is some other cause. Used in this way, as a check, the calculation of the caloric value of foods is often of great assistance.

Intervals in Artificial Feeding.—It is impossible to lay down any arbitrary rules as to the intervals between feedings and the number of feedings at different ages. Some babies do well at all sorts of intervals, just as they do on the breast. If the intervals between feedings were as important as some think them to be, there would be more difference between the babies who were fed in the past at two hour intervals in Boston and those who are fed at four hour intervals in Chicago. I doubt very much, if a baby from one of these cities was seen in San Francisco, if any one there could tell whether it came from Boston or Chicago. It is more important, however, to feed a baby that is on an artificial food regularly than one that is on the breast. The breast-fed baby will often thrive well, if fed at any and all times. The artificially-fed baby, however, is likely to be upset, unless it is fed regularly and offered the same amount at each feeding. The intervals advisable are, in general, the same in the artificially-fed as in the breast-fed baby. I am in the habit of feeding at two and one half hour intervals during the first month, although sometimes I begin with three hour intervals. From that time on I use the three hour interval. I do not approve of the four hour interval, at any rate, before babies are nine months old, but am perfectly willing to admit that many babies thrive on three hour intervals from the first and on four hour intervals after they are a few weeks old. I also can testify that, in past years, many babies thrived on two hour intervals during the first weeks or months and on two and one half hour intervals later. It is advisable to drop the night feeding, when the baby is one or two months old, and the evening feeding, when it is four or, at the latest, six months old. If a baby is on four hour intervals, it is necessary to keep up the evening feeding longer, in order to give the baby a sufficient amount of food without giving it too large a feeding at one time.

It is evident that the intervals between feedings must vary with the amount of food given at a feeding, the strength and composition of the food, and the digestive capacity and gastric motility of the individual

infant. If a large amount is given at a feeding, the intervals between feedings must be longer than when a smaller amount is given, as the time required for the stomach to empty itself will be longer. It takes longer for a food rich in fat to leave the stomach than for one poor in fat, because fat delays the emptying of the stomach. A mixture in which the protein is largely in the form of whey protein will leave the stomach more quickly than one in which the protein is almost entirely casein. A food which contains a large amount of casein leaves the stomach more slowly than one which contains a smaller amount of casein. Foods rich in carbohydrates and low in fat and protein leave the stomach quickly. Furthermore, there is no doubt that the digestive capacity and gastric motility vary in different infants. The intervals should, therefore, be determined in each case according to the conditions actually present in that case, not by any set rules.

Amount of Food at Single Feeding, Number of Feedings and Daily Amount of Food.—It is impossible to consider the amount of food taken at a single feeding and the number of feedings separately, as it is evident that they must vary together. Both being, moreover, dependent on the daily amount of food, the first thing to determine, therefore, is the amount of food to be given in twenty-four hours. It is evident that this amount of food must contain the required number of calories and that a greater quantity of a dilute food must be given than of a strong food. Experience has shown that the amount of food taken by well babies in twenty-four hours increases rapidly during the first few months and less rapidly during the remainder of the first year. Experience has also shown that the average well baby takes of a food suitable for its age, about ten or twelve ounces at the end of the first week, twenty ounces when it is a month old and thirty-two ounces when it is four months old. It takes from thirty-six to forty ounces at six months and forty-eight ounces at nine months. In a general way, the average baby takes about one half ounce at a feeding in the first few days and from an ounce to one and one half ounces when it is a week or ten days old. It takes about two and one half ounces when it is a month old, four ounces at three months, six ounces at six months and eight ounces at nine months. It is evident, however, that these figures are only averages and that they are also only relative. If a baby is fed at long intervals, it will naturally take more at a feeding than when it is fed at short intervals. It can take amounts considerably above its anatomic gastric capacity, because the food begins to leave the stomach at once. It is also evident that, if the food is strong, the baby will not need to take as much of it at a feeding as when it is more dilute. That is to say, it is impossible to lay down any hard and fast rules as to the intervals between feedings and the amount to be given at a single feeding at given ages. The intervals and the amount at each feeding must vary with the circumstances in the individual case. It is impossible, therefore, to construct a table as to how much and how often a baby of a given age should be fed. The best that can be done is to make one which shows what the average baby usually does and which can serve as a guide in beginning to feed a normal baby.

Eight feedings at two and one half hour intervals means every two and one half hours from 6 A.M. to 9 P.M. or 10 P.M., and once between the evening feeding and morning.

Seven feedings at three hour intervals means every three hours from 6 A.M. to 9 P.M. or 10 P.M., and once between the evening feeding and morning.

TABLE XII

Age	24° amount, ounces	Number of feedings	Amount at a feeding, ounces	Intervals, hours
1 week	12	8	1½	2½
4 weeks	20	8	2½	2½
		7	3	3
4 months	32	6	5–5½	3
6 months	36–40	5	7–8	3
9 months	48	5	9–10	4

Six feedings at three hour intervals means every three hours from 6 A.M. to 9 P.M. or 10 P.M.

Five feedings at three hour intervals means every three hours from 6 A.M. to 6 P.M.

Five feedings at four hour intervals means every four hours from 6 A.M. to 10 P.M.

Composition of Food.—Nature provides a food of essentially the same strength throughout the whole period of lactation. It is somewhat weaker toward the end of the period. In spite of these facts, experience has shown that young babies cannot take and thrive on as strong an artificial food as older babies and that it is advisable to start with a weak food and gradually strengthen it as a baby grows older. Experience has also shown that, on the average, babies of certain ages digest and thrive best on certain strengths of food. My own belief is, as already stated, that the average well baby thrives best on artificial foods in which the relations of the fat, sugar and protein in the mixture are similar to those in human milk. On the basis of my experience and belief, I have prepared the following table, as a guide to those who have had less experience, as to how to begin to feed a baby. It must be understood, however, that it is intended only as a guide to the beginner in the choice of the first mixture to be given to a baby. After that, the composition of the food must be changed to meet the indications furnished by the symptoms, the stools, the appearance and weight of the individual baby at the given time. One who is accustomed to the feeding of babies needs no table to guide him. He is able to judge very closely from the history, appearance and weight of the baby what food is likely to agree with it. It must never be forgotten, moreover, that babies cannot be fed by rule and that the food must be changed until it meets the needs of the individual infant.

TABLE XIII

* A GUIDE TO THE COMPOSITION OF THE FOOD FOR AVERAGE WELL BABIES

Age	Fat	Sugar	Protein
First food	1.00	5.00	0.50
First week	2.00	6.00	0.75
1 month	3.00	7.00	1.00
2 months	3.50	7.00	1.50
4 months	4.00	7.00	1.75
6 months	4.00	7.00	2.25
8 months	4.00	7.00	2.50

* Taken from Morse & Talbot's "Diseases of Nutrition and Infant Feeding." The Macmillan Company.

In spite of what has been said above, I am sure that certain physicians will continue to say that I believe that all babies of a given age should be given the same food and that I believe in a routine method of feeding. I do not. I believe that there are certain fundamental principles which should be followed in the feeding of normal infants, but cannot emphasize too strongly the facts that the food must always be fitted to the needs and digestive capacity of the individual infant and that ill babies cannot be fed in the same way as well babies. Objections are certain to be made to this general plan of feeding on the grounds that the mixtures advocated are dilute. It seems to me that this is an advantage rather than a disadvantage. In addition to the fact that Nature provides a dilute food for the infant, it is well known that water leaves the stomach very quickly and that, being quickly absorbed, it does not interfere with intestinal digestion. Moreover, dilute foods leave the stomach more quickly than concentrated. Furthermore, an abundance of water favors the normal carrying on of the metabolic processes of the body and elimination.

It is said, in criticism, that this general plan amounts simply to varying the dilution of the food elements and that the relations of the elements to each other remain the same, as if every one who used it did not know it. It certainly is so and is intended to be so. The general relations of the food elements are those indicated by the composition of human milk, while the food is more dilute in the beginning, because experience has shown that young babies cannot digest as strong mixtures as they can later. The strength is gradually increased to correspond to the increase in the baby's digestive powers, and the proportion of protein is gradually increased for the same reason. The object is to get the baby on to whole cow's milk as soon as it is able to digest it, then on to other foods, and finally, when it is old enough, on to a general diet, but to do this without making it sick at any time from overfeeding.

The Various Food Elements.—It is possible, not only to vary the amounts and the relations of the different food elements to each other, but to use these elements in different forms. Various forms of fat and sugar may be used or some kind of starch substituted for part of the sugar. A considerable proportion of the protein may be given in the form of the whey proteins instead of as casein, and the casein can be modified in various ways. The salts may also be changed to a certain extent.

Fat.—It is a simple matter to vary the amount of fat in the food, but, if cow's milk is used, the character of the fat can only be changed in so far as it differs in the milk of different breeds of cows. The emulsion of the fat of cow's milk may be made finer, however, by homogenization. In this way the fat droplets are made much smaller. Olive oil may be used in the place of butter fat and homogenized in the same way. I cannot say that I have ever seen any advantage from its use. The composition of the fat of human milk can be very closely imitated in all respects by the use of a combination of different sorts of fat, such as tallow oil, cocoanut oil and tallow, as has been done by Gerstenberger and Ruh in their "Synthetic Milk Adapted." The fat is also homogenized. Not even a baby, however, would mistake the taste of this preparation for that of human milk. While the composition of the fat in this food is like that in human milk, it is not like human milk in other ways and is not susceptible of modification, except by dilution, to meet the needs of the individual baby.

Very few babies are able to take more than 4% of fat continuously in their food without being disturbed. It is, therefore, inadvisible,

except in occasional cases, to give more than 4% of fat. Most babies, more than a few months old, can take this amount of fat, however, without being disturbed and, it seems to me, do better on it than they do on smaller amounts. When milk mixtures are prepared in the home, however, it is advisable not to give more than 3 or 3 ½% of fat in a mixture, because, owing to errors in the method of calculation, the mixtures always contain rather more fat than they are supposed to.

In the past, more often than at present, babies were not infrequently given too large amounts of fat. There are several reasons for this mistake. One of them is that mothers, nurses and physicians are all anxious to have babies gain rapidly in weight and believe that the best way to make them gain is to give them fat, forgetting that, although fat has a high caloric value, it is very likely to disturb the digestion, if given in too large amounts, and that the carbohydrates are as useful as fat for this purpose. Another reason is that everyone knows that fat is a laxative. They forget that an excessive amount of fat is one of the most common causes of constipation in infancy, the fat combining with calcium and magnesium to form soap stools. Increasing the fat with the intention of relaxing the bowels, naturally simply increases the constipation. Other reasons why babies get too much fat in their food are that physicians are not careful enough in their directions as to the preparation of modified milk and that physicians, nurses and mothers forget that some milks are richer in fat than others and that creams are of different strengths. All table cream, for example, contains at least twice as much fat as gravity cream, while the percentage of fat in the top eight ounces of a quart of milk is twice as great as that in the top sixteen ounces. Unless proper care is used both by the physician in the calculation and by the mother or nurse in the preparation of the formula, it is certain to contain either more or less, usually more, fat than is intended.

The caloric value of fat compared with that of carbohydrates and proteins is as 9.3 is to 4.1. It is evident, therefore, that unless a reasonable amount of fat is used in the food, it is difficult to meet the caloric needs of an infant without using an excessive amount of carbohydrates or proteins or an excessive quantity of food.

Sugars.—Milk sugar being the only form of sugar present in human milk and in the milk of animals, it seems reasonable to suppose that it is the sugar most suitable for the growth of the young organism, whether human or animal. It is hardly necessary, however, to make use of this argument in favor of milk sugar, because there are several reasons which show that it is the most suitable form of sugar for well infants. It is more slowly and more completely absorbed than the other disaccharides. Being more slowly absorbed, it is present for a longer time and at lower levels of the intestines than the others and is thus more conducive to the development or persistance of the normal, fermentative flora throughout the intestinal tract. Few organisms, moreover, other than those normal to the intestinal tract of infants, utilize lactose before it is broken down, while many can utilize the other double sugars. It furnishes, therefore, a more efficient protection against abnormal bacterial processes in the intestines. It is probably true that the net energy value of milk sugar is less than that of malt sugar or of preparations which contain a certain proportion of dextrose, such as corn syrup. It is not probable, however, that in well babies this difference is of any practical importance. It is safe to say that the lactose of cow's milk is the same as that of human milk.

It is inadvisable to give more than 7% of milk sugar continuously in an infant's food. Larger amounts may, however, be given safely for considerable periods when, for any reason, it is necessary to keep one or more of the other food elements low. Eventually, however, fermentation, with diarrhea, is almost certain to develop. If larger amounts of sugar than 7% are desirable for any reason, it is usually better to give part of the sugar in the form of one of the combinations of maltose and the dextrins, which are more quickly absorbed and consequently less likely to cause fermentation. Milk sugar is never found in the urine or feces under normal conditions, unless more than 7% of sugar is given. It is true that the assimilation limit of milk sugar is lower than that of the other disaccharides, but this limit is far above the amount which is present in any reasonable mixture. It was said that lactose caused fever more readily than the other sugars. It having been proved, however, that "sugar fever" is not caused by sugar, it is evident that this statement is of no importance. Gain in weight is for a time more rapid with maltose and saccharose than with lactose, because of the easier assimilation and more rapid absorption of these sugars, which influences the quantity of water eliminated by the kidneys. Lactose, in reasonable amounts, under normal conditions, has a slight laxative effect, as does maltose, while saccharose is slightly constipating. When given in excess, lactose is more likely than the other disaccharides to cause diarrhea, probably because of its relatively slow absorbability. The order is lactose, saccharose, maltose and the dextrin-maltose mixtures. The larger the proportion of maltose in these dextrin-maltose mixtures, the more likely are they to be laxative in action.

It is evident, therefore, that, under normal conditions, lactose is the preferable sugar for infant feeding. This is frequently not the case, however, when there are disturbances of digestion. In such instances, if they are fermentative in character, the amount of milk sugar should be diminished or one of the other sugars substituted for it. While it is true that under normal conditions the preferable sugar for the well infant is milk sugar, it is equally true that the tolerance of the normal infant for sugar is so great that it will usually thrive on any form of sugar, provided the amount is not too large.

Maltose.—Pure maltose is never employed in the feeding of infants, because it is altogether too expensive to be used in this way. The various preparations to which this term is erroneously applied are mixtures of the various dextrins and maltose. The proportions of the dextrins and maltose in these mixtures vary, as do also the proportions of the various forms of dextrins. The composition of the preparations of this type which are most commonly used in this country is as follows:

TABLE XIV

Food	Maltose per cent.	Dextrins per cent.
Loeflund's malt-soup extract	58.91	15.42
Neutral maltose (Maltzyme Co.)	63.00–66.00	8.00–9.00
Maltose (Walker-Gordon Laboratory Co.)	57.10	30.90
Mead's dextri-maltose, No. 1	51.00	42.00
Mead's dextri-maltose, No. 2	52.00	43.00
Mead's dextri-maltose, No. 3	51.00	41.00
Mellin's food	58.88	20.69
Malted milk	49.15*	18.80

*About 10% is milk sugar.

The properties of maltose and the dextrins are not the same. Maltose is a disaccharide, while the dextrins are polysaccharides. Maltose is a crystalloid, fermentable and dialyzable; the dextrins are reversible, protective colloids, non-fermentable and non-dialyzable. It is evidently, therefore, of considerable importance which of these preparations is used. The dextrins, being protective colloids, probably have much the same effect in favoring the digestion of casein that starch and lactalbumin have. Maltose has no such action. The dextrins have to be changed to maltose and then to dextrose before they are absorbed. It is evident, therefore, that the larger the proportion of dextrins in the maltose-dextrins mixtures, the slower is the absorption of sugar and vice versa. The combinations which contain relatively large amounts of maltose are more laxative than those containing relatively large amounts of the dextrins, because there is a larger amount of sugar present in the intestines at one time. On the other hand, there is less danger of overtaxing the absorptive powers of the intestines and flooding the system with sugar when there is a relatively large amount of the dextrins. If it is desired to give sugar in the form in which it can be readily and rapidly absorbed, the proportion of maltose should be large. It is possible to give sugar in a still more easily available form by using corn syrup, in which there is about 33% of dextrins, 21% of maltose and 15% of dextrose.

Maltose is split into dextrose and dextrose and can be immediately utilized; lactose is split into dextrose and galactose; and saccharose into dextrose and levulose. Only the dextrose half of these sugars is, therefore, immediately available without further change. The net energy value of malt sugar is probably somewhat greater than that of lactose and saccharose, because, being converted at once into dextrose, no further energy is required, as there is to convert galactose and levulose. The immediate utilizability of malt sugar, and to a greater extent, of corn syrup is probably of some advantage in feeding feeble, emaciated babies, who have no store of glycogen in the liver, because the energy derived from the sugar is immediately available and can be used in the digestion of the rest of the food. This is of no advantage, however, in the feeding of normal babies. Maltose being more quickly absorbed, is less favorable to the maintenance of the normal intestinal flora than lactose. It is said that maltose is especially conducive to the growth of the bacillus acidophilus, and that this organism, if present in large numbers, may cause irritation of the intestines and an intolerance for sugar. In view of the fact that the bacillus acidophilus is used in the treatment of intestinal disorders, it seems doubtful whether this criticism is warranted.

It is a matter of considerable importance in disturbance of the digestion what form of sugar is used. When there is indigestion as the result of the fermentation of milk sugar, the combinations of the dextrins and maltose can be used more quickly after the sugars have been temporarily cut down than can lactose. The preparations which contain relatively large amounts of dextrins are preferable, because they are broken down more slowly. When there is excessive fermentation from maltose, milk sugar can often be used to advantage in its place. All forms of sugar are contraindicated, when the fermentation in the intestines is caused by the gas bacillus and similar organisms. Maltose is more dangerous than lactose, because it undergoes butyric acid fermentation more easily. Lactose is preferable to maltose in the treatment of intestinal disturbance due to the abnormal decomposition of proteins, because it is more slowly broken down and absorbed and, consequently, exerts a more prolonged action.

Cane Sugar.—I can see no reason for using cane sugar in place of milk sugar in feeding normal infants, except that it costs less. It is true that many infants thrive on it. It is probable that they thrive, not because of it, but in spite of it, the average normal infant being able to digest and utilize any of the sugars, provided they are not given in excess. Cane sugar undergoes alcoholic fermentation instead of lactic acid fermentation, like milk sugar, and, in consequence, is less suitable for the development and maintenance of the normal intestinal flora. If, for any reason, there are objections to the use of milk sugar, the combinations of maltose and the dextrins meet the indications far better than does cane sugar and, therefore, should be used instead.

Starch.—There are no physiologic contraindications to the use of starch in the feeding of infants, even of the new-born, because the amylolytic ferments are present and active in both the saliva and the pancreatic secretions at birth. This fact does not prove, however, that infants ought to have starch in their food or even that it is advisable for them to have it. It merely shows that there is no physiologic reason why it should not be given to them, if for any reason it seems advisable. It also does not prove, or even suggest, that babies should be given foods made up largely or almost entirely of starch. Clinical experience shows that, in general, it is not advisable to give starch to babies before they are two months old and that it is inadvisable to give large amounts of starch to babies before they are ten months old. Clinical experience also shows, however, that many babies do better on foods which contain starch than on those which do not. When starch is used in the food of infants, it should be used with the same care as are the sugars and other food elements. The amount of starch added to the food should be carefully calculated. One of the chief advantages in using starch in an infant's food is that it acts as a protective colloid and in this way prevents the formation of large casein curds. Starch is usually added to foods in cereal waters and gruels. Their action is due to the soluble starch which they contain, not to salts or to cellulose in suspension. It has been found that percentages of starch higher than 0.75% have no more colloidal action than 0.75% and that percentages lower than this are not as efficient. When starch is added for its colloidal action, 0.75% is, therefore, the optimum amount. This amount of starch is practically never beyond the digestive capacity of any infant. It is evident that, when starch is used in this way, it adds very little to the caloric value of the food. The caloric value of starch is, for practical purposes, the same as that of sugar, the nutritive value which is lost because of the greater energy required in breaking it down being, for practical purposes, negligible. Starch is often useful, when there is an intolerance for sugar or when sugars cause excessive fermentation. The reason that babies can sometimes take starch when they cannot sugar is that starch, having a more complicated molecular structure, has to be broken down through the various dextrins to maltose and finally to dextrose before it is absorbed, and, being broken down slowly, the end product, dextrose, is absorbed almost immediately. In consequence, there is less fermentable material in the intestines than when the sugars are given and, therefore, less opportunity for fermentation to occur. If excessive amounts of starch are used, however, it may cause marked disturbances of digestion and nutrition. The fermentation of starch results in the formation of free fatty acids, which have a strong irritant action on the intestines. Their action is the same, whether they are derived from starch or fats. On the other hand,

an excess of starch not infrequently causes constipation. The stools in such instances are hard, dry and light brown, and resemble the soap stool, except in color.

Starch is most often used in infant feeding in the form of cereal waters or gruels. The nutritive value of these preparations depends on the amount of starch which they contain. The kind of starch from which they are made makes no difference in their strength. The cereal waters, as usually prepared, contain about 1.50%, and the gruels about 3%, of starch. The cereal waters contain about 0.20% of protein and from 0.01% to 0.05% of fat; the gruels about twice as much. It is evident that, as these preparations are used merely as diluents, the amount of food value which they add to the mixture is very slight. What is added is practically entirely from the starch, that from the protein and fat being negligible. It is true, however, that cereal diluents made from the whole grain contain somewhat more protein than those made from flours.

It makes relatively little difference from what flours the cereal diluents are prepared. It is commonly supposed that barley water is constipating and oatmeal water laxative. As a matter of fact, however, their action is not at all constant, apparently depending on the idiosyncrasy of the individual infant, barley being constipating in one instance and laxative in another. The same is true of oats. Wheat and rye starch are said, however, to be broken down less rapidly than barley and oat starch and, therefore, less likely to cause acidity and fermentation.

One of the chief objections to the use of foods made up largely of starch, which is usually combined with sugar, which does not apply when starch is used in the form of the cereal waters and gruels as a diluent, is that such foods are very poor in salts. The deficiency of salts causes a disturbance in the retention of salts and water, which results in impairment of the nutrition and diminution in the resistance to infection. Serious disturbances of nutrition from the excessive use of starchy foods, although apparently common abroad, are, however, fortunately relatively rare in this country.

Protein.—Protein, as already stated, is essential to life and the only form of food which can replace the nitrogenous waste of the body and from which new cells can be built up. It can also serve, however, as a source of energy, and life can be sustained for considerable periods of time on a purely protein diet. Such a diet is, however, a wasteful one, throws an excessive amount of work on the organs of digestion and metabolism and seriously overtaxes the organs of elimination. The protein need of the infant is, of course, relatively greater than that of the adult, because it not only has to replace old tissue, but to build up new tissues. The average protein need in infancy is probably somewhat below 1.50 grams of protein per kilo, or 0.7 grams per pound, of body weight. If 1.50 grams of protein per kilo of body weight are given, it is, therefore, probably sufficient, although certain babies may require at times as much as two grams per kilogram, or 0.9 grams per pound, of body weight. The most available and the most easily digestible form of protein for infants is the protein of milk. The protein of human milk is more digestible than that of cow's milk. A part of the protein may be given in the form of vegetable protein, but vegetable protein cannot permanently replace animal protein in an infant's food.

No one believes now, of course, that the protein of milk is the cause of all the disturbances of digestion in infants taking cow's milk, as was at one time supposed. On the other hand, it is probable that at present

the tendency is to attach too little importance to the protein of cow's milk as a cause of disturbance of digestion and metabolism. One explanation of this tendency is that the disturbances caused by the proteins, like those caused by the salts, are less definite and less easy to recognize than those caused by fats and carbohydrates. An excess of protein may, for example, cause fever and a condition of semistupor, and under certain conditions irritate the kidneys.

The relation of the casein to the whey proteins in human milk is approximately as one to two, while in cow's milk it is as three to one. It is possible that there may be chemical differences between the casein of cow's milk and that of human milk. If there are, they are probably of little practical importance as regards the digestibility of the two milks. The difficulty lies in the greater proportion of casein in cow's milk. The absolutely greater amount of casein in cow's milk not only favors the formation of large, tough, casein curds, but the smaller proportion of whey protein diminishes its colloidal action in the prevention of the coagulation of the casein. It is the formation of large curds which renders the casein of cow's milk so much more difficult of digestion by the infant than that of human milk. If the formation of large, casein curds in the stomach can be prevented, the casein of cow's milk is easily digestible.

The average normal infant can usually digest without difficulty the amount of casein present in the type of mixtures which contain the same relative proportions of food elements that are present in human milk. Many of them cannot, however, easily digest the amounts of casein which are present in whole milk or in the dilutions of whole milk or skimmed milk which are now so commonly used. The reason for the introduction of or for the emphasis laid on many of the procedures which are now being used in infant feeding is that the large amount of case in in the mixtures at present in fashion has made it necessary to do something to prevent the formation of large, casein curds and thus make it possible for babies to digest them.

Methods of Preventing the Formation of Casein Curds.—*Reduction of the Amount of Casein.*—The simplest way to prevent the formation of casein curds is to diminish the amount of casein in the food. If this is done, great care must be taken, however, not to reduce the casein so much that the minimum protein needs are not covered.

Whey Mixtures.—One of the best ways of giving the protein in an easily digestible form is the whey mixture. The whey protein is not coagulated by rennin and, therefore, cannot form curds. Moreover, by its colloidal action, it hinders the formation of large, casein curds. This method, which was much used in the past, has been almost forgotten recently as the result of the efforts to make infant feeding easy for the doctor without regard to the baby and of the neglect to make use of the facilities of the food laboratories. By using whey mixtures it is possible to keep up the amount of protein in the mixture and thus avoid protein starvation. Whey mixtures have a further advantage, when there is vomiting, in that they leave the stomach very quickly, the whey protein not being acted on by rennin. Whey mixtures are less valuable when they have to be prepared in the home instead of in a laboratory, because, when gravity cream is used, as it has to be in the home, the amount of cream which has to be used in order to give the required amount of fat in the mixture contains a considerable percentage of casein and, therefore, reduces the amount of protein which can be given in the whey. When high percentages of cream are used, as they can be in a milk laboratory, the casein can be made very low and the whey protein high.

Cereal Diluents.—Another way to interfere with the formation of large, casein curds is by the addition of starch in the form of the cereal diluents, the starch acting as a protective colloid. When starch is added for this purpose, the amount should be 0.75%, as this percentage has as much effect as higher percentages, while smaller percentages are less active.

Boiling.—One of the most effective, as well as one of the simplest, methods of preventing the formation of large, casein curds is boiling the food. When rennin is added to boiled milk *in vitro*, coagulation takes place more slowly than in raw milk and the curd which is formed is soft and fine, instead of hard and dense, as in raw milk. The separation of the curd and whey is also much less complete than in raw milk. The experiments of Brennemann and others show that the same differences in the coagulation of the casein of raw and boiled milk by rennin occur in the stomach as *in vitro*. The food must be boiled hard at least five minutes in a saucepan in order to prevent the formation of large curds. Simmering in a double boiler does not do it.

Alkalies.—The addition of an alkali to milk unquestionably hinders or prevents the formation of large, hard curds in the stomach. How it does this is not definitely known. There is no doubt, however, that the coagulation of the milk in the stomach by rennin is delayed by the addition of an alkali, because rennin does not act in an alkaline medium. How much it is delayed, depends on the amount of alkali added and on the acidity of the milk, which in clinical work is always an unknown quantity. The more acid the milk, the more alkali is required to neutralize it, and the less is left to neutralize the hydrochloric acid secreted by the stomach, and vice versa. During this period of delay some of the uncoagulated milk leaves the stomach. The amount of milk which passes into the duodenum naturally varies largely with the length of time before coagulation takes place. The reason that milk can leave the stomach before the reaction of the stomach contents is acid is that milk, like water, can leave the stomach independent of the pyloric reflex.

Whatever the action of the alkali may be, there is no doubt that it consists partly in neutralizing the acidity of the milk and partly in neutralizing the hydrochloric acid secreted by the stomach, thereby changing the combination of calcium salts with casein. It is evident that, as regards the neutralizing of the acidity of the milk, whatever alkali is used, the amount to be added should be determined by the amount of milk and cream in the food, which determines its acidity and which alone contains casein, not in relation to the total quantity of the mixture. It is impossible, of course, in everyday work to know how much alkali to add, because the acidity of the milk must be an unknown quantity. Experience has shown, however, that, when lime water is used as the alkali, from 25 to 50% of lime water must be added to average milk in order to produce any appreciable effect. One and one half grains of bicarbonate of soda, which is often used in place of lime water, has about the same effect as an ounce of lime water. Their action is, however, not quite the same. Lime water swells the mucoid protein of the milk, which probably has some effect on the precipitation of the casein, while the carbonic acid gas which is formed from bicarbonate of soda during digestion tends to make the curds more porous.

Citrate of Soda.—The action of citrate of soda is different from that of lime water and bicarbonate of soda. It is of considerable value in preventing the formation of large, tough, casein curds. As already explained,

the citrate of soda combines with the calcium caseinate of the milk to form sodium caseinate and calcium citrate. Rennin splits sodium caseinate into sodium paracaseinate, which is very soluble. There is, therefore, no precipitation or curdling. Two grains of citrate of soda should be added to the ounce of milk or cream in the mixture in order to get the maximum effect.

Pancreatization.—Another way of preventing the formation of large curds, which was formerly much used and, although almost forgotten at present, is still valuable, is the partial predigestion of the food. This is commonly known as peptonization, but is in reality pancreatization, the active ferment being the trypsin of the pancreas. The casein is wholly or in part so changed by this method that it cannot be acted upon by rennin. The formation of large curds in the stomach is thus either wholly or entirely prevented.

Dried Milk.—The casein in dried milk is separated into minute particles. When water is added to prepare it for use, the casein particles are held in suspension in a very finely divided form. It is claimed that these finely divided particles are swollen in the acid medium of the stomach and precipitated as separate particles instead of large curds. This sounds reasonable and is probably true, but I have not seen any chemical or experimental proof of these assertions. Casein in the form of a dry powder may also be added to milk mixtures in order to increase their protein content. The casein in them is in the same form as in dried milk and, therefore, for the same reason, is not coagulated into large curds by rennin.

Protein Milk.—Protein milk contains a relatively large proportion of casein. The formation of large casein curds is prevented in this food, because a large proportion of the casein in it has been already precipitated by rennin in the form of calcium paracasein. It cannot, therefore, be acted upon again by rennin in the stomach. The paracasein curds have been, moreover, very finely divided by being rubbed through sieves in the preparation of the food. The digestibility of the casein in the buttermilk is increased by the lactic acid present in the buttermilk.

Buttermilk and Acidified Milks.—In these preparations the formation of large, casein curds is not prevented in the same way as in the mixtures just detailed. By the formation of acid in buttermilk or by the addition of hydrochloric, lactic or other acids to the milk the hydrogen-ion content of the milk is so changed that its buffer value is diminished and the digestion of the casein made easier. If, however, these foods did not contain a large amount of casein, it would not be necessary to acidify them in order to make them more easily digestible.

Salts.—The various salts, with the exception of iron, are present in sufficient quantities and in proper proportions in human milk. In most modifications of cow's milk, especially when dilutions of whole milk are used, there is an excess of salts and the proportions and composition of the various salts are different from those in human milk. A normal infant can, as a rule, thrive in spite of this excess of salts and in spite of their, for the infant, abnormal relation to each other. It is probable, however, that a part, perhaps a large part, of the disturbances of digestion in infants fed on cow's milk mixtures are due to the excess and abnormal relations of the salts which they contain. It is not reasonable, however, to attribute all disturbances of digestion to them, as some pediatrists do. Our knowledge concerning the salts, the part which they play in normal digestion and metabolism and the symptoms of the dis-

turbances which they caues is so limited, that it is impossible to pay much attention to them in the regulation of the diet, either in health or disease.

The Relation of the Different Elements of the Food to Each Other.—It is wrong to consider the different elements of the food and the disturbances of digestion which they may cause as if they were separate entities and entirely independent of each other. It is undoubtedly true that, in a certain number of instances, disturbances of the digestion are caused by single elements of the food. In many other instances, however, in which the disturbance seems to be due to a single element, it is unquestionable that the real difficulty lies in the relation of the various elements to each other. Our knowledge of the connection between disturbances of the digestion and the various food elements is extremely rudimentary. It is very important, therefore, to be extremely careful not to accept each new item of information as the final solution of the problem.

THE PRESCRIBING OF MODIFIED MILK*

The first thing to be done in prescribing modified milk for an infant is to determine what percentages of fat, sugar, protein and starch are to be in the mixture. The next thing to decide is what form of sugar is to be used. The next, whether a part of the protein shall be given in the form of whey protein or not; if not, whether anything is to be done to make the digestion of the casein easier; and, if so, what method is to be adopted. It must then be decided whether the mixture is to be given raw, pasteurized or boiled, and, if pasteurized, at what temperature. After all these points have been settled, the twenty-four hour amount of food must be decided upon and, finally, into how many feedings this quantity is to be divided and at what hours they are to be given. It is advisable after deciding upon the total quantity of the food to calculate its caloric value and the amount of protein which it contains, in order to know whether the caloric and protein needs are being covered or greatly exceeded. In most instances, it will be found that both the caloric and protein needs are covered. It is advisable, as a rule, to give the food which has been decided upon, even if its caloric and protein content do not correspond to the established standards, because, if the composition of the food has been determined to fit the indications in the individual instance, at the given time, the meeting of these indications is usually more important than the caloric value and protein content of the food. The knowledge of the caloric value and protein content of the food is, however, of great assistance in determining the composition of subsequent foods. Everyone of these points must be decided every time that a modified milk mixture is prescribed. No one of them can be omitted. They must be decided, moreover, not by following blindly the rules of some authority on infant feeding or by taking a formula from a table in some textbook, but on the indications in the given case at the given time.

When the composition and the amount of the food have been decided, it may be prepared at a milk laboratory or in the home. When the milk is to be prepared at a milk laboratory, it is only necessary to write a prescription for the food, embodying the points already determined. Most milk laboratories have a prescription form in which it is only necessary to

* With the kind permission of the Macmillan Company, this chapter is copied very largely from the corresponding chapter in the "Diseases of Nutrition and Infant Feeding," by Morse and Talbot.

fill in the blank spaces. This form, while a convenience, is in no way a necessity. The physician who is competent to prescribe modified milk mixtures, has no need of a form. The following is a copy of the prescription form furnished by one of the milk laboratories.

℞ _____

		Per Cent
Fats		
(a) Carbo-hydrates	Lactose (Milk Sugar) Maltose (Malt Sugar) Dextri-Malt Sucrose (Cane Sugar) Starch	
(b) Dextrinize		
(c) Proteins	Whey Casein	
(d) Peptonize.		
(e) Sodium Citrate	% of milk and Cream % of total mixture	
(f) Sodium Bicarb.	% of milk and cream % of total mixture	
(g) Lime Water	% of milk and cream % of total mixture	
(h) Lactic Acid Bacillus	To inhibit the saprophytes of fermentation	
(i) Other Ingredients		

Heat at.....°F.....

Number of Feedings .....

Amount at each Feeding..... oz

ORDERED FOR

....

ADDRESS .....

DATE .....19.....

.....M.D.

There is no doubt that a milk laboratory can prepare mixtures of modified milk more accurately than they can be prepared in the home, because they are able to work with materials of known composition. This cannot be done in the home. Furthermore, as milk laboratories all own their own farms, they can be sure of having a clean milk from which to prepare the food. The individual preparing the food at home, unless able to procure a certified milk, can never be sure whether the milk is clean or not. Mistakes are unquestionably made at times in the milk laboratories, no matter how much care is exercised by those in control of the laboratories, because the employees are human and are, therefore, liable to be careless. I am positive, however, that less mistakes are made in the milk laboratories than by mothers and nurses in the home.

The price of modified milk prepared at milk laboratories is no higher than it should be, when the character of the materials used, the labor required in the modification of the milk and the cost of labor and delivery are taken into consideration. It is, nevertheless, prohibitive for poor people. If the milk has to be sent any distance and express charges

added, only the well-to-do can afford to have it. Comparatively few babies can be fed, therefore, on modified milk prepared at a milk laboratory. The vast majority, either because of expense or distance from a laboratory, must be fed on mixtures prepared in the home. Unfortunately, physicians, at present, do not appreciate, as they should, the value of milk laboratories and the advantages which can be obtained by using them. Two of the reasons for this failure are the general improvement in the milk supply and the present tendency to use whole milk mixtures.

℞ _____

		Ounces
Whole Milk		
Skimmed Milk		
Cream	% Fat	
Lactic Acid Milk	% Fat	
Protein Milk	% Fat	
Dryco		
Whey		
Lactose (Milk Sugar)		
Dextri Maltose No.		
Sucrose (Cane Sugar)		
Dextrose		
Maltose 50%		
Barley Water	%	
Rice Water	%	
Oat Water	%	
Lime Water		
Sodium Citrate	Grs. per oz. Milk and Cream Grs. " " Total Mixture	
Sodium Bicarbonate	Grs. per oz. Milk & Cream Grs. " " Total Mixture	
Boiled Water		
TOTAL		

Heat at .....°F.....

Number of Feedings .....

Amount at each Feeding .....oz

ORDERED FOR

.....

ADDRESS .....

DATE .....19.....

..... M.D.

The Home Modification of Milk

Modifications of milk for infant feeding cannot be prepared as accurately in the home as at a milk laboratory, because it is impossible, in the home, to know the exact composition of the materials used in the preparation of the mixtures. If reasonable care is used in their preparation, however, the inaccuracies are not as great as would be supposed. In the vast majority of instances, they are not great enough to disturb the digestion or to interfere with the nutrition and development of babies fed upon

them, because the average infant does not notice small variations in the composition of an artificial food any more than it does similar variations in the composition of breast milk. Extreme accuracy is necessary only in exceptional cases. In general, therefore, the modifications of milk prepared at home are sufficiently correct for all practical purposes and it is rarely necessary, on this account, to have recourse to a milk laboratory. There is nothing about the calculation of the formulae for the preparation of modified milk in the home which is too complicated for the intelligence of any physician who is competent or which takes more time than he ought to be willing to give to it. There is, moreover, nothing about the procedures involved in the preparation of modifications of milk in the home which are too complicated for the ordinary mother or nursemaid to comprehend and carry out. They are no more difficult than the procedures in ordinary cooking. When mothers or nursemaids make mistakes, it is almost always because the physician, either through ignorance or carelessness, has failed to explain the details of the preparation of the food to them.

Materials Used in the Home Modification of Milk.—As it is impossible, except in rare instances, to analyze the milk and cream used in the preparation of modified milk in the home, it is necessary to adopt certain arbitrary standards as to the composition of these substances. It must be remembered that any form of milk containing more than 4% of fat is technically cream. It is wrong, therefore, to think and speak of cream as a definite entity without qualification. It is more correct to think of creams, with the fat content always specified. The following figures as to the composition of some of the creams, whole milk, skimmed milk and whey are approximately correct.

TABLE XV

	Fat	Milk sugar	Protein
Whole milk	4.00	4.50	3.50
7% cream	7.00	4.45	3.40
10% cream	10.00	4.40	3.25
16% cream	16.00	4.20	3.05
32% cream	32.00	3.40	2.50
Skimmed milk	1.00	5.00	3.55
Separated milk ("fat free")	0.25	5.00	3.65
Whey	0.25	5.00	0.90

If average milk, that is, milk containing 4% of fat is allowed to set six hours or longer, the upper sixteen ounces of a quart of bottled milk contain 7%, and the upper ten ounces, 10%, of fat. The cream layer, that is, "gravity cream," without regard to how many ounces of it there are on a quart, contains about 16% of fat. If the whole milk from which the cream is obtained contains more than 4% of fat, the cream will contain a proportionately larger amount. Ordinary "thick cream," as it is called, contains on the average, 32% of fat. The composition of this type of cream is so variable, however, that it is not safe to use it in the preparation of modified milk in the home, unless the percentage of fat is definitely known.

Skimmed milk is the milk which is left after the gravity cream has been removed by a dipper or by pouring. If some of the upper layers of the milk are removed in addition to the cream, the part which is left

contains less than 1% of fat. Separated ("fat free") milk is the milk which is left when the cream has been removed by centrifugalization. The whey obtained from separated milk contains 1% of protein.

The following table copied from Chapin and Pisek (Diseases of Children, 1909, p. 138) shows the percentage of fat in each of the top nine ounces of a quart of bottled milk which has set six hours or longer and the fat content of the top ounces, from two to thirty ounces, under the same conditions.

TABLE XVI

TABLES SHOWING PERCENTAGE OF FAT IN A QUART BOTTLE OF MILK

First ounce contains	25.0% fat
Second ounce contains	23.0% fat
Third ounce contains	19.0% far
Fourth ounce contains	18.5% fat
Fifth ounce contains	10.5% fat
Sixth ounce contains	4.8% fat
Seventh ounce contains	3.4% fat
Eighth ounce contains	2.2% fat
Ninth ounce contains	1.8% fat
Top 2 ounces mixed contain	24.0% fat
Top 3 ounces mixed contain	22.5% fat
Top 4 ounces mixed contain	21.4% fat
Top 5 ounces mixed contain	19.2% fat
Top 6 ounces mixed contain	16.8% fat
Top 7 ounces mixed contain	15.0% fat
Top 8 ounces mixed contain	13.3% fat
Top 9 ounces mixed contain	11.5% fat
Top 10 ounces mixed contain	10.5% fat
Top 12 ounces mixed contain	9.0% fat
Top 14 ounces mixed contain	7.8% fat
Top 16 ounces mixed contain	7.0% fat
Top 18 ounces mixed contain	6.3% fat
Top 20 ounces mixed contain	5.8% fat
Top 22 ounces mixed contain	5.4% fat
Top 24 ounces mixed contain	5.0% fat
Top 26 ounces mixed contain	4.7% fat
Top 28 ounces mixed contain	4.5% fat
Top 30 ounces mixed contain	4.3% fat

It is also useful to know that the remaining thirty ounces, after the top two ounces have been removed, contains 2.80% of fat; the remaining twenty-eight ounces, after the top four ounces have been removed, 1.85% of fat; the remaining twenty-six ounces, after the top six ounces have been removed, 1.30% of fat; and the remaining twenty-four ounces, after the top eight ounces have been removed, 0.75% of fat.

It is evident that "top milk" may mean any number of ounces, from one to thirty-one, from a quart. It is also evident that the so-called "top milks" are merely creams of varying percentages and that modifications of milk made from "top milks" differ in no way from those made from creams, except in name. Since "top milks" vary as much in their fat content as do creams, it is evidently just as important in prescribing for the preparation of modified milk at home to specify exactly what top milk is to be used as it is to specify what sort of cream is to be used. It is also evident that it is equally important, when part of the cream is removed, to know the fat content of the milk which remains.

Method of Calculation of Formulæ for the Home Modification of Milk.—There are many methods for the calculation of the formulae for

modifications of milk to be prepared in the home. Most of them are inaccurate in that the fat in the skimmed milk is disregarded, many of them in that the percentage of protein in the cream and skimmed milk is considered to be the same. All of them are accurate enough, however, for everyday work. It makes but little difference which method is employed, provided that method is understood and used correctly. Equally good results can be obtained with all. The one important thing is that the food be calculated in percentages of the various food elements. It makes little difference how these elements are obtained. Methods which take the fat in the skimmed milk and the differences in the protein content of the various creams and milks into account are too complicated for ordinary clinical use and are, fortunately, unnecessary.

I have found the following method of calculation a satisfactory one in my own practice and also that medical students understand it quickly and apply it easily. These are its chief recommendations. It is unquestionably inaccurate in many ways, as are all simple methods of calculation. It must be remembered, however, in criticising methods of calculation for their inaccuracies, that, if the same method is used consistently, the inaccuracies are always similar and that different modifications of milk prepared by the same method are accurate relatively to each other. That is to say, if a baby, who is taking a mixture supposed to contain 3.50% of fat, but which really contains 4% of fat, shows symptoms of fat indigestion, a reduction of 0.50% in the percentage of fat will have the same effect in relieving these symptoms, although it is a reduction from 4% to 3.50% instead of one from 3.50% to 3%, as it is supposed to be. That is, changes in the percentages are correct, even if the given percentages are incorrect.

Gravity cream and skimmed milk are used in this method. The gravity cream is estimated to contain 16% of fat and the skimmed milk to be fat free. The mixtures, therefore, all contain a somewhat higher percentage of fat than they are supposed to contain. The protein content of both the gravity cream and skimmed milk is calculated to be 3.20%. This percentage is higher than that really present in the cream and lower than that in the skimmed milk. Numerous analyses, made by the Kjeldahl method, of the protein content of mixtures prepared in this way have shown, however, that the percentage of protein in them is not far from what it is calculated to be. The percentage of sugar is estimated at 4.50 in both the gravity cream and skimmed milk.

By *gravity cream* is meant all the cream which is visible on milk which has set for six hours or longer. All the cream must be removed and the required number of ounces taken from it. If there is not enough cream on one bottle, the cream must be removed from two bottles and the required number of ounces is then taken from the mixture of the two. The cream should be removed with a cream dipper. The results obtained by pouring are not nearly as accurate as those obtained by dipping. The same result may be obtained by siphoning off the milk below the cream and leaving the cream in the bottle. Most women, however, find siphoning a more difficult operation than dipping. Most bottled milk has been in the bottles many more than six hours before it is delivered. When the milk bottle is full, the cream rises even during transportation. It is not necessary, therefore, to wait six hours after the milk is delivered before preparing the food, provided the cream line is distinct.

By *skimmed milk* is meant what is left after the gravity cream has been removed. The percentage of fat in the mixture will be more nearly

correct if the lowest ounces are used instead of the same number of ounces from the whole of the skimmed milk.

A rounded tablespoonful of milk sugar is considered in this method of calculation to weigh one half ounce. It will be found that this is not far from the true weight. By a rounded tablespoonful is meant what is contained in a tablespoon when it is dipped into milk sugar and then gently shaken. That is, it is rounded, not heaped nor level. Every cook knows what is meant by this term. The weight of equal quantities of milk sugar and the maltose-dextrins mixtures is nearly enough the same for practical purposes. If it is desired to use a level tablespoonful instead of a rounded tablespoonful, between three and four tablespoonfuls is equal to an ounce by weight.

The estimated composition of the materials used in the preparation of the mixture is, therefore, as follows:

TABLE XVII

	Fat	Milk sugar	Protein
Gravity cream	16.00%	4.50%	3.20%
Skimmed milk	0.00%	4.50%	3.20%
Milk sugar	1 rounded tablespoonful = ½ ounce.		

It is necessary, of course, to decide what percentages of the different food elements the food is to contain and how much of it is to be prepared for the twenty-four hours before beginning the calculations as to the amount of the different ingredients to be used. It is usually advisable to make the quantity large enough to allow for an extra bottle, so that, if a bottle is broken, the baby need not go hungry.

Suppose that it is desired to prepare thirty-two ounces of a mixture containing 3% of fat, 6% of milk sugar and 2% of protein. The fat in the food must be derived from the cream, because that is the only substance containing fat to be used in the preparation of the food. If the food was composed entirely of gravity cream, it would contain 16% of fat. Since it is to contain but 3% of fat, it is evident that only three sixteenths of the mixture can be gravity cream. Three sixteenths of 32 ounces = 6 ounces. Six ounces of gravity cream will, therefore, provide the 3% of fat desired in the mixture.

The gravity cream contains protein as well as fat. There are six ounces of gravity cream in the thirty-two ounce mixture. The protein content of gravity cream is 3.20%. The protein content of a thirty-two ounce mixture containing six ounces of gravity cream is evidently $\frac{6}{32}$ of 3.20%, or 0.60%. Two per cent. of protein is, however, desired in the mixture. The gravity cream has provided only 0.60%. One and forty hundreths per cent. of protein, the difference between the percentage of protein desired and that furnished by the gravity cream, must be obtained in some other way. It must be obtained, moreover, from some substance which does not contain fat. Skimmed milk is such a substance. Skimmed milk contains 3.20% of protein. In order to get 1.40% of protein in the mixture by the use of skimmed milk, it is evident that $\frac{1.40}{3.20}$ of the mixture must be skimmed milk. $\frac{1.40}{3.20}$ of 32 ounces = 14 ounces. Fourteen ounces of skimmed milk will, therefore, provide the additional 1.40% of protein desired.

Both gravity cream and skimmed milk contain 4.50% of milk sugar. Twenty ounces of gravity cream and skimmed milk are required to furnish the desired percentages of fat and protein. These twenty ounces in a thirty-two ounce mixture must add $^{20}/_{32}$ of 4.50% of sugar to the mixture. Twenty thirty-seconds of $4\frac{1}{2}$, or $^{20}/_{32}$ of $^{9}/_{2} = {}^{180}/_{64}$, or practically 3% of milk sugar. It is, however, desired to have 6% of milk sugar in the mixture; that is, 3% more of milk sugar is required. This additional sugar must be added in the form of dry milk sugar. Three per cent. of thirty-two ounces is $^{3}/_{100}$ of 32. This will give the amount of sugar desired in ounces. The sugar is to be measured in rounded tablespoonfuls or half ounces. If the figures given above are multiplied by two, the result will be the number of rounded tablespoonfuls needed. That is, $^{3}/_{100}$ of $32 \times 2 = {}^{192}/_{100}$ rounded tablespoonfuls, or, for all practical purposes, two rounded tablespoonfuls.

There are twenty ounces of cream and milk in the mixture. The total quantity of the mixture is to be thirty-two ounces. The difference between thirty-two ounces and twenty ounces is twelve ounces. Twelve ounces of water must be added, therefore, to make up the desired amount. The milk sugar goes into solution and, therefore, does not add to the amount. The milk sugar should be dissolved in the water while it is hot. The sugar solution should be allowed to cool before it is mixed with the other ingredients.

If it is desired to add lime water to the mixture, for example, in the proportion of 25% of the cream and milk in the mixture, five ounces would be required, that is, 25% of the twenty ounces of gravity cream and skimmed milk. Only seven ounces of water would then be necessary to make up the required amount. The effect of lime water is not prevented by pasteurization, but is destroyed by boiling. Lime water should not be added, therefore, to a mixture before it is boiled. It is unnecessary, anyway, to add lime water after a mixture has been boiled, because the effect of the boiling on the casein makes it unnecessary to add lime water.

The results of the steps just described are shown in figures below.

TABLE XVIII

	Ounces	Fat	Sugar	Protein
Gravity cream	6	3.00	3.00	0.60
Skimmed milk	14			1.40
Milk sugar	2 rounded tablespoonfuls		3.00	
Water	12			
	32	3.00	6.00	2.00

Mixtures Containing Starch.—It is almost as important to have the percentage of starch in the food correct as to have those of the fat, sugar and protein. Starch is usually added in the form of the cereal waters. The strength of the cereal water which is to be used in the preparation of the food must be known, therefore, in order to get the desired percentage of starch in the mixture.

Two rounded teaspoonfuls of barley or oat flour to the pint of water have been found by analysis to give a 1.50% decoction of starch, while four rounded teaspoonfuls to the pint of water gives a 3% decoction.

The flour should be mixed with a small amount of water, after which the remainder of the water is added. The mixture is then boiled for twenty minutes, after which, as some of the water has boiled away, enough water is added to make up the original pint. It should then be strained through several thicknesses of cheesecloth. It should be cooled, but not enough to make it jelly, before being mixed with the milk and cream.

If it is desired to have 0.75% of starch in the mixture and a cereal water containing 1.50% of starch is to be used, it is evident that one half of the mixture must be made up of the cereal water. If a 3% cereal water is used, one quarter of the mixture will be required to give 0.75% of starch. Suppose that it is desired to have 0.75% of barley starch in the mixture which has just been calculated. In order to get 0.75% of starch in a thirty-two ounce mixture, using 1.50% barley water, it will be necessary to use $\frac{0.75}{1.50}$ of 32 ounces, or sixteen ounces. The mixture contains twenty ounces of gravity cream and milk, leaving a balance of only twelve ounces. It is evident, therefore, that, as sixteen ounces of 1.50% barley water are required to get 0.75% of starch in the mixture and there is only room for twelve ounces, it is impossible to have 0.75% of starch in the mixture, if 1.50% barley water is used. If 3% barley water is used, $\frac{0.75}{3.00}$ of 32 ounces, or eight ounces, will be required. There being room for twelve ounces of diluent, the 0.75% of starch can be introduced in this way. As only eight ounces are used and there is room for twelve, four ounces of water must be added.

If preferred, the amount of starch to be added to the mixture to give any percentage of starch required in the mixture may be calculated directly. Suppose for example, it is desired to have 0.75% of starch in a forty-eight ounce mixture. Two rounded teaspoonfuls of flour to the pint, gives 1.50% of starch in the mixture. One rounded teaspoonful to the pint, gives 0.75% of starch in the mixture. There are three pints in forty-eight ounces. Therefore, three rounded teaspoonfuls of flour will be required to give 0.75% of starch in forty-eight ounces. This amount of flour should be cooked in the number of ounces of water in the mixture and then mixed with the gravity cream and skimmed milk.

Whey Mixtures.—It is impossible to make mixtures containing a high percentage of whey protein with a low percentage of casein, provided they contain more than 1 or 2% fat, if gravity cream is used in the preparation of the food, as it usually is in the home. The explanation is that the gravity cream, which it is necessary to use in order to get the desired percentages of fat, contains a considerable amount of protein and by its bulk diminishes the amount of whey, and consequently the amount of whey protein, which can be added. It is usually desirable, when whey protein is prescribed to have as much of it in a mixture as is possible. For practical purposes, therefore, when whey mixtures are prepared in the home with gravity cream, the amount of gravity cream required to give the desired percentage of fat is calculated and the rest of the mixture made up with whey, the amount of whey protein added being determined later. Smaller percentages of whey protein can be added, of course, if desired.

Suppose that it is desired to give a baby a twenty-four ounce mixture containing 3% of fat and 6% of sugar. Three sixteenths of 24 ounces =

4½ ounces. Four and one half ounces of gravity cream will, therefore, be required. This will put $\frac{4\frac{1}{2}}{24}$ of 3.20% or 0.60%, of protein in the mixture. This protein is chiefly in the form of casein. It is evident that there is room for 19½ ounces of whey in the mixture, the difference between twenty-four and four and one half being nineteen and one half. Whey, for practical work in the home modification of milk, may be calculated to contain 0.90% of whey protein. $\frac{19\frac{1}{2}}{24}$ of 0.90% gives a little more than 0.70% of whey protein, which is, therefore, the amount added by the whey. Both the gravity cream and the whey contain 4.50% of milk sugar. The whole mixture being made up of gravity cream and whey, contains, therefore, 4.50% of milk sugar. It being desired to have 6% of milk sugar in the mixture, the difference between 6.00% and 4.50%, or 1.50%, must be added in the form of dry milk sugar. $\frac{1\frac{1}{2}}{100}$ of $24 \times 2 = 0.72$ of a rounded tablespoonful. A level tablespoonful of milk sugar will, therefore, just about make up the required percentage of sugar.

The mixture contains 3% of fat, 6% of sugar, 0.70% of whey protein and 0.60% of casein. It is evident, therefore, that, if gravity cream is used, it is impossible to get less than 0.60% of casein in the mixture with 3% of fat, or less than 0.80% of casein with 4% of fat. The percentage of whey protein in the mixture is really somewhat higher and that of the casein somewhat lower than has been calculated, because about one quarter of the protein furnished by the cream is in the form of whey protein. It is not necessary for everyday work, however, to take this small difference into consideration.

Higher percentages of whey protein and lower percentages of casein can be obtained with given percentages of fat, if creams containing higher percentages of fat are used. It is possible, for example, even in the home, to get cream containing 24% of fat by taking only the top two ounces of the quart. It is also possible to have any percentage of casein desired with the given percentage of fat by using skimmed milk in the mixture. The amount of whey protein which can be put in the mixture is, of course, correspondingly diminished. When it is desired to work off of a whey mixture on to an ordinary mixture, without increasing the total amount of protein, it is best done by gradually replacing the whey by skimmed milk and water. One ounce of skimmed milk and three ounces of water contain approximately the same amount of protein as four ounces of whey.

Whey is made by putting a pint of skimmed milk into a clean saucepan and heating it until it is lukewarm (not over 100° F.). Take off of the stove. Add two teaspoonfuls of essence of pepsin or liquid rennet or two junket tablets. Stir just enough to mix. Let it stand until firmly jellied. Then break up with a fork until it is finely divided. Strain through a linen cloth or several thicknesses of cheesecloth. What goes through is whey. If whey is to be mixed with cream, milk or skimmed milk, it must be brought to 150° F. in order to kill the rennin. If whey is not brought to this temperature before it is added to milk or cream, the rennin in it will curdle them.

The Pancreatization of Modified Milk.—After the mixture is prepared, it can be pancreatized by the addition of one of the so-called "peptonizing" powders or tablets. The food may be heated at "blood heat"

(not over 115° F.) for ten minutes, then brought quickly to a boil. The ferments are destroyed by the boiling and the food, therefore, will not become bitter. It is better, however, to add a part of the contents of a "peptonizing tube" or part of a tablet to each feeding just before it is to be used. The feeding is then heated for from ten to fifteen minutes at "blood heat," or from 100° F. to 115° F., being allowed to drop to 100° F. toward the end of this time, and immediately given to the baby. The advantage of this method is that the ferments are still active when the food is ingested. The contents of a "peptonizing" tube or a "peptonizing" tablet are usually intended for the pancreatization of a pint of milk. The proportion of milk in each feeding being known, it is a simple matter to calculate how much of the powder or tablet to add. "Peptonized milk" prepared by the so-called "cold process" is, of course, not predigested at all, because the pancreatic enzymes do not act in the cold. The only opportunity which they have to act, when milk is prepared by this process, is after they are taken into the stomach. Their action ceases, however, when the reaction of the stomach contents becomes acid.

Whole Milk Mixtures.—When whole milk mixtures are used, it is just as important to know and calculate the percentages of the different food elements in the mixtures as when they are prepared with cream and skimmed milk. If it is desired to have 2% of fat in the mixture, since whole milk contains 4% of fat, two fourths or one half of the mixture must be milk. The percentage of protein must, of course, vary directly with that of the fat. If one half of the mixture is milk, the percentage of protein is one half of 3.50, the percentage of protein in whole milk, or 1.75%. The percentage of sugar is, of course, likewise one half of that in whole milk, or 2.25%. If it is desired to have a higher percentage of sugar in the mixture, it can be added in the form of dry sugar. Suppose, for example, it is desired to have 7.25% of milk sugar in a whole milk mixture containing 2% of fat, and the total quantity of the mixture being forty ounces. The difference between 7.25% of sugar, the amount desired, and 2.25%, the amount provided by the milk, is 5%. Five per cent. in forty ounces is $\frac{5}{100}$ of 40 or 2 ounces. Two ounces is equal to four rounded tablespoonfuls.

Skimmed Milk Mixtures.—The same principles are applicable when skimmed milk mixtures are used. The fat in the skimmed milk can be estimated at 1.50% or at 0, according to how great a degree of accuracy is desired.

The Determination of Percentages in Mixtures.—It is often of great importance to find out just what a baby has been taking in order to know how to change the food, if it is not agreeing with it. To do this, it is necessary to determine the percentages of the different elements in the food. This is not a difficult matter. Suppose that a baby is taking a food made up as follows: gravity cream, twelve ounces; skimmed milk, eighteen ounces; lime water, six ounces; barley water, twelve ounces; milk sugar, four rounded tablespoonfuls. The barley water is made with two rounded teaspoonfuls of barley flour in a pint of water.

The total quantity of the mixture is forty-eight ounces. Gravity cream contains 16% of fat. Twelve ounces of gravity cream in a forty-eight ounce mixture will give, therefore, $\frac{12}{48}$ of 16% of fat, or 4% of fat. Both gravity cream and skimmed milk contain 3.20% of protein. There are thirty ounces of gravity cream and skimmed milk in the mixture. Thirty ounces in a forty-eight ounce mixture will give

$^{30}/_{48}$ of 3.20% of protein, or 2.00% of protein. Both the gravity cream and skimmed milk also contain 4.50% of sugar. Thirty ounces of gravity cream and skimmed milk in a forty-eight ounce mixture will, therefore, furnish $^{30}/_{48}$ of $4\frac{1}{2}$, which is the same as $^{30}/_{48}$ of $^{9}/_{2}$, or almost 3.00% of milk sugar. Four rounded tablespoonfuls of milk sugar are equal to two ounces. Two ounces of sugar in a forty-eight ounce mixture is equal to $^{2}/_{48}$ of 100%, or 4%. The total percentage of sugar is, therefore, 7%. Two rounded teaspoonfuls of barley flour in a pint of water makes a 1.50% decoction of starch. Twelve ounces of barley water of this strength in a forty-eight ounce mixture gives $^{12}/_{48}$ of 1.50%, or about 0.40% of starch. There are six ounces of lime water in the mixture and thirty ounces of gravity cream and skimmed milk. Six thirtieths of 100% = 20%. The lime water in the mixture is, therefore, 20% of the milk and cream. The mixture thus contains 4% of fat, 7% of milk sugar, 2% of protein and 0.40% of starch, while the lime water is present in the proportion of 20% of the cream and milk.

The Determination of the Caloric Value of Mixtures of Modified Milk.—The method given below is longer than some of the other methods in common use. It is more accurate than many of them, however, and has this in its favor, namely; that it is impossible to carry it out without fully understanding what the caloric value of food really means.

Suppose that a baby is taking thirty ounces of a food containing 4% of fat, 6% of sugar, 2.25% of protein and 0.75% of starch. Thirty ounces is equal to 900 c.cm. Four per cent. of fat means that there are four grams of fat in each 100 c.cm. of food. The baby is taking 900 c.cm. of food, that is, it is taking nine times the amount of fat in 100 c.cm. of food, or nine times four grams, which is thirty-six grams. The caloric value of one gram of fat is 9.3 calories. Thirty-six grams of fat will give thirty-six times 9.3 calories, which is 334.8 calories.

The caloric value of sugar, starch and protein is the same, each gram yielding 4.1 calories. The caloric value of these elements can, therefore, be calculated at the same time. There are 6 grams of sugar, 2.25 grams of protein and 0.75 grams of starch, or a total of 9 grams in each 100 c.cm. There are, therefore, nine times 9 grams, or 81 grams, in 900 c.cm. Each gram is equivalent to 4.1 calories. 81 grams provide 81×4.1 calories or 332.1 calories. The sum of the 334.8 calories furnished by the fat and the 332.1 calories furnished by the sugar, starch and protein is 666.9 calories, which is, therefore, the caloric value of the mixture.

There are various other methods for calculating the caloric values of milk mixtures. A simple, but less accurate, method is that recommended by Fraley (Archives of Pediatrics. 1912. **29,** 123). Letting F equal the percentage of fat, S equal the percentage of sugar and starch, P equal the percentage of protein and Q the total quantity of food, then $2F + P + S \times 1\frac{1}{4}Q$ = Calories. This formula always gives the caloric value a little lower than it really is. It gives, for example, 637 calories as the value of the food just calculated above, when the real value is 666.9 calories.

The caloric value of a food is of importance only in its relation to the weight of the baby that is taking it. Suppose that the food whose caloric value has just been calculated is being taken by a baby that weighs eleven pounds. Dividing the number of calories in the food by the weight gives the number of calories which it gets per unit of weight. That is, 666.9 calories divided by eleven gives sixty calories, which is the number

of calories which the baby is getting per pound of weight. A kilogram is equal to $2\frac{2}{10}$ pounds. Eleven pounds is equal, therefore, to five kilograms. Dividing 666.9 calories by five gives the number of calories which the baby is getting per kilogram of body weight, that is, 133.

Determination of the Protein Content of Milk Mixtures.—It is very easy to determine the protein content of a food by using the same principles employed in estimating the caloric value. Suppose that a baby weighing fifteen pounds is taking forty-eight ounces of a food containing 2.50% of protein. Forty-eight ounces is equal to 1440 c.cm. There are 2.5 grams of protein in each 100 c.cm. of food, or 14.4×2.5 grams in the whole amount. 14.4×2.5 grams = 36 grams. The baby weighs fifteen pounds. It gets, therefore, 2.4 grams of protein per pound of body weight in this food. Fifteen pounds is 6.8 kilograms. Dividing thirty-six grams by 6.8 gives 5.3 grams, which is the amount of protein which the baby gets per kilogram of weight from this food.

SPECIAL PREPARATIONS OF MILK AND THE PROPRIETARY FOODS

Special Preparations of Milk.—Many special preparations of milk are now being used in feeding infants with, according to the statements of those who use them, remarkably favorable results. It is rather difficult, in many instances, to know whether a special food does or does not belong among the proprietary foods.

Buttermilk.—Buttermilk made from sweet milk in the manufacture of butter is, of course, nothing but skimmed milk. It contains from 0.50% to 1.00% of fat, about 4.50% of milk sugar and 3.80% of protein. The proportions of the casein and the whey protein are the same as in whole milk. When buttermilk is made from sour cream in the manufacture of butter, its composition is not constant. Average figures are: fat, 0.50% to 1.00%; milk sugar, 3.00% to 3.50%; protein 2.50% to 2.70%. The proportion of whey protein is relatively higher than in whole milk. The casein is finely divided as the result of centrifugalization and is separated to a certain extent from its calcium base. It is no longer acted upon by rennin. The caloric value of buttermilk varies between 300 and 400 calories per litre. A fair average figure is 360 calories per quart. Good buttermilk should not contain over 0.50% of lactic acid. The acidity increases with time, although it rarely reaches over 0.75%, at which point the buttermilk separates into curds and whey. The lactic acid organisms which cause the souring of the milk, as well as other organisms, are alive and active. It is evident that, if the milk is first boiled and then pure cultures of lactic-acid-forming organisms added to it to sour it, the resulting buttermilk will contain a purer flora than when the milk is soured naturally. The organism most commonly used is the B. Bulgaricus. It is liable, however, to make the taste of the milk too acid. Another organism, perhaps better, is the B. acidi paralactici. Better results are usually obtained with a "starter" than by the use of a new culture each time. Cultures are preferable to tablets, many of which are inert. Heating buttermilk destroys the fine division of the casein and causes it to clot in large masses like ordinary cow's milk. It also destroys the bacteria which it contains. The clotting during heating may be prevented by constant, violent stirring or beating.

Buttermilk has been used as a food for infants since at least as early as 1770. There is no doubt that good results have been obtained with

it. Most of those who have used it, however, have added from ten to twenty-five grams of flour, usually wheat, and from thirty-five to ninety grams of cane sugar to the litre of buttermilk and then boiled it with much stirring. It is evident that the nutritive value of the butter-milk is materially increased by these additions. The caloric value of buttermilk prepared in this way is between 525 and 700 calories per litre, according to the amount of flour and sugar added. The bacteria which it contains are, however, destroyed.

There are several possible explanations for the good results obtained with buttermilk in infant feeding. Its relatively low content of fat and sugar and its relatively high content of protein are sufficient to account for its usefulness when there is an intolerance for fat or sugar. The finely divided form of the casein and the fact that it is no longer acted upon by rennin make the digestion of the casein easier. In such instances the presence of living lactic-acid-forming organisms in the food is probably not of any great advantage. They are, however, antagonistic to the growth of the gas bacillus and similar organisms and to that of proteolytic bacteria. Buttermilk ought to be useful, therefore, in the treatment of disturbances of digestion and infections of the intestinal tract caused by these organisms. It is of no advantage in these conditions, however, if it is pasteurized or boiled, because the lactic-acid-forming bacteria are destroyed. When starch and sugar are added to buttermilk and the mixture boiled, it loses all the advantages due to its low sugar content and the living organisms which it contains. The casein is, however, still in a form relatively easy of digestion. It is very probable, in the light of recent investigations as to the optimum hydrogen-ion concentration for gastric digestion in infancy and the favorable effect of lowering the buffer value of cow's milk by the addition of acid, that part, at least, of the good results obtained with buttermilk are due to the lactic acid which it contains, from 0.50% to 0.70% being the most favorable degree of acidity.

Acidified Milk.—The addition of acid to cow's milk has been especially advocated by Faber and by Marriott and his coworkers. Marriott found that the average hydrogen-ion concentration at the height of digestion in normal infants fed on human milk was 3.75%, while in infants fed on undiluted cow's milk it was 5.10%. By the addition of acid to cow's milk, it is possible to overcome the greater "buffer" action of cow's milk, so that the hydrogen-ion concentration at the height of digestion is the same as with breast milk. He found that, when milk containing from 0.50% to 0.70% of lactic acid was fed to normal infants, the hydrogen-ion concentration of the gastric contents at the height of digestion was almost the same as when breast milk was given. He also found that the addition of pure lactic acid had the same effect as when the acidity was produced by bacteria. The addition of acid is preferable to the souring by bacteria, because it is simpler and more accurate and there is no chance for contamination with other organisms. He prefers lactic acid to hydrochloric acid, because the organic acids are completely oxidized in the body, while the inorganic acids are not destroyed and must be eliminated, throwing a rather severe strain on the acid-base regulating mechanism, and possibly leading to acidosis. Faber and others, however, use hydrochloric acid and have had no unfavorable results from it. Faber recommends 25 c.cm. of normal (about 0.4%) hydrochloric acid to 100 c.cm. of milk. Marriott thinks that lactic acid is preferable to acetic, citric and butyric acids, because they tend to cause diarrhea.

Others, however, have used these acids, either plain or in the form of vinegar or fruit juices, with, to them, satisfactory results.

Acidified Whole Milk.—Marriott and others recommend acidified whole milk as a routine food for infants and report very favorable results from it. The food which they recommend is as follows: a good grade of cow's milk is first sterilized by boiling for five minutes. The milk is then cooled and the scum removed. One drachm of U. S. P. lactic acid is added for each pint of milk, a drop at a time. The milk should be cool, because, if it is warm when the acid is added, or if the acid is run in too rapidly, large clumps of curds will separate. When properly prepared, a smooth, homogeneous preparation should result. The taste, general physical appearance and concentration of acid is the same as in ordinary buttermilk. Its acidity is p_{H4}. This degree of acidity almost completely inhibits bacterial growth. Milk prepared in this way keeps well, even if not placed in a refrigerator. An ounce of corn syrup is added to the day's feeding for infants up to two weeks of age and from one and one half to two ounces for older infants. The syrup is stirred into the lactic acid milk until thoroughly mixed. They give six feedings a day at four hour intervals to young or undernourished infants, and five feedings otherwise. The amount at a feeding is determined by the appetite and symptoms presented by the baby. Roughly, the average infant takes two ounces at one week, three or four ounces at a month, six ounces at four months and seven or eight ounces from the sixth month on.

They believe that it is important not to give infants dilute foods, that the reason that the strength of most mixtures has to be varied with the age of the infant is because of the incapacity of young infants to digest sweet cow's milk, and that when the high buffer value of cow's milk is diminished by the addition of acid, cow's milk is as easily digested by babies as human milk.

The food which nature produces for a baby is a dilute one. It seems rational to believe, therefore, that a dilute food is better fitted for the human infant's digestive capacity and metabolic processes than a concentrated one. Human milk contains a relatively large amount of fat compared with protein. An excess of protein throws an unnecessary strain on the organs of elimination, at least, if it does not do harm in other ways. This unnecessary strain is not obviated by diminishing the buffer value of cow's milk and thus making gastric digestion easier. If large amounts of protein are not given, it is usually not necessary to thus diminish the buffer value of the milk. So quickly absorbable a sugar as corn syrup seems less suitable for the development and maintenance of the normal intestinal flora than milk sugar. It is impossible to know, moreover, what the effect of modifying the gastric digestion may have on the intestinal digestion.

It seems to me that it is inadvisable to use a routine food and also inadvisable to use whole milk mixtures. The reason for my beliefs have already been stated. Babies that have been fed in this way have been brought to me usually for one of three reasons: they were started on it when only a few days or weeks old and were immediately upset; after thriving on it for a time, they had begun to fail; they were markedly constipated. It seems to me, nevertheless, that the addition of lactic or some other acid to a mixture, which has been prepared to fit the needs of the individual infant at the given time, may be very useful by making the digestion of the casein in the mixture easier. All the advantages of the addition of acid to milk can be attained, therefore, by the addition

of lactic acid, in the proportion of four drops to the ounce of milk, without any of the disadvantages of a routine food with a high casein content. The use of corn syrup, which contains a relatively large amount of easily absorbable carbohydrate, is also very useful, when it is important to give a carbohydrate which is quickly absorbed. As a routine sugar, however, it is not as suitable as milk sugar.

Protein Milk.—(Eiweissmilch, albumin milk) Finkelstein and Meyer, because of their belief that sugar was the especial and primary cause of intestinal fermentation and that the fermentation of the sugar depended on the concentration of the whey and the relative proportions of casein and sugar in the mixture, developed a food to prevent the fermentation of sugar. They called it Eiweissmilch. This food is prepared as follows: Heat one quart of whole milk to 100° F. Add four teaspoonfuls of essence of pepsin and stir. Let the mixture stand at 100° F. until the curd is formed. Put the mass in a linen cloth and strain off the whey from the curds. Remove the curd from the linen cloth and press it through a rather fine sieve two or three times with a wooden mallet or spoon. Add one pint of water to the curd during this process. The mixture should now look like milk and the precipitate must be very finely divided. Add one pint of buttermilk to this mixture. The buttermilk is added because of the small amount of milk sugar which it contains and the beneficial action of lactic acid and because buttermilk can be kept for a long time. This food contains 2.50% of fat, 1.50% of sugar, 3.00% of protein and 0.50% of salts. One quart contains about 370 calories. They do not limit the use of this food to intestinal fermentation from sugar, but claim that it is worthy of employment in all the disturbances of nutrition in infants which are accompanied by diarrhea, no matter of what sort or variety. They soon found, however, that, because of its low caloric value, babies did not thrive on this mixture for any length of time and, therefore, recommend the addition of one of the combinations of maltose and the dextrins to it, as soon as possible. It is very hard to prepare protein milk in the home, although it is not a difficult matter in milk laboratories or institutions. Consequently, various makers have manufactured a powdered protein milk. The composition of the Merrill-Soule powdered protein milk is fat 27%, lactose 24%, protein 38%, ash 5%. The total acidity is 5.75%. When one part of this powder, by weight, is mixed with eleven parts of water, by weight, the composition of the mixture is—fat 2.25%, lactose 2.00%, protein 3.16% and ash 0.42%. The total acidity is 0.48% and it contains eleven calories per ounce. Hoos' albumin milk, when mixed with water, is said to contain—fat 2.50%, milk sugar 1.50%, protein 3.00%, salts 0.40%, plus lactic acid. Mead, Johnson & Company's powdered protein milk contains, according to the makers' analysis, fat 30.90%, carbohydrates 19.10%, protein 37.00%, ash 4.60% and free lactic acid 3.00%.

The principle of the treatment of fermentative conditions caused by sugar with a food low in sugar and salts and high in protein is a rational one, as is the substitution of the maltose-dextrins mixtures for lactose. It hardly seems rational, however, to believe that all disturbances of nutrition accompanied by diarrhea are due to the same cause and should be treated in the same way. Neither does it seem reasonable to believe that any single food is suitable for all babies, whether sick or well, without regard to their individual digestive capacities.

It is possible to take advantage of the main principles of this method of treatment of the intestinal fermentative conditions and at the same

time avoid the disadvantages of a routine food by using mixtures prepared with cream containing a high percentage of fat and by adding casein to the mixture. By using cream containing a high percentage of fat, the percentages of the salts and sugar can be kept low. The casein can be added in the form of precipitated casein, prepared according to Finkelstein and Meyer's method, or as one of the numerous dried and powdered casein preparations on the market. The amount of sugar can be increased at will by the addition of any desired form of sugar. It is easier to use one of the powdered caseins than to make precipitated casein. Some of the casein powders are: Larosan, Protolac, Casec, and Aprotein. Larosan contains 2.18% of fat, 82.35% of proteins, and 5.67% of ash (Mellin's Food Company). Protolac contains 2.86% of fat, 80.61% of proteins and 6.77% of ash (Mellin's Food. Co.). Casec contains 1.50% of fat, 89.00% of proteins and 5.00% of ash (Manufacturer's analysis). Aprotein contains 0.43% of fat, 3.78% of lactose, 81.47% of protein and 5.90% of ash (Manufacturer's analysis). It is evident that, when it is desired to give a food almost entirely protein, these powders may be given mixed with water.

Dried Milk.—There is nothing new about the use of dried milk in infant feeding. It has been used for many years in a number of proprietary foods, such as Nestlé's, Allenbury's and Mammala, in combination in them, of course, with other things. The use of dried milk powders is, however, relatively new. There are a number of different brands on the market. The analysis depends on how much, if any, cream was removed from the milk before it was dried. The Dryco brand is made from partly skimmed milk and contains, according to the analysis of the makers, approximately 12% of fat, 46% of lactose, 32% of protein and 7% of salts. One ounce, by weight, contains 127 calories and one level tablespoonful, sixteen calories. Eight level tablespoonfuls are said to weigh one ounce. One level tablespoonful of Dryco in one ounce of water makes a mixture containing 1.50% of fat, 5.50% of sugar and 4.00% of protein. Klim powdered whole milk contains, according to the makers, fat 28%, lactose 38%, proteins 26.74,% and ash 5.76%. The caloric value of an ounce is 149. One level tablespoonful in two ounces of water gives a mixture containing 3.50% of fat, 4.70% of sugar, and 3.30% of protein, with a caloric value of twenty to the ounce. Klim skimmed, dried milk, according to Root (Charlotte Medical Journal. May, 1920) contains fat 1.35%, milk sugar 50%, proteins 38%, salts 8%. One of the newest dried milk preparations, Momilk, contains, according to the makers, fat 10%, lactose 47%, protein 33% and ash 7%.

It is evident that dilutions of these preparations will contain a relatively low percentage of fat, so low as to be almost negligible in the Klim skimmed dried milk, a relatively very high percentage of protein and a fairly high percentage of sugar. Babies suffering from fat indigestion naturally do well on them. The only real advantage in these preparations over mixtures of fresh milk containing the same percentages is that the casein in them is perhaps more easily digested. The relationship between the fat and protein in dried milk dilutions cannot be changed, however, while it can be in the fresh milk mixtures. It is claimed that the casein is divided into minute particles during the process of drying, that it cannot, for this reason, be coagulated into large curds by rennin and that, on this account, it is more easily digestible than the casein of fresh milk. This sounds reasonable and is probably true, but I have not seen any chemical or experimental proof of these assertions. It is doubt-

ful, moreover, if the casein is in any more easily digestible form than when it is acted upon by citrate of soda or when the milk is boiled. The nitrogen absorption and utilization are probably as good on dried as on fresh milk, but no better. It is not free from bacteria, but the number is smaller than in the milk from which the powder was prepared. It would seem justifiable to suspect that the vitamins of the milk might be injured in the process of drying and that this might lead to the development of some of the deficiency diseases. Hess and Unger have shown, however, that some dried milk, at least, has antiscorbutic power. How much it contains depends upon the age of the milk before it is dried, the time of year it is obtained, the feed of the cows, the method of preparation and possibly how long it is kept before it is used. The lack of the antiscorbutic vitamin is of but little importance, however, as the development of scurvy can be easily prevented by giving orange juice or some other antiscorbutic.

It is evident that dried milk will prove useful in the same conditions that dilutions of whole milk, partially skimmed milk and skimmed milk are useful, that is, in conditions where it is of advantage to have a relatively low sugar in combination with a relatively high protein. The dried milk dilutions are more easily digested than dilutions of whole milk or skimmed milk, unless something has been done to them to make the digestion of the casein easier. There is, however, a very distinct field of usefulness for the dried milk preparations, that is, in countries where it is impossible to get pure milk or to take care of it properly and in travelling. Dried milk does not spoil in the can and the mixture can be made fresh each time just before it is to be used.

Synthetic Milk Adapted.—This preparation, advised by Gerstenberger, is on the market under the name of S.M.A., in both a concentrated and powdered form. A protein S.M.A. acidulated is also on the market. According to the manufacturers' advertisements, the composition of this food, when it is prepared according to directions, is fat 3.50% to 3.60%, carbohydrate 7.20% to 7.50%, protein 1.20% to 1.30%, ash 0.25% to 0.30%. It is said that the p_H varies between 6.8 and 7.0. The caloric value of an ounce is twenty.

S.M.A. is an effort to imitate human milk and to prepare a single food which is suitable for all babies at all times. In order to imitate the composition of human milk, butter fat is almost entirely eliminated from the food and replaced by a mixture of approximately 10% of tallow oil, 15% of cocoanut oil, 20% of cocoa butter, 10% of cod liver oil and from 45% to 55% of tallow. These fats were chosen in order to obtain saponification, iodine, Reichert-Meissl and melting point values like those of the fat of human milk and to provide an adequate amount of the fat soluble vitamin. The cod liver oil is introduced especially as a prophylactic against rickets. The whole mixture is homogenized.

This food is open to the objections to which all foods whose constituents cannot be changed to fit the indications in the individual case are open. It is very doubtful whether having the characteristics of the fat like those in human milk is of any great advantage. On the other hand, it does correspond in the relative proportions of the different food elements to the indications given by human milk. It is lacking in the antiscorbutic element, so that some antiscorbutic must be given with it.

Proprietary Foods.—It is impossible for the proprietary foods to have anything in them which will nourish a baby except the food elements; namely, fat, carbohydrates, proteins and mineral matters. These

elements are all easy to procure and can be put into modified milk in any form and in any amount desired.

There are many objections to the use of the proprietary foods. In the first place, they are expensive, because it is evident that not only the manufacturer, but every one who handles them, must make a profit. This additional expense is entirely unnecessary, because modifications of milk can be easily prepared in the home from simple materials which contain everything which is in the proprietary foods. Another objection to the proprietary foods is that their use tends to develop slip-shod methods on the part of physicians. Instead of thinking what an individual baby ought to have, they simply advise the use of the food which they happen to be using at the time or choose some food at random. Still another objection is that parents, being misled by the advertisements concerning these foods, think that feeding babies is a simple proposition and attempt it themselves. Another objection is that when proprietary foods are used, it is impossible to modify them to fit the needs of the individual infant. It not infrequently happens that a baby does better on some proprietary food than it did on modified milk. This does not prove, however, that this proprietary food is better than modified milk. It merely shows that the combination of the different food elements in this food was the one suitable for the individual baby. There was nothing in the proprietary food which could not have equally well been put in a modified milk. The difficulty was not with the modified milk, but with the person who prescribed it, who either did not understand or did not know how to meet the indications in the given case. If he had, the baby would have thrived as well, if not better, on modified milk as on the proprietary food. It is noteworthy in this connection that while much is said in praise of a given food, when a baby does well on it, nothing is said about all the other proprietary foods upon which it did not do well. Nothing is said, moreover, about the great number of babies that thrive on modified milk after they have done badly for a long time on a great variety of proprietary foods. The manufacturers of proprietary foods never give pictures in their advertisements of the babies which have not done well on their foods or have died while taking them. The sales of their food would not be as large as they are at present, if they did.

The proprietary foods can be divided into four main groups. (1) The condensed milks. (2) The malted foods, in which the whole or a considerable part of the carbohydrates is in the form of maltose and the various dextrins. (3) The sugary and starchy foods, in which there is a considerable proportion of starch in addition to the soluble carbohydrates. (4) Starchy foods, which are composed almost entirely of starch.

The Condensed Milks.—Condensed milk is, of course, simply milk from which a considerable proportion of the water has been removed. In most instances some of the cream has been removed before the milk was condensed. When the condensed milk is diluted with water again, the composition is, of course, that of milk or of wholly or partially skimmed milk. When sugar has been added, as it is in many brands, the percentage of sugar when the milk is diluted is, of course, higher than in milk. The kind of sugar in the dilution depends entirely on the kind of sugar which has been added.

Condensed milk is, of course, almost never given undiluted. The usual dilution is one part of condensed milk to nine parts of water. When, for example, Eagle Brand Condensed Milk is used in this dilution, the mixture contains 0.96% of fat, 5.49% of sugar, and 0.80% of protein.

When an unsweetened condensed milk, like the St. Charles Brand, is used in the same dilution, the mixture contains 0.87% of fat, 1.09% of sugar and 0.88% of protein. It is evident from these analyses why a baby that has a disturbance of digestion from overfeeding does well on these mixtures. It would do equally well, however, on a modified fresh milk containing the same percentages. It is also evident from these analyses that a baby must take a very large amount of this food to cover its caloric and protein needs. Furthermore, the relations between the different food elements do not resemble, even remotely, those in human milk. Condensed milk is, furthermore, not the uniformly sterile product that it is commonly supposed to be. Some specimens are sterile, but many are not. The bacterial content of some of them is very high. It grows steadily higher after the can is opened.

The Evaporated Milks.—The evaporated milks are simply milks from which a smaller proportion of water has been removed than in the case of the condensed milks. No sugar is added to them. When they are diluted with water, the composition is, of course, that of whole milk or of some dilution of whole milk.

The Malted Foods.—Some of the malted foods are made up entirely of maltose and the dextrins and contain no proteins except those vegetable proteins which are derived from the grains used in the preparation of the sugars. Examples of such foods are Mellin's Food and Mead's Dextri-Maltose. Foods of this type are intended to be used with milk. These foods are really not foods, but simply special preparations of sugar. When they are used with milk, the resulting mixtures are merely modifications of milk with a large part of the sugar in the form of the dextrins and maltose. Babies that for any reason need their sugar in this form do well on them, therefore, and better than they would on mixtures prepared with milk sugar. It is also evident that the foods of this type which contain a large proportion of maltose are more laxative than those which contain a large proportion of the dextrins.

A number of the other foods in this class contain milk as well as malt sugar and the dextrins. They, therefore, contain, in addition to malt sugar, the dextrins and vegetable protein, a certain amount of fat, milk sugar and animal protein derived from the milk. Samples of foods of this sort are Horlick's Malted Milk, Laibose and Allenbury's Foods, #1 and #2. These foods may be used either with or without milk. In most instances they are intended to be used without milk. When prepared with water, they are markedly deficient both in fat and protein. When used in this way, the relations of the percentages to each other cannot, of course, be changed and, in consequence, the food cannot be fitted to the needs of the infant. When used with milk, the composition of the mixture is changed, of course, according to the amount of milk or cream added.

The only rational use for foods of this class is to provide sugar in the form of maltose and the dextrins instead of in the form of milk sugar or cane sugar. They ought not be used in a routine way or according to the directions which come with them. A milk mixture should be prepared to fit the needs of the individual infant and then as much of these sugars added as is required to make up the desired percentage of carbohydrate. The same results can be obtained by adding a cereal gruel and then dextrinizing the food.

The Sugary and Starchy Foods.—The most common samples of this class of foods are Eskay's Albuminized Food, Nestlé's Food,

Table XIX.—The Composition of Some of the Proprietary Foods

Food	Percentages					Source of analysis
	Fat	Sugar	Protein	Starch	Ash	
Condensed Milk—Eagle Brand	9.61	54.94 (Cane 42.91; Milk 12.03)	8.01	.....	1.78	Own advertisement.
Condensed Milk—St. Charles	8.70	10.95 Milk	8.80	.....	1.40	Jordan & Mott. Am. Jour. Public Hygiene, 1910, xx, 391.
Ramogen	16.50	34.65 (Cane 20.91; Milk 13.74)	7.00	.....	1.50	Own advertisement.
Mammala	12.12	55.34 Milk	24.35	.....	4.93	Mellin's Food Co.
Horlick's Malted Milk	8.78	67.95 (Milk 10.65; Malt 38.50; Dextrins 18.80)	16.35	.....	3.86	Dennett. Simplified Infant Feeding. 1915, p. 338.
Mellin's Food	0.16	79.57 (Malt 58.88; Dextrins 20.69)	10.35	.....	4.30	Own advertisement.
Mead's Dextri-maltose, Nos. 1–2–3	.....	92–95 (Malt 51–52; Dextrins 41–43)	.....	.....	[1] 3.00	Own analyses.
Justfood	.....	93.95 (Malt 24.25; Dextrins 69.70)	1.10	.....	0.32	Own advertisement.
Laibose	17.00	55.00 (Milk 25.00; Malt 25.00; Dextrins 5.00)	18.00	.....	4.00	Own analysis.
Allenbury's Foods, No. 1, Milk	18.60	66.55 (Milk 42.00; Malt 14.00; Dextrins 10.00)	10.66	.....	3.95	Own advertisements, except proportions of different sugars. These given approximately from analyses made by Mellin's Food Co.
No. 2, Milk	15.88	70.90 (Milk 36.00; Malt 20.00; Dextrins 13.00)	9.90	.....	3.71	
No. 3, Malted	1.05	25.11 (Malt 16.50; Dextrins 8.50)	10.23	60.01	0.60	
Dennos Food	1.17	16.64 (Cane 13.32; Dextrins etc. 3.32)	11.10	57.00 (4.27 Cellulose)	0.79	Mellin's Food Co.
Nestlé's Food	5.50	58.93 (Milk 6.57; Cane 25.00; Malt Dextrins ..... 27.36)	14.34	15.39	2.03	Own advertisement.
Eskay's Albuminized Food	3.52	55.82 (Milk 54.12; Dextrins 1.70)	6.70	29.90	0.99	Own analysis.
Ridge's Food	0.26	7.80 Milk, largely	12.50	73.67	0.61	Own analysis.[2]
Benger's Food	0.92	3.34 Dextrins and Sugars	12.12	77.02	0.97	Own analysis.[3]
Imperial Granum	1.04	1.80 (Dextrose 0.42; Dextrins 1.38)	14.00	73.54	0.39	Holt. Diseases of Infancy and Childhood, 1911, p. 162.[4]

[1] No. 1, Sodium chloride, 2%; No. 3, Potassium bicarbonate, 3%.
[2] Proprietors wish to emphasize the fact that the directions call for boiling, which "gelatinizes the starch in the form of colloids."
[3] Proprietors wish to call attention to the fact that the food contains active amylolytic and tryptic ferments, which, when the food is prepared according to the directions, "convert the starch into soluble sugars and modify the casein."
[4] Proprietors wish to direct attention to the fact that Imperial Granum is intended to be used with milk.

Allenbury's Food #3, and Dennos Food. The fat content of the foods of this class is, as a rule, almost infinitesimal. When diluted with water, they amount to little more than a starch and sugar mixture. When mixed with some form of milk, they correspond to a modified milk with a cereal diluent. The kind of sugar in the mixture varies, of course, with the kind of sugar in the food. Eskay's food contains a small amount of egg albumin. It is difficult to see of what special use this can be to a baby, even if it does serve as an excuse for the name of the food.

The Starchy Foods.—The most common examples of the starchy foods are Imperial Granum and Ridge's Food. These foods are really starch foods rather than starchy foods. The analysis of Imperial Granum and ordinary wheat flour shows that the composition of the two is the same, except that there is 1.80% of dextrose and the dextrins in Imperial Granum and none in wheat flour. It is well known, however, that, when wheat flour is heated, a small percentage of the starch is changed into dextrins and dextrose. It is hard to see, therefore, what advantage there is to the consumer in Imperial Granum over wheat flour baked in the oven. It would seem cheaper for people to bake their own flour.

The various barley flours deserve the name of "Foods" as much as do many of those that claim it. All of them serve simply as a source of starch. When prepared with water, without milk, they amount, of course, simply to a solution of starch and are entirely unsuitable as a food for a baby except temporarily during disturbances of digestion. When they are used with milk, unless in considerable amounts, they add, as already explained, but little to the nutritive value of the food. The starch which they contain acts, however, as a protective colloid and, therefore, makes the digestion of the casein easier.

FEEDING DURING THE LATTER PART OF THE FIRST YEAR AND IN THE SECOND YEAR

Breast-fed babies have usually been weaned by the time they are nine or ten months old and are taking whole cow's milk with or without the addition of one of the cereal waters. The method of feeding them during the later months of the first year is, therefore, the same as when babies have been artificially-fed. When a baby is nursed until the end of the first year, it is usually advisable not to give it anything in addition, except cow's milk, perhaps with a cereal diluent, before it is eleven months old.

Orange Juice.—When a baby is artificially-fed, it is advisable, if the milk is pasteurized or boiled continuously, to begin to give it orange juice when it is about three months old in order to prevent the development of scurvy. It is customary to begin to give orange juice, even if the milk is given raw, when babies are about six months old. This is unnecessary, however, because scurvy almost never develops if the milk is given raw. On the other hand, it does no harm and should be given, if the baby is constipated. One tablespoonful of orange juice daily is usually and two tablespoonfuls always sufficient to prevent the development of scurvy. It is unnecessary and usually inadvisable to give more. It should all be given at one time. It may be given either plain or diluted with water. There is no objection to adding a little granulated sugar to it, if it is very sour. It is usually given an hour before some feeding, when the stomach is approximately empty, in order not to interfere with the digestion of the milk. It is probable, however, in

the light of recent experiences with acidified milk, that it not only does no harm, but may possible be of some advantage to give it at the beginning of a meal.

Cereals.—When babies are artificially fed, it is advisable, if they are digesting their milk well, to give one of the simple cereals when they are about nine months old. It may sometimes be given earlier. It should be given at the beginning of two feedings daily, at 9 A.M. and 6 P.M., if the baby is on three hour intervals, and at 10 A.M. and 6 P.M., if it is on four hour intervals. The most easily digestible cereals are strained oatmeal, barley jelly and farina. A level tablespoonful is enough at first. This may be gradually increased to two rounded tablespoonfuls by the end of the year. The cereal should have some of the baby's mixture on it, with a little salt, but no sugar. It should be given with a spoon, not mixed with the food in the bottle. Cream of wheat may be added to the list of cereals after one or two months. All cereals which are given to infants and children must be thoroughly cooked. Even the simplest ones should be cooked for several hours, no matter what the directions on the packages say.

Beef Juice and Broths.—A few weeks after beginning the cereals, it is advisable to give beef juice or mutton or chicken broth at the beginning of the 12 M. feeding, if the baby is on three hour time, and at the beginning of the 2 P.M. feeding, if it is on four hour time. The beef juice obtained by half broiling steak, cutting it up into small pieces and squeezing out the juice should be used. Beef juice prepared in this way contains about 0.60% of fat and 2.90% of protein with a considerable amount of extractive matters. Dish gravy is not the same as beef juice. It contains a considerable amount of cooked fat and is quite indigestible, unless cooled and skimmed. The various beef extracts and liquid preparations of beef juice on the market should never be used, as they contain but little nourishment, while the liquid preparations contain an appreciable amount of alcohol. One teaspoonful of beef juice is sufficient in the beginning. This may be increased to two tablespoonfuls, or one ounce, if it does not disagree with the baby. It may be given plain or diluted with water and should be salted to taste. No more than two ounces should ever be given to babies, even in their second year. It is liable to disturb the digestion and not infrequently makes babies nervous and sleepless. Two ounces of mutton or chicken broth is enough to give at first. This may be increased to four ounces. The nutritive value of broths, however, is very slight. It is unwise, therefore, to fill a baby up with broth and in that way prevent it from taking its milk. The chief value of broth is as a vehicle for other foods. There is no objection to cooking vegetables in the broth and then straining them out.

Zwiebach, Bread Crumbs, Rice and Macaroni.—When a baby is ten or eleven months old, zwiebach and bread crumbs may be added to the beef juice and broths, and, a little later, boiled rice and plain boiled macaroni, which has been put through a ricer.

Bread and Crackers.—If the baby has a sufficient number of teeth, it may also be given zwiebach, thoroughly stale bread or plain white crackers "in its hand" to eat after one or two feedings daily. They should not be given between meals. The best crackers are Uneeda biscuits, other similar crackers, and pilot wafers. Graham crackers should not be given, as they are too sweet and are not easily digested.

Junket, Baked Apple, Apple Sauce and Prune Juice.—There is no objection to giving the whole or part of the milk at the mid-day feeding

in the form of junket. If the baby is constipated, a little baked apple, apple sauce or prune juice may be given with the mid-day meal. Baked apple is, for some reason, more easily digested than apple sauce.

At some time during the first half of the second year, it is advisable to change from the regular intervals used in the past to the irregular intervals usual for older children. Then, if a baby wakes early, it gets a drink of milk on waking and has its breakfast in the neighborhood of 8 o'clock. If it wakes late, it has nothing before its breakfast. If the bath is given in the morning, the breakfast may be given a little later. In such cases, however, milk must be given on waking. The baby has some milk or milk with a piece of bread or cracker between 11 and 11:30, just before its nap. It has its dinner between 1 and 2, according to when it wakes up, and has its supper between 5 and 5:30, being tucked in for the night by 6, at the latest. During the first half of the second year the breakfast should consist of milk, cereal and bread, toast, zwiebach or cracker. The orange juice is usually given about one hour before the lunch. The dinner consists of broth or beef juice, with bread, zwiebach, rice or macaroni. It may have milk or a plain dessert, such as junket, plain blanc-mange, corn starch pudding, prune juice, baked apple, or apple sauce. Its supper is the same as breakfast, except that, if it is constipated and needs prune juice, baked apple or apple sauce, they may be given with the supper instead of with the dinner. The bread may be given in the form of milk toast, that is, toasted bread soaked in hot milk without thickening.

Potato and Eggs.—When a baby is a year and a half old, baked potato and eggs, boiled for two minutes or coddled for four minutes, may be added to the diet. They should be given at dinner. If the egg does not disturb it, it may have baked custard as another dessert, but never on the same day that it has the boiled or coddled egg. It is not advisable to give eggs more than three times a week before a baby is two years old. Butter may be given at about the same time. Further additions to the desserts are plain tapioca and apple tapioca. It should go without saying that they are to be served without sugar or cream, either plain or whipped. Wheatena, Wheat Germ, Germea and Ralston may be added to the list of cereals.

Green Vegetables.—I do not believe that green vegetables should be given to children before they are two years old, unless there is some especial reason for it. It is true that they sometimes help constipation, but constipation can be relieved in babies more safely in other ways. It is often said that they are necessary in order to give a proper amount of iron and of other salts to babies. It is very striking, however, that most of those who recommend them most highly on this account fail to state how much iron babies need and also how much iron there is in green vegetables and other foods. The data as to the real need of babies for iron and the amount of iron contained in the various foods are very incomplete. It is probably not far from the truth, however, to say that the normal baby under six months of age does not need more than 0.5 mg. of iron, calculated as Fe., daily, not more than 1.5 mg. daily during the rest of the first year and 2.0 mg. during the second year. The need is probably less rather than more. The iron content of the vegetables is not as great as would seem from their color, because the color of green vegetables is due to chlorophyll and that of carrots to carotin, not to any form of iron. A level tablespoonful of cooked, strained spinach contains 0.59 mg. of iron, a level tablespoonful of string beans 0.3 mg. and

a level tablespoonful of carrots 0.15 mg. It is evident, therefore, that a baby who gets one teaspoonful of carrots or string beans really gets very little iron. An ounce of beef juice contains 0.2 mg. of iron and three tablespoonfuls of oatmeal contains 0.3 mg., while a level tablespoonful of prune pulp contains 0.7 mg. of iron and the yolk of an egg 1.4 mg. It is evident, therefore, not only that it is possible to give iron to babies in other ways than in the green vegetables, but that they get a considerable amount in their cereals and beef juice and that it is far easier to give them iron in prune pulp and yolk of egg than in any other way. A quart of milk contains a far greater amount of the salts, except iron, than any baby needs. It is evident, therefore, that babies taking the simple diet detailed above will not suffer from the lack of iron or other salts, because it does not contain green vegetables.

It is also sometimes said that babies will suffer from a lack of vitamins, if they are not given green vegetables. This is not true, because there is an abundant supply of both the fat soluble A and the water soluble B in a quart of milk, and of the antiscorbutic vitamin in orange juice, without taking into consideration the vitamins present in the other foods.

TABLE XX

DIET AT EIGHTEEN MONTHS

Milk	Zwieback	Plain macaroni
Butter	Plain crackers	Baked potato
Mutton broth	Barley jelly	Junket
Chicken broth	Strained oatmeal	Baked custard
Beef juice	Cream of wheat	Cornstarch pudding
Soft-boiled eggs	Wheat germ	Plain blanc mange
Coddled eggs	Ralston	Orange juice
Stale bread	Farina	Prune juice and pulp
Milk-toast	Rice	Baked apples

6:00 A. M.	Milk, 8 ounces.
8:00 A. M.	Cereal.
Breakfast:	Bread, zwieback, or cracker. Milk, 8 ounces.
10:00 A. M.	Orange juice.
11:00–11.30 A. M.	Bread, zwieback, or cracker.
Lunch:	Milk, 8 ounces.
1:00–2.00 P. M.	Broth, beef juice, or egg.
Dinner:	Rice, baked potato, macaroni, bread, or zwieback. Junket, baked custard, cornstarch, blanc mange, prune juice, or baked apple. Milk, 4 to 8 ounces, if desired.
5:00–5.30 P. M.	Cereal.
Supper:	Bread, zwieback, crackers, or milk-toast. Milk, 8 ounces. Prune juice or baked apple, if constipated, and not had at dinner

FEEDING AFTER TWO YEARS

The milk in the early morning should be continued or be replaced by a piece of bread and butter or a cracker, if the child continues to wake up early and does not have its breakfast until late. If it wakes up late and has its breakfast early, it should be stopped. The lunch before the nap

should be continued as long as the child has its nap, that is, until it is prevented from having its nap, or at least a rest, by going to school. If it has its dinner at twelve and its rest afterward, it needs nothing between breakfast and dinner, but will then need something in the middle of the afternoon. It should have its supper between five and six and, even when it is five or six years old, should be in bed by half past six, or, at the latest, seven o'clock. It should have nothing whatever between meals.

Meat.—At two years the simple meats, like the white meat of roast or boiled chicken, lamb or mutton chop and scraped beef, may be given. Meats, contrary to the general impression, are easily digested by young children and almost never cause trouble. The red meats contain no more extractives and are no more irritating to the kidneys than are the white meats and fish.

A fundamental principle in feeding is that foods that are cooked over are very much less digestible than foods which have been cooked but once. Cold meat is much more digestible than meat which has been made into hash or croquettes and recooked. There is no reason why children should not have cold meat. Minced meat, that is, chopped meat, heated with water or water with a little thickening, is quite easily digested. Children should not be given gravy, because of the cooked fat which it contains. For the same reason, they should not be given dish gravy, unless it is cooled and the fat removed.

Bacon.—I do not approve of giving bacon to babies. It is generally admitted that fried fat is very indigestible not only for babies, but also for children and adults. I fail to see why fried pig's fat, of which bacon is largely composed, should be any more digestible than any other form of fried fat. No one thinks of giving roast pork or fried or boiled ham to babies and young children and yet these forms of pig meat should be more easily digested, because of the way they are cooked, than is bacon. There is, furthermore, no reason to believe that the meat of the pig is any better suited for babies and young children than that of sheep or kine. I, therefore, do not give bacon to babies and young children.

Green Vegetables.—The green vegetables may be given at two and one quarter or two and one half years. The most easily digestible ones are spinach, string beans, peas, asparagus, and cooked celery. Peas should always be mashed in the beginning and, preferably, put through a sieve. Spinach should always be put through a sieve and string beans usually. It is not necessary to strain asparagus and celery. There is no objection to the use of canned peas, string beans and spinach. Canned asparagus is usually somewhat indigestible.

TABLE XXI

DIET AT TWO YEARS

Milk	Stale bread	Baked potato
Butter	Toast bread	Plain macaroni
Mutton broth	Milk-toast	Junket
Chicken broth	Zwieback	Baked custard
Beef juice	Plain crackers	Cornstarch pudding
Soft-boiled eggs	Barley jelly	Plain blanc-mange
Coddled eggs	Strained oatmeal	Orange juice
Lamb chop	Cream of wheat	Prune juice and pulp
Mutton chop	Wheat germ	Baked apple
Roast chicken	Ralston	Apple sauce
Boiled chicken	Farina	
Scraped beef	Rice	

6:00 A. M.	Milk, 8 ounces.
8:00 A. M.	Cereal.
Breakfast:	Bread, toast, zwieback, or cracker. Milk, 8 ounces.
10:00 A. M.	Orange juice.
11:00–11:30 A. M.	Milk, 8 ounces.
Lunch:	Bread, toast, zwieback, or cracker.
1:00–2:00 P. M.	Broth, broth and meat, beef juice, meat or egg.
Dinner:	Rice, baked potato, macaroni, bread, zwieback, or cracker. Junket, baked custard, cornstarch, blanc-mange, prune juice or pulp, baked apple, or apple sauce. Milk, 4 to 8 ounces, if desired.
5:00–5:30 P. M.	Cereal.
Supper:	Bread, toast, milk-toast, zwieback, or cracker. Prune juice or pulp, baked apple or apple sauce, if constipated, and did not have at dinner. Milk, 8 ounces.

Meat and Fish.—During the third year other simple meats, such as roast lamb, roast or boiled mutton, roast beef and beef steak may be added to the diet. Boiled and broiled fish may also be given. The most digestible fish for young children in the Eastern states are cod, halibut, schrod and haddock. The oily and rich fishes, such as swordfish and salmon, should not be given to them. Fish should never be fried.

Cereals.—The list of cereals may be increased by the addition of Pettijohn's Breakfast Food, hominy, cracked wheat, and shredded wheat biscuit. Shredded wheat biscuit is the only one of the cereals which are not cooked in the home that I consider suitable for young children. It is true that children like the cereals which are not cooked in the home, especially some of the sweet ones. It is also true that they are easier to serve, since they require no preparation. Nevertheless, I do not believe that they are as easily digested as the cereals which are cooked in the home. I especially object to the sweet ones, because the sweetness is due to the fact that the starch has been largely changed into sugar.

Vegetables.—Potatoes may be given mashed, boiled, and stewed or creamed, as well as baked. Carrots may be given, but at first should be mashed. Both summer and winter squash may be given; also plain boiled cabbage and cauliflower. Cabbage and cauliflower are very easily digested, if they are not served with cream sauce. Cabbage should never be given raw. I do not approve of tomatoes, beets and corn for children. Corn, even when green and when the kernels are cut, often causes severe or fatal indigestion in children. Beets are sweet and tough, even when young, and difficult to digest. Tomatoes are strongly acid and when given raw, or even cooked, are very likely to disturb the digestion of children, as well as that of many adults. It is true that the juice of cooked tomatoes is an antiscorbutic and that it has been used successfully both for the prevention and cure of scurvy in young babies when orange juice, for some reason, could not be obtained or was too expensive. It is apparently believed that, because some babies have been cured of scurvy by tomato juice, tomato juice is a necessary, or at any rate, an advisable, article of diet for all babies and children. This belief is wrong.

Fruits.—Pears and peaches may be given cooked at three or four years. In general, it is not advisable to give them uncooked before a child is five years old. Peaches are usually more easily digested than pears. The pulp of the orange may be given at four years. Grape fruit

TABLE XXII

DIET AT TWO AND ONE HALF YEARS

Milk	Milk-toast	String beans
Butter	Zwieback	Spinach
Mutton broth	Plain crackers	Asparagus
Chicken broth	Plain educators	Cooked celery
Beef juice	Barley jelly	Orange juice
Soft-boiled eggs	Strained oatmeal	Prune juice and pulp
Coddled eggs	Cream of Wheat	Baked apple
Dropped eggs	Wheat germ	Apple sauce
Lamb chop	Wheatena	Junket
Mutton chop	Germea	Baked custard
Roast chicken	Ralston	Cornstarch
Boiled chicken	Farina	Bread pudding
Scraped beef	Rice	Rice pudding
Stale bread	Baked potato	Plain blanc mange
Toast bread	Plain macaroni	Plain tapioca
Whole wheat bread	Peas	Apple tapioca

6:00 A. M.	Milk, 8 ounces.
8:00 A. M.	Cereal.
Breakfast:	Bread, toast, zwieback, or cracker; occasionally an egg. Milk, 8 ounces.
10:00 A. M.	Orange juice.
11:00–11.30 A. M.	Milk, 8 ounces.
Lunch:	Bread, toast, zwieback, or cracker.
1:00–2.00 P. M.	Broth, broth and meat, beef juice, meat, or egg.
Dinner:	Rice, baked potato, macaroni, bread, zwieback, or cracker. Peas, string beans, spinach, asparagus, or cooked celery. Junket, baked custard, cornstarch, blanc mange, bread pudding, rice pudding, plain tapioca, apple tapioca, prune juice and pulp, baked apple, or apple sauce. Milk, 4 to 8 ounces, if desired.
5:00–5:30 P. M.	Cereal.
Supper:	Bread, toast, milk-toast, zwieback, cracker toast, or cracker milk-toast. Prune juice or pulp, baked apple, or apple sauce, if desired. Milk, 8 ounces.

is not suitable for young children. It is very acid and the pulp is coarse and indigestible. Some children can digest bananas without difficulty from the time they are three or four years old. Other children always have trouble from them. It is advisable, therefore, to find out whether the individual child can digest them or not. If it can, bananas are a valuable form of food. They are more digestible when thoroughly ripe and when baked. I do not believe that children should eat raw apples before they are six years old. They are difficult to digest for many children as well as adults, even when they are scraped.

Berries.—Uncooked berries should not be given to children until they are more than six years old. Cooked strawberries and blueberries may, however, be given cautiously after children are three years old. Melons and nuts are not suitable foods for young children.

Desserts.—The list of desserts may be increased during the third year by the addition of prune whip, the simple gelatines, and bread and rice puddings, made, of course, without raisins. These desserts should be served without additional sugar, cream, either plain or whipped, or other sauces. Soft custard is much less easily digested by most children than is baked custard. Chocolate should never be used in the preparation of children's desserts.

TABLE XXIII

DIET AT FOUR YEARS

Milk	Milk-toast	Winter squash
Butter	Oatmeal	Cooked celery
Mutton broth	Pettijohn	Carrots
Chicken broth	Cream of Wheat	Lettuce
Bouillon	Wheat germ	Cabbage
Vegetable soup	Wheatena	Cauliflower
Milk soups	Germea	Stewed prunes
Purées of peas and beans	Ralston	Baked apples
	Farina	Apple sauce
Soft-boiled eggs	Hominy	Cooked pears
Dropped eggs	Samp	Cooked peaches
Scrambled eggs	Rice	Cooked apricots
Lamp chop	Cracked wheat	Oranges
Mutton chop	Shredded Wheat Biscuit	Bananas
Beef steak		Cooked strawberries
Roast lamb	Baked potato	Cooked blueberries
Roast mutton	Boiled potato	Junket
Boiled mutton	Mashed potato	Baked custard
Roast chicken	Stewed potato	Cornstarch
Boiled chicken	Plain macaroni	Bread pudding
Broiled chicken	Plain spaghetti	Rice pudding
Minced meat	Peas	Plain tapioca
Turkey	String beans	Apple tapioca
Boiled fish	Spinach	Plain blanc mange
Broiled fish	Beet greens	Prune whip
White bread	Swiss chard	Gelatins
French bread	Asparagus	Plain cookies
Whole wheat bread	Lima beans	Sponge cake[1]
Plain crackers	Summer squash	Vanilla ice-cream[1]

On waking, if it is early, bread or cracker; orange juice, if desired.

7:00–8:00 A. M.	Cereal.
Breakfast:	Bread.
	Meat, fish, or egg.
	Milk.
11:00 A. M.	Milk.
Lunch:	Bread or cracker.
1:00–2:00 P. M.	Soup—not necessary.
Dinner:	Meat, fish, or egg. Do not give egg if had egg at breakfast.
	Potato, rice, macaroni, or bread.
	Green vegetable.
	Dessert or fruit.
	Milk, if desired.
5:30–6.00 P. M.	Cereal.
Supper:	Bread or cracker in some form.
	Cooked fruit.
	Milk.

If nap is taken after dinner, dinner should be at 12.00 M. and lunch at 4.00 P. M.

[1] Not oftener than once a week.

Cookies, Cake and Ice Cream.—Plain cookies may be given as a dessert or with supper after children are four years old. They should never be given between meals. The only form of cake allowable for children between four and six years of age is sponge cake. I am sure, however, that they would be better off without this, as they would likewise be much better off without ice cream.

Milk.—Children should continue to drink a quart of milk daily until they are at least six years old, and preferably longer. It is inadvisable to add cocoa or chocolate to it. They add little to the nutritive value of the milk and do disturb the digestion of many children. Children will take milk without any additions without question, if they are brought up properly and it is not suggested to them that something should be added to their milk. Cream, while nutritious, is not a suitable article of diet for children. It is almost certain to disturb their digestion, if given continuously. No young child should be given tea or coffee. Milk soups are allowable, but stews and chowders are quite indigestible. So is oyster stew, because of the heated butter in it.

Eggs.—Children should never be given fried eggs, and omelettes, even when plain, are much less digestible than are eggs which are boiled, coddled, dropped or scrambled. Few children can take eggs more than once daily without getting into trouble. Many of them cannot take them more than three times a week. It is better to give eggs at breakfast than at supper.

Bread and Crackers.—It is very important to have bread thoroughly cooked, because, if it is not, it is extremely indigestible. Whole wheat bread has but very little, if any, more nutritive value than bread made from white flour. It does contain a larger amount of water-soluble B vitamin, and if a child ate nothing but bread it would be far better for it, on this account, to eat whole wheat bread. The child who takes milk and a reasonable, general diet, however, will get far more of the vitamin B in them than it needs. Graham bread and rye bread, unless light and thoroughly cooked, are quite indigestible. Properly cooked Graham bread is seldom seen. Bread should be at least forty-eight hours old before it is given to children. They should not be given, under any conditions, hot bread, biscuits, or griddle cakes. Bread which is dried in the oven or thoroughly toasted through is very easily digested. Bread which is just browned or burned on the outside, the center being damp and sticky, is extremely indigestible. Unfortunately, this is the kind of toasted bread which most people are in the habit of eating. Buttered toast is indigestible because it is soggy and the fat in the butter has been heated. Toast should not be buttered until it is eaten. Brown bread is always heavy, soggy, and sweet, and is entirely unfit for consumption by children.

Many people have an idea that crackers are not nutritious. This idea is erroneous because crackers are composed almost entirely of starch. Bulk for bulk and weight for weight, crackers are far more nutritious than bread, because bread contains so much water and is so porous, while crackers contain practically no water and are much less porous. The best crackers for children are the plain white crackers, like Uneeda Biscuit, Pilot Wafers, and Pilot Biscuit, soda crackers, and the old-fashioned Boston crackers. They should not be given the sweetened, flavored, and fancy crackers. Graham crackers are entirely unsuitable. They are much too sweet and not easily digested. Oatmeal crackers should not be given, as the starch in the oatmeal is not thoroughly cooked.

Bran crackers may be given in the treatment of constipation, but it must be remembered that the bran has no nutritive value.

Sugar.—There is no article of food which causes more disturbances of digestion in childhood than sugar. As money is said to be the root of all evil, so sugar may be said to be the root of all the disturbances of digestion in childhood, neither statement being, of course, strictly true. Further than this, sugar is a very common cause of loss of appetite in children, and destroys their appreciation of proper food. It also, more than any other one thing, is responsible for the decay of children's teeth. Candy, therefore, should never be given to children. It can do them no good and may do them much harm. It is idle, of course, to claim that two or three pieces of candy a day will disturb the average child's digestion or prevent its normal development. Children that have two or three peices, however, usually want more, and are quite likely to get more. It is true that some kinds of candy are richer and more indigestible than others, but they are all made of sugar, and plain sugar is bad for children. Children should be brought up not to eat sugar on anything. There is no objection to putting a little sugar in the food during its preparation, but no sugar should be put on it when it is served. Sugar is, of course, not a necessary article of diet for children. The starches are changed to sugar before they are absorbed and utilized, and, therefore, can supply the requisite amount of carbohydrates. They are much less likely to disturb the digestion than is sugar, because the sugar formed from starch is quickly absorbed, while, when large quantities of sugar are taken, there is an opportunity for excessive fermentation and indigestion.

Ice Cream, Ice Cream Soda and Sweet Drinks.—These are always inadvisable for and usually harmful to children. They are harmful chiefly because of the sugar which they contain, but partly because they are too cold, partly because they are too rich, and partly because they are usually taken between meals. Children would be better off without any of them. Ice cream is probably less harmful than the others. Vanilla ice cream is not as rich as other kinds. The majority of people are so willing to take the chance of injuring their children's health in order to give them temporary pleasure that I have found it useless to attempt to cut ice cream entirely out of the diet of children. I, therefore, compromise, and allow children to have plain vanilla ice cream without any sauce on it once a week.

Water.—Children need a great deal of water. They are very active and consequently lose much water in the perspiration and in the expired air. They, like adults, need more water in hot weather than in cold, and in dry weather than in moist. Strange as it may seem, many young children are so interested in their play that they do not realize that they are thirsty, and have to be reminded that they need a drink. Children should be watched to see that they get enough water in the twenty-four hours. In general, it is wiser for them to take their water between meals. There is, however, no objection to the drinking of water with the meals, provided it is not used to wash down imperfectly chewed food. It is, indeed, a good plan to finish a meal with a drink of water. A child cannot drink too much water; it may drink too little. It is not necessary to boil the water, provided it is pure, after the first year.

Light Suppers.—Children should not have meat or vegetables for supper until they are at least six years old. Broths and eggs should be given only occasionally. Milk, cereal, bread or crackers in some form, and cooked fruit are amply sufficient for them.

TABLE XXIV

DIET AT SIX YEARS

Milk	Plain crackers	Shell beans
Butter	Milk-toast	Summer squash
Mutton broth	Oatmeal	Winter squash
Chicken broth	Pettijohn	Cooked celery
Bouillon	Cream of Wheat	Carrots
Vegetable soup	Wheat germ	Lettuce
Milk soups	Wheatena	Cauliflower
Purées of peas and beans	Germea	Cabbage
	Ralston	Stewed prunes
Soft-boiled eggs	Farina	Baked apples
Dropped eggs	Hominy	Apple sauce
Scrambled eggs	Samp	Cooked pears
Shirred eggs	Cornmeal	Cooked peaches
Plain omelette	Rice	Cooked apricots
Lamb chop	Cracked wheat	Oranges
Mutton chop	Shredded Wheat Biscuit	Grapes
Beef steak		Bananas
Roast lamb	Baked potato	Cooked strawberries
Roast mutton	Boiled potato	Cooked blueberries
Boiled mutton	Mashed potato	Junket
Roast chicken	Stewed potato	Baked custard
Boiled chicken	Plain macaroni	Cornstarch
Broiled chicken	Plain spaghetti	Bread pudding
Minced meat	Cream cheese	Rice pudding
Turkey	Cottage cheese	Plain tapioca
Boiled fish	Peas	Apple tapioca
Broiled fish	String beans	Plain blanc mange
White bread	Spinach	Prune whip
French bread	Beet greens	Gelatins
Whole wheat bread	Swiss chard	Plain cookies
Corn bread	Asparagus	Sponge cake[1]
Gems	Lima beans	Vanilla ice cream[1]

On waking, if it is early, bread or cracker; orange juice, if desired.

7:00–8:00 A. M.	Cereal.
Breakfast:	Bread.
	Meat, fish, or egg.
	Milk.
11:00 A. M.	Milk.
Lunch:	Bread or cracker.
1:00–2:00 P. M.	Soup—not necessary.
Dinner:	Meat, fish, or egg. Do not give egg, if had egg at breakfast.
	Potato, rice, macaroni, or bread.
	Green vegetable.
	Dessert or fruit.
	Milk, if desired.
5:30–6:00 P. M.	Cereal.
Supper:	Bread or cracker in some form.
	Cooked fruit.
	Milk.

The dinner hour may have to be earlier if there are two sessions of school. If there is only one session, the rest after dinner should be continued. If there are two sessions, the child should be kept as quiet as possible between the sessions.

Feeding after Six Years.—No one, whether child or adult, really needs any greater variety of food than is given in the diet list for six years. In fact, everyone would probably be better, if they never ate

[1] Not oftener than once a week.

anything not on this list. Nevertheless, almost all adults do eat a more varied and usually a far more indigestible diet than this. It is apparently impossible to convince parents of the advisibility of keeping children on a simple diet as they grow older. Nevertheless, the attempt should be made and the diet increased as slowly as circumstances permit. It is just as important to keep sweets away from children after they are six years old as before. It is also just as important for them to eat regularly and not to eat anything between meals. They should be made to eat slowly, chew their food properly and should not be allowed to wash it down with liquids. It is very important to prevent them from eating when they are excited, hot or tired. It is a very good plan for them to rest for at least fifteen minutes before each meal. It is also advisable for them to rest for a short time after eating. Furthermore, it is the duty of parents to provide a suitable diet for their children, even if it is not what they like themselves. If they can not prevent their children from eating indigestible food, when it is on the table, they should see that it is not put there. "Lead us not into temptation, but deliver us from evil."

SECTION V

DISEASES OF NUTRITION

MALNUTRITION

Malnutrition is a symptom, not a disease. It is almost always the result of some error in hygiene, care or feeding or one of the manifestations of some disease. In rare instances it is due to congenital debility. Applying some high-sounding name like dystrophy, marasmus or atrophy to it not only does not make it a disease, but, unfortunately, not infrequently does serve as a cloak for ignorance and prevents physicians from searching out and removing the cause of the malnutrition. Malnutrition is defined in the dictionary as "poor nutrition." As the term is ordinarily used in medicine, is signified that a baby is not gaining or is losing in weight or that a child does not correspond in height and weight to certain standard tables. There are, however, many other manifestations of malnutrition beside disturbances of development in height and weight. It is not necessary to mention them in detail. Some of the more marked in infancy are dryness of the skin, cold extremities, irritability and backwardness in development; in childhood, dryness of the skin and hair, cold extremities, sweating, irritability, fatigue, insomnia and backwardness at school. It is often difficult, however, to distinguish between the symptoms of malnutrition and those due to the causative condition. Great care must be taken in childhood not to call children who are simply small, malnourished. This question has been discussed in the chapter on Nutrition.

Etiology and Treatment.—The treatment of malnutrition is so dependent on the etiology that it is impossible to consider it without taking up the etiology at the same time. Malnutrition being simply a manifestation of disease or of some error in hygiene, care or feeding, it cannot be properly treated until the cause is found. Treatment consists in the removal of the cause.

In infancy the cause is usually an insufficient amount of food or some disturbance of digestion or metabolism, the latter almost always being secondary to some disturbance of the digestion. The treatment necessarily consists in increasing the amount of food or in fitting the food to the digestive capacity of the individual infant. There are no special rules to be followed, whether it is simply called malnutrition or dignified by such names as dystrophy or atrophy. It must be treated by regulation of the diet so as to give the baby a sufficient amount of a food which it can digest and utilize. This can only be determined by a careful study of the food, symptoms and stools in the individual instance. Other causes of malnutrition in infancy are, excitement, insufficient rest and sleep, lack of fresh air and sunlight, and undue exposure to cold. Less frequent causes are glandular or chronic diffuse tuberculosis. These conditions should always be suspected, when a baby that has no marked disturbance of the digestion and is getting a sufficient amount of food is not gaining and thriving. The diagnosis of these conditions, the value

of the tuberculin test in the diagnosis and the treatment are discussed in the chapter on Tuberculosis. Syphilis is very seldom the cause of malnutrition in infancy unless there are other marked symptoms of the disease. The treatment is discussed in the chapter on Hereditary Syphilis. Adenoids and chronic inflammatory processes in the upper respiratory tract are not infrequent causes of malnutrition in infancy. Chronic disease of the tonsils is seldom a cause at this age. Treatment consists, of course, in the removal of the adenoids or tonsils and local treatment of the inflammatory processes. Pyelitis is an occasional cause of disturbance of the nutrition, but the disturbance of nutrition is usually associated with other definite symptoms of the disease. Congenital debility and prematurity are also causes of malnutrition in infancy. It is important, in this connection, to remember that there may be marked disturbance of the nutrition without the development of either rickets or scurvy. The search for the cause of malnutrition in infancy should not be given up until it is found. Such a search means, of source, a careful investiga-

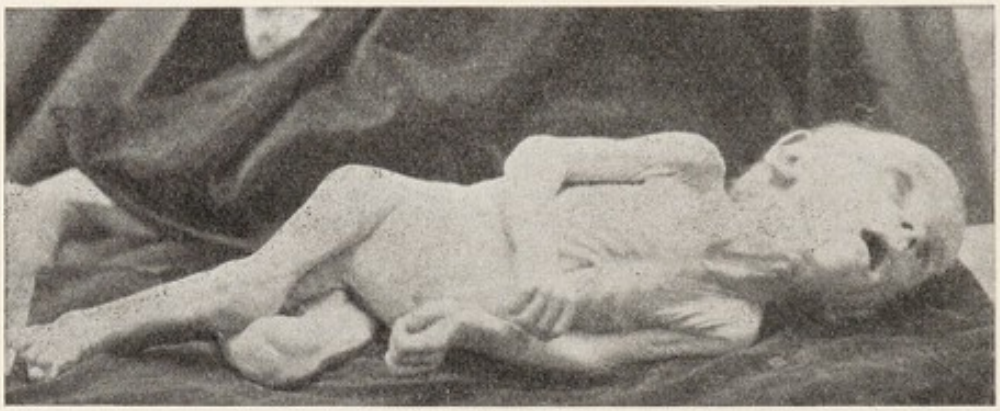

FIG. 38.—Malnutrition. Always fed on condensed milk. Never any disturbance of digestion.

tion of every detail of the life and care and a complete physical examination. When the cause is found, the treatment consists primarily in its removal.

Malnutrition in childhood may be due to a great variety of causes. The most common are the ignorance, carelessness and foolishness of parents; next, the failure of physicians to pay sufficient attention to keeping children from being sick and to explain the details of their care and feeding. This failure is, however, often not the fault of the physician, but of the parents, who, however much they are willing to lavish on their children when they are sick, are unwilling to spend a cent to keep them well. Malnutrition in children is not infrequently due to an insufficient intake of food. This, as Emerson has shown, is seldom because of poverty. It is almost always due to faulty eating habits or to an improper selection of food. Children eat hurriedly in order to get out to play, are too tired or too nervous to eat, or spoil their appetites for proper food by eating sweets. Parents are ignorant of the relative caloric values of different foods and allow their children to fill themselves up with articles which have very little nutritive value and leave out the things which are really nutritious. A diet of clear soups, tea, green vegetables and fruit may be filling, but it is not nutritious. One of milk, eggs, meat, cereals and bread may not be as attractive to some children, but its caloric value is high. The only way in which it is possible to find

out how much nourishment a child is getting is to keep an accurate account of just what and how much it eats for several days, and then calculate its caloric value. Another common cause of malnutrition in childhood is indigestion. In the vast majority of instances this is due to an excess of sweets. The symptoms of indigestion, except loss of appetite, may not be very marked. In other instances they are more marked and are easily recognized. The treatment of malnutrition due to an insufficient amount of food or to indigestion is, of course, the provision of a sufficient and suitable diet. Satisfactory results cannot be obtained, however, unless every detail of the diet is explained and careful records kept.

One of the most common causes of malnutrition in childhood is overfatigue. This may be due either to excessive physical exercise or to mental or nervous overstrain and excitement. The tendency of most young children is to play too hard. They do not realize that they are overtired and keep on until they are obliged to stop. Older children, especially boys, are likely to be forced in athletics and overtired in this way. Unfortunately, many of those in charge, or nominally in charge, of athletics in the schools are interested only in the success of the school teams and do not appreciate the limitations of growing boys. This latter criticism applies to those in charge of many of the summer camps, both for boys and girls. Not a few of the children who go to them return home in much worse shape physically than when they went. Fathers are often responsible for their children, especially their sons, being overtired. They want their children to play with them, are anxious to have them excel and do not realize that the growing child has neither the endurance nor the stamina to keep up with the adult without being injured. The strain of school work is also very great as children grow older. It, alone, is more than many of them are equal to. When extras, such as music, dancing and Hebrew, are added, it is too much for most of them. When they begin to go to parties, dances, dinners and on automobile rides, the strain is too much for almost all of them. Young children are not put to bed early enough. Their noon rests are stopped too soon. Older children are allowed to stay up to late, sometimes to study, but more often to amuse themselves. The result is that they do not get enough sleep and their nutrition suffers on this account. In general, life nowadays is too fast for children. They are not able to do their school work, all the extras which are added to it and lead the social life which many of them do, without being pulled down. Another cause of malnutrition is unhappy home surroundings. Children do not thrive in homes where there is constant quarrelling and bickering. Neither do they thrive in homes where the parents are so occupied with business and society that they do not give proper attention to their children. The treatment of overfatigue, whatever its cause, is, of course, regulation of the life to abolish the undue strain. It is often very difficult to do this, however, especially with older children, because many parents are more interested in the standing of their children at school and their social advancement than in their health and nutrition. It is necessary to go in to every detail of the life. The bedtime must be fixed and the time for rest prescribed. Unnecessary extras and social duties must be cut out. Exercise must be limited. The richer the parents, the more difficult, as a rule, this is to do. It is much harder to get them to take proper care of their children's health than it is people of more moderate means. It is a simple matter to establish nutrition clinics among the

poor. It is almost an impossibility to do anything of this sort among the rich. They have so much to do, or think they have, that they cannot, or will not, devote the necessary time to it.

Lack of fresh air and sunlight are also causes of malnutrition in childhood. Relatively, however, they are not as important as often supposed. The treatment, of course, is obvious. It may, however, require a revision of the child's schedule of life. It is also, at times, difficult to overcome the prejudice of certain people against open windows at night.

Adenoids and chronic infections of the tonsils and of the accessory sinuses of the nose and throat are common causes of malnutrition in childhood. The deformities of the mouth which they produce and the interference with the respiration resulting from them are also often causes. So also are decayed teeth. Treatment again is obvious. Adenoids and diseased tonsils should be removed, decayed teeth should be removed or treated, infections of the accessory sinuses should be treated and deformities of the mouth corrected.

Tuberculosis, usually glandular in type, is an occasional cause of malnutrition in childhood. It is a very infrequent cause, however, in comparison with improper food and habits of life. The diagnosis and treatment of tuberculosis in childhood is discussed in the chapters on Tuberculosis.

Many other diseases and chronic conditions may be the cause of malnutrition. They can only be discovered by a complete physical examination, which should be made, of course, as a routine in every case of malnutrition. It also goes without saying that every detail of the life must be investigated in every case. When the expense is not prohibitive, much can often be learned by having a competent nurse or social worker live in the household for a few days. She will often find out what the trouble is, when it cannot be found out in any other way. When the cause is discovered, it must, of course, be removed or treated. Incidentally, improper sex habits are almost never the cause of malnutrition in childhood. Congenital debility is an occasional, but rare, cause of malnutrition in childhood.

OBESITY

When the weight is 20% above the average for the height, the diagnosis of overweight, or obesity, is justifiable. Overweight in infancy, especially in the breast-fed, is, in my experience, of little importance and does not justify the fuss that is made over it by many physicians and nurses. I have never seen it do a baby any harm to be fat. If anyone thinks that it is injuring it, it is a simple matter to cut down the food a little. Overweight in early childhood is not of much importance, but, as a child grows older, it warrants more attention.

Etiology and Diagnosis.—The tendency of all students and of many physicians is to attribute obesity to some abnormality in the secretions of the ductless glands. As a matter of fat, obesity in childhood is almost never the result of such an abnormality. The only recognizable example of obesity in childhood associated with an abnormality of the secretion of the ductless glands is *Fröhlich's syndrome.* This is due to an insufficiency of both parts of the pituitary body. In this condition, the child becomes very fat, the accumulation of fat taking place in the lower abdomen, buttocks and thighs. The genital organs are infantile and the secondary sex characteristics do not develop. The skin is thin, soft and

smooth. There may or may not be an increased sugar tolerance. The appetite is usually not very large. Dieting, moreover, has very little effect on the weight. In obesity, the distribution of the extra fat tissue is general and not especially marked about the lower abdomen, buttocks and thighs. The genitals are of normal size, although, in the case of boys, the penis may appear small and sunken, if there is a large amount of fat tissue. It is true that there is sometimes a slight tendency to obesity when there is an insufficiency of the thyroid gland, but, in such instances, it is associated with failure to gain properly in height and with other symptoms characteristic of thyroid insufficiency. I have never seen *adiposis dolorosa* in childhood, although it is said that it sometimes begins at that time.

Obesity is often attributed to heredity. It is probable that there may be a certain family tendency toward overweight. The usual connection, however, is that habits of overeating and laziness are common to the family. The cause of obesity in childhood is almost invariably overeating. Overeating is also often associated with lack of sufficient exercise. The insufficient exercise, however, is usually due to the discomfort caused by the excessive weight when the child exercises. Insufficient exercise alone is almost never, in childhood, the cause of obesity. Overeating is usually due to the ingestion of an excessive amount of sweets and fats, most often of sweets, occasionally to the taking of too much of a proper diet.

Symptomatology.—Obese children are fat all over. They are usually also above the average height for their age. They look well and have a good color. They are happy and good natured. They would not be fat, if they were not. Their digestion is good. If it was not, they would not be fat. Physically they are indolent, because it makes them uncomfortable to play. They are short of breath and, because of their weight, cannot keep up with their playmates. They are further handicapped by their poor muscular development resulting from lack of exercise. Mentally, they are normal, but not infrequently lazy. On physical examination they show no more abnormalities than the normal child. In this way, they differ materially from the malnourished child, who usually shows a considerable number of abnormalities.

Treatment and Prognosis.—Treatment consists in cutting down the amount of food and increasing the exercise. It is advisable to cut down the food and diminish the weight before increasing the exercise to any great extent. Before cutting down the food, it is necessary to find out how much and what kind of food is being taken. Its caloric value must be calculated. It is usually safe to cut down the caloric value of the food one third at once. If this is not sufficient to cause loss of weight, it can gradually be diminished, one hundred calories at a time, until the desired result is obtained. It is not advisable to cut it down sufficiently to cause a loss of more than two pounds a week. It is evident that, in order to attain success, it is necessary to have the cooperation, not only of the family but also of the child. Fortunately, it is much easier to get children to follow a diet strictly than it is to get adults. It is also evident that it is absolutely necessary to keep an exact record of all the food taken and to calculate its caloric value. Furthermore, it is necessary to explain both to the parents and the child just how to estimate the amount of food and how to calculate its caloric value. Sweets must be cut out entirely and the fats much diminished. It is usually not necessary to limit the proteins a great deal. In order to prevent dissatis-

faction and to supply sufficient bulk in the food, considerable amounts of clear soups and green vegetables should be given.

Exercise should be begun cautiously. It should be mild at first and steadily, but gradually, increased. It must be remembered in this connection that the muscles are feeble from lack of use, that they have more weight to move than they should, and that the heart is not accustomed to doing much work. If the directions are followed and the treatment carried out consistently, success always results. It must be remembered, however, that it is necessary for the child to keep on a restricted diet, not only until its weight becomes normal, but afterward, in order to prevent it from increasing again. Fortunately, children soon get accustomed to eating less and after a time do not care to overeat. They also learn to like exercise, and help to keep their weight down by playing.

It is not necessary to limit the amount of water taken. Water does not make fat. It is never right to use drugs, like thyroid extract, to reduce weight.

PREMATURE INFANTS

There are very few authentic cases of the survival of infants born before the twenty-seventh or twenty-eighth week of pregnancy. Very few survive any length of time, if the weight is under two pounds or the length less than thirteen inches. It is not of much practical importance, except for medico-legal reasons, to know the exact age of a premature infant. No matter how young it is, how little its weight and length, or how poor its prospects of surviving, it should always be treated as if its chances for life were of the best.

The premature baby is not merely a small baby; it is an undeveloped baby. It is not ready to be born or to live under extrauterine conditions. The younger it is, the less developed it is and the less prepared to struggle against the abnormal conditions in which it is placed. It is intended to float in warm water of a constant temperature; it has, instead, to be handled and exposed to air of all degrees of temperature. Its circulation is compelled to change from the fetal to the adult form months before it is ready for the change. It is compelled to breathe air into lungs only partially ready for use with an undeveloped thorax and respiratory muscles. It is obliged to use digestive organs only partially completed, instead of obtaining nourishment already prepared through the circulation. In short, it is not prepared for an independent existence and has to depend for its life on organs only partially ready to perform their functions. The more these facts are appreciated, the more care and attention will be given to these infants.

In a general way, all the defects and weaknesses of the infant at term are exaggerated in the premature infant. Certain points in their development are, however, worthy of more detailed consideration. The lungs at full term are poorly enough fitted for use; they are even less so before term. They contain comparatively little alveolar structure and on account of the loose attachment of the blood vessels are very prone to congestion and inflammation. The pulse and respiration are irregular in rhythm, partly from lack of nervous control and partly because of the underdeveloped condition of the organs and muscles concerned. All the functions of digestion, although present, are feeble. That for sugar is more developed than are those for fat, starch and proteins. The function of the sweat glands is not developed at full term and, hence, is

not, of course, in premature infants. The premature infant is thus deprived of one of the most important ways of losing heat. High external temperatures are, therefore, extremely dangerous for it and may comparatively easily cause a "heat stroke."

On account of their small size, the surface area of premature infants is proportionately larger than that of full term babies. Their heat regulatory centers are, moreover, poorly developed. Furthermore, they have practically no fat tissue to conserve heat. Consequently, they lose heat very rapidly. They cannot, therefore, bear low temperatures or exposure. They must be protected in every way against cold and exposure. The importance of this protection can hardly be exaggerated. It is perfectly possible for a single slight chill to turn the scale from life to death and undo the labors of weeks or months. On account of their greater loss of heat, premature babies need relatively more food. They also need more food because of their greater needs for growth. Their basal metabolism, under good conditions, is, however, strikingly low, probably because they have a relatively small amount of active heat-forming tissues. Because of their slight muscular activity, less than 10% of the basal metabolism is used up in muscular exercise. The loss of calories in the excreta is, moreover, usually less than 10% of the food intake, unless there is diarrhea. In spite of these slight advantages, the disadvantages under which they labor greatly outweigh them. They require from one fifth to one quarter more calories per kilo than do normal infants. It is impossible to state exactly how many calories per kilo a premature baby needs, because experience shows that some babies need far more than others. Talbot thinks that approximately two hundred calories daily are necessary. I have, however, seen most satisfactory gains on caloric intakes varying between 120 and 150. In spite of their greater need for food, they are, however, less able to take and digest it than full term babies. It is evident, therefore, how great the disadvantages are under which they labor.

Treatment.—In the first place, everything should be prepared as far as possible for the care of a premature infant before it is born. The exposure incident to getting things together for the baby after it is born may easily be the cause of its death.

There are two objects to be attained in the care of premature infants. The first of these is to keep the baby alive; the second, to develop its organism to the stage normally reached at full term. The second of these is often forgotten, but is almost as important as the first. It must always be remembered that in all probability a baby has not reached full term development at the time that the day arrives when it would normally have been born.

The two most important points in the care of premature infants are the maintenance of the animal heat and the provision of a suitable food. It is unquestionably of advantage to protect them from noises, bright lights and handling, because in this way the normal intrauterine conditions are more nearly approached, but the importance of all these measures is infinitely less than the maintenance of animal heat and the getting of a suitable food. Premature babies must be let alone and not handled. Handling cannot possibly do them any good and is almost certain to do them harm. They ought not be picked up or disturbed in any way. Premature babies ought not be regarded as curios and shown to everyone who happens to come along. Every person that sees them disturbs them to some extent and increases the chances of exposure and the

dangers of infection. No one but the immediate family should be allowed to see them and they should be allowed but one look. This applies especially to grandparents and children.

Maintenance of Animal Heat.—While attempting to keep up the infant's animal heat, it must not be forgotten that both fresh air and pure air are necessary for its well being. It cannot thrive on air which has lost its oxygen and it will be infected by bacteria-laden air even if it is kept warm. There are several ways by which the animal heat may be kept up. These are—special rooms in hospitals, incubators and substitutes for incubators. There are certain advantages in special rooms in hospitals. The temperature of the room can be kept at any point desired. Theoretically, the air can be kept pure and at any degree of humidity desired; practically, it seldom is. When a premature baby is in the room with several others, it is likely to be disturbed by them and their

FIG. 39.—Padded crib.

care and they are likely to be disturbed by it and its care. They are less likely, moreover, to get individual attention and have a greater exposure to infection, because of the considerable number of persons who go into the room, than the baby that is alone. They are, however, better off in one of these rooms than they would be in an open ward or at home, unless they can have good nursing and intelligent care.

The ideal incubator is one which will maintain any temperature desired constantly and at the same time provide a sufficient supply of pure, fresh, warm, moist air. I have never seen one which will do this. Most of them will maintain a constant temperature or can be made to do so. None of them provides a sufficient supply of pure, fresh, warm air. None of them can do so, unless some better system of ventilation is devised than has been up to the present time. One result of the lack of fresh air is a diminution in the baby's vitality and in its resistance to infection. Bacteria grow most luxuriantly at the temperature at which the incubator is kept, so that another result of the lack of fresh air is an increased liability to infection. My experiences lead me to believe, moreover, that premature infants do better, if they have air to breathe

of a slightly lower temperature than that at which the air of the incubator is kept. I prefer, therefore, some substitute for an incubator to an incubator itself.

The best and most available substitutes for an incubator are a padded crib or basket. If a crib is used, it must be a small one. An oval clothes basket or a basinette is very satisfactory. The bottom and sides of the crib or basket must be padded thickly with cotton. The top should be covered with a blanket which reaches to a little below the baby's neck. The temperature of the baby's immediate surroundings can be kept at any temperature desired by the judicious use of hot water bottles or bags or, better, by electric heating pads. This temperature should be between 95° F. and 90° F. The temperature should be taken from a thermometer which is wrapped in the baby's clothing and not from one hung in the crib. The dangers of overheating and of chilling have already been mentioned. Both can be, to a certain extent, guarded against by the regular observation of the infant's rectal temperature. Premature babies, whose rectal temperature is about 99.5°F., usually do best. It should not be allowed to go below 98° F. or above 100° F., if it can be prevented. The temperature of the room should be kept between 75° F. and 80° F., thus giving the baby air to breathe of a somewhat lower temperature than that of its immediate surroundings. The

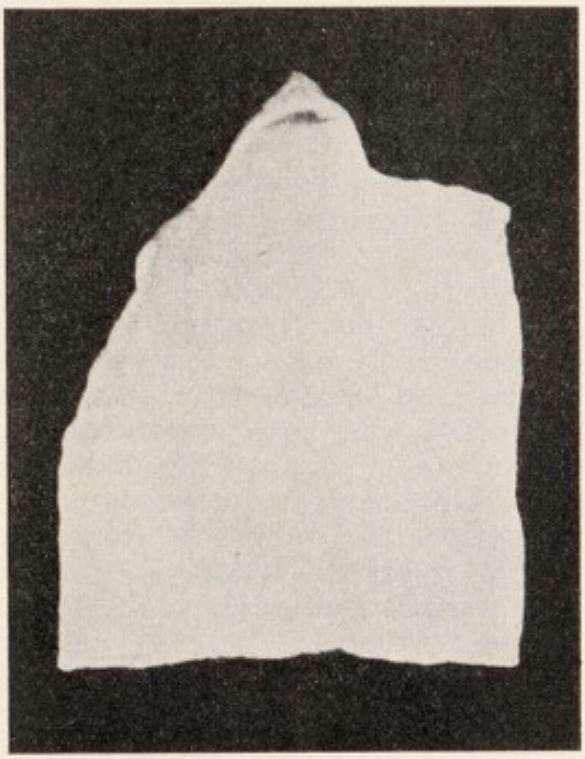

FIG. 40.—Gown for premature infant.

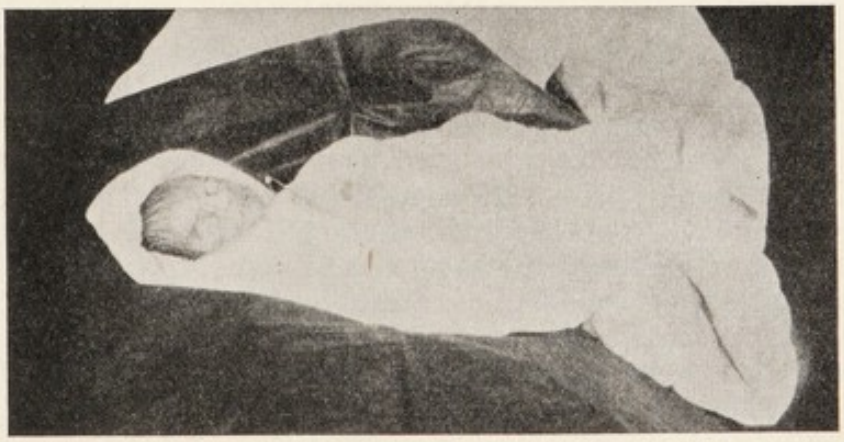

FIG. 41.—Premature infant in gown.

infant should be kept in the room by itself. The room should be sunny and have an open fireplace, with a fire in it, if possible. The crib or basket should be protected from drafts by screens. If it is possible to do so without getting the temperature too low, a window should

be open. It is of some advantage to have the air moist. Practically, however, it is almost impossible to keep it so.

Other important methods of keeping up the animal heat are those which prevent the loss of heat. Most of these methods have an additional advantage in that they prevent handling. The baby ought not be bathed, not even at birth. It should be oiled with olive oil then and every two or three days afterward. This gradually cleans it and keeps the skin in good condition. It should be oiled in its crib, not in the nurse's lap. It ought not be dressed, but should be wrapped in absorbent cotton, or better, in a quilted gown with a hood. The gown is made by quilting cotton between two layers of cheesecloth. This protects the baby better than cotton alone and makes the care much easier. A diaper may be used for fairly strong babies. Absorbent cotton makes a satisfactory substitute for the feeble.

Feeding.—The best food for premature babies is human milk. All the reasons which make human milk the best food for full-term babies are doubly applicable in the case of premature infants. If much premature, they cannot be put to the breast, because of the consequent exposure and handling. Very feeble babies are, moreover, unable to suck. It is a very common mistake to put babies to the breast that are not strong enough and to think that they are getting food, when they are really getting nothing. It has been suggested that the nurse lean over the crib and put the nipple in the baby's mouth or that she nurse a strong baby on one breast while the premature baby is put to the other. These expedients seem unnecessary and unsatisfactory, because it is impossible to know just how much the premature baby gets and even these maneuvers tire it almost as much as the ordinary way of nursing. It is far better to get the milk with a breast-pump or to express it and feed it to the baby in some other way. Furthermore, many premature infants, in fact most in the beginning, are unable to digest full-strength human milk. It is safer, therefore, at any rate at first, to dilute the milk with water in the proportion of two or three parts of water to one part of milk. A small amount of milk sugar may or may not be added. Premature infants usually do better on milk two weeks or more old than on colostrum. In the beginning, therefore, the milk of a wet nurse is better than that of the mother.

The most suitable food, if human milk cannot be obtained, is some modification of cow's milk. In my experience, it is inadvisable, even if the power of digesting starch is present, to give starch to premature infants. They are usually able to digest sugar better than fat and fat better than proteins. The various methods for preventing the formation of large casein curds and for facilitating the digestion of casein may be tried. In my experience, however, the best method, in most instances, of preventing the formation of casein curds is the whey mixture. I have also found that it is even more important to give premature than full-term babies weak mixtures in the beginning. It is very easy to increase the strength of the mixture, if the baby is not satisfied. If too strong a mixture is given in the beginning, it may kill the baby, and will almost certainly cause disturbances of digestion which will require days or weeks to correct. It is never a mistake to give too weak a mixture in the beginning, even if for a time it has to be strengthened every day or two. While I appreciate that premature babies need relatively a greater caloric intake than full-term babies and that they cannot gain unless the caloric intake is sufficient, nevertheless, I feel that in the beginning it is

far more important to plan a food so that it will not upset the baby, or perhaps kill it, than it is to try to meet the caloric needs. This matter can be attended to later.

The strength of the first mixture given must necessarily depend on the age, weight and general condition of the individual infant. Strong babies and those near full term can take stronger foods than feeble or very premature infants. The following mixtures are suitable: Fat, 1%; sugar, 3%; proteins, 0.25%; fat, 1.50%; sugar, 4%; proteins, 0.50%; fat, 2%; sugar, 5%; proteins, 0.75%. As large a proportion of the protein as possible should be given in the form of whey protein. If whey mixtures are not used, one of the other procedures to make the digestion of the casein easier, such as boiling or the addition of citrate of soda or of lactic acid, may be tried.

The amount to be given at a feeding must necessarily vary with the number of feedings and with the capacity of the individual infant. It is never a mistake to begin by giving too little. Irreparable harm may be done by giving too much. It is easy to increase the amount, if the baby can take care of it. I usually begin with one or two teaspoonfuls at a feeding.

A feeding should be given in from six to twelve hours after birth, according to the condition and strength of the baby. It should then be fed at regular intervals, not waiting for two or three days before getting on to them, as in the case of full-term infants. There is much difference of opinion as to how often a premature baby should be fed. I am quite certain that it is inadvisable to feed them as often as every hour or every hour and a half as used to be done, as these short intervals leave almost no time for rest and sleep. On the other hand, I am also convinced that it is not advisable to feed them at such long intervals as four hours, unless the baby is fed through a catheter. I usually start with two hour intervals and change to two and one half hour intervals as soon as possible.

When the infant is strong enough to take food from a nipple, it should be fed from the bottle. Many babies are not strong enough to do this, however, and have to be fed in some other way. The most satisfactory way of feeding such babies is with the "Breck Feeder." This consists essentially of a graduated glass tube, open at both ends. On the smaller end is a nipple about the size of the rubber of a medicine dropper. This is perforated and goes into the baby's mouth. On the other end is a large rubber finger-cot. By squeezing the finger-cot milk is forced into the baby's mouth and efforts toward sucking aided or induced. Some babies are too feeble to take food even in this way and have to be fed with a dropper or a spoon. If a dropper is used it is advisable to put a small piece of rubber tubing on the end of it to prevent injury to the mouth. When it is difficult to get premature babies to suck and swallow well and, therefore, impossible to give them sufficient food, it is advisable to feed them through a stomach tube. There is very little shock to passing a tube in a small baby and they are much less tired by it than by the exertions consequent to attempts at feeding in other ways. The tube should be passed through the mouth, not through the nose. An ordinary soft, rubber catheter attached to a funnel should be used, the size being #12 French, #8 American, or #5 English. It is important not to pass the catheter in too far. The distance from the gums to the cardia is approximately six inches. The milk should be allowed to run into the stomach, the funnel not being raised more than six or eight inches above the level of the body. In order to prevent vomiting as the result of dribbling of the milk from the catheter into the

pharynx during its removal, the tube should be pinched firmly and withdrawn very quickly.

Nursing.—It is evident from what has been said that it will require the whole time of two ablebodied women to care for and feed one premature infant. No one person can do it properly, as the constant attention and frequent feedings never allow more than one hour of rest or sleep, and usually much less. Everything depends on the care and watchfulness of the attendants. Their position is a far more important one than that of the physician.

I realize, of course, that such ideal conditions can only be attained when people are well-to-do and do not have to consider expense. There is no reason, however, why they should not be kept as an ideal in other cases in which the circumstances are less favorable and approached as nearly as possible. When everything can be done regardless of expense, I think premature babies do much better in their own homes than in a hospital. They receive more individual attention and are much less exposed to infection. It is almost impossible to exaggerate the dangers of infection in premature babies and the importance of its prevention. For the same reasons, I prefer even only moderately good surroundings at home to those of a hospital.

Stimulation is seldom of much use. Strychnia, in doses of $\frac{1}{1000}$ of a grain, is the best, when constant action is desired. Caffein and camphor may be used in emergencies, the dose being about one half that for infants during the first year. Oxygen is very useful when there is cyanosis and will sometimes carry babies through very critical periods. It is always well, therefore, to have oxygen close at hand. Babies who have a plentiful supply of fresh air rarely need it, however.

Prognosis.—The prognosis depends chiefly on the age and weight of the infant and the care which it receives. The older the infant, the better the prognosis. Every day counts. There is, of course, nothing in the old saying that seven months' babies are more likely to survive than eight months' babies. It probably originated in the fact that seven months' babies were given special care while eight months babies were treated in the same way as those born at full term. The prognosis is almost absolutely bad when the weight is under two pounds. It is very fair when it is over four pounds. Every ounce of weight over two pounds increases the chances of survival. The prognosis is better when the premature birth is the result of an accident or some acute disease in the mother, than when it is the result of some chronic disease, uremia or placenta prævia. The importance of care and of attention to the minutest details of the treatment has already been mentioned. Too much stress cannot be laid upon it.

The prognosis is very much better in babies that are fed upon human milk than in those that are artificially fed. Nevertheless, many of them do well when fed artificially, as is shown by the following chart. It may surprise some of the younger generation, who are quite sure that they have discovered the use of calories in infant feeding, that this chart, which is copied from an article published by me in 1904, shows the daily caloric intake.

Premature infants that are doing well usually run a slightly elevated temperature; those that are doing badly almost always run a subnormal temperature. A drop in the temperature should always be regarded as a sign of danger, even if all other conditions seem favorable. Premature infants that are apparently doing well in other ways often go many

weeks without any gain in weight. This is not a cause for discouragement, because during this time they are almost always gaining steadily in development and approaching the status of the normal, full-term infant. We should be satisfied for the time, if we are developing a normal baby. It is easy enough to make it gain later.

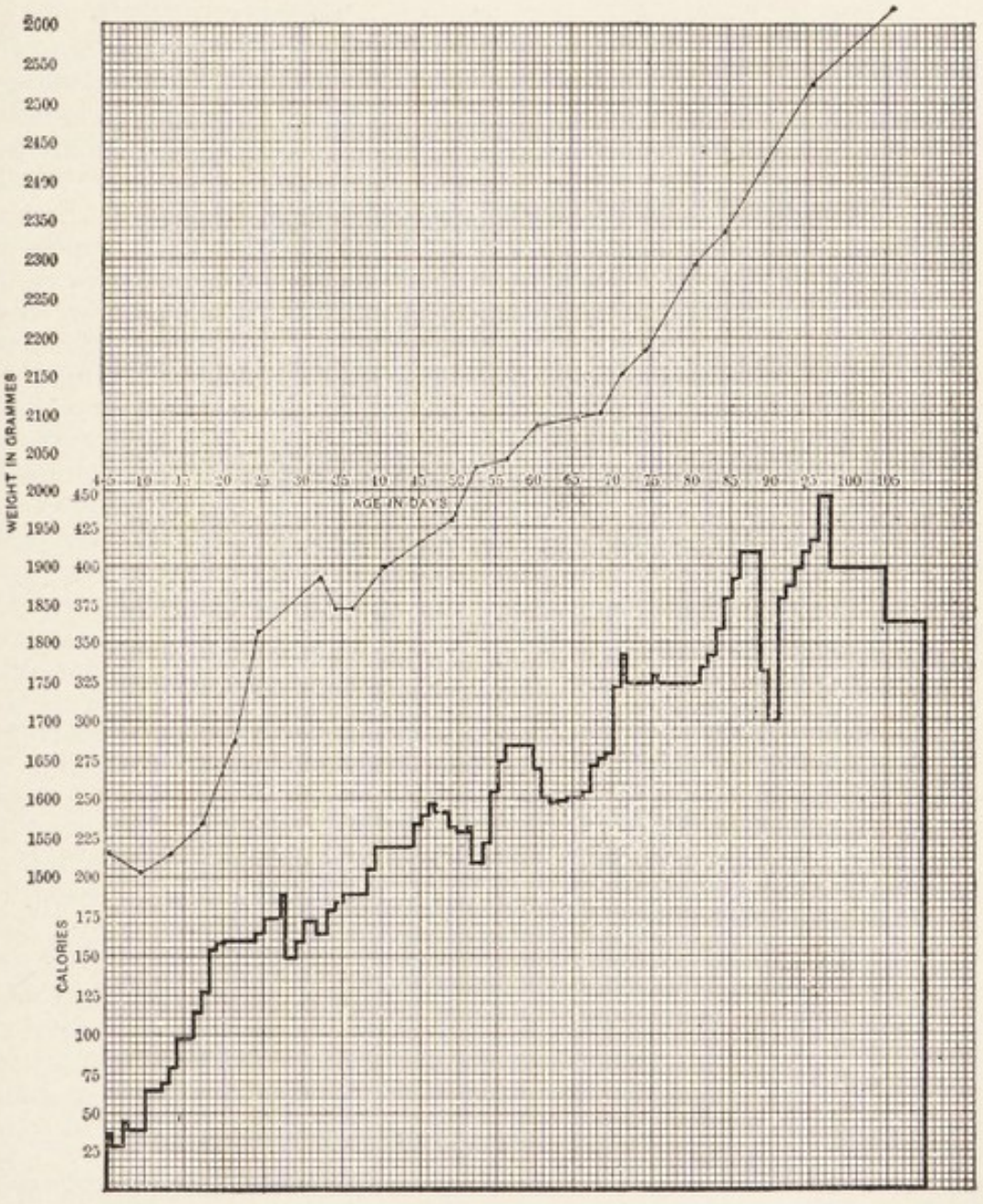

CHART I.—Chart of premature infant. Shows gain and daily caloric intake.

Premature babies are very apt to die suddenly without any apparent cause. It is never safe to consider them out of danger until they are thriving under normal conditions. Up to this time the prognosis should always be guarded. If they survive, they become as vigorous and as large adults as do full-term babies.

SCURVY

Infantile scurvy, sometimes called Barlow's disease, is the same disease as scurvy in adult life. It seldom develops during the first six months of life and is most common during the second six months. It is less frequent during the second year and very uncommon during childhood. It is much less common in the breast-fed than in the artificially-fed, in babies fed on raw than in those on heated milk and in those fed on foods prepared with milk than in those fed on foods without milk.

Etiology.—Infantile scurvy is due to a deficiency of the antiscorbutic vitamin C in the food. There is very seldom a deficiency of this vitamin in human milk. If there is, on account of a lack of it in the mother's diet, babies on the breast develop scurvy in the same way as when there is a deficiency of it in other foods. One of the reasons that babies seldom develop scurvy during the first six months is that many more of them are breast-fed at this time than later. Another reason is that, unless the deficiency is excessive, a number of months is required before active scurvy develops. The amount of the antiscorbutic vitamin in cow's milk depends very largely on the feed of the cows. There is, therefore, likely to be less of it in winter milk than in summer milk. The amount of the antiscorbutic vitamin in cow's milk is, at the best, not very large. At least a pint of raw milk is required daily by an infant to supply the requisite amount of this vitamin. The explanation of the development of scurvy in babies taking raw milk is, therefore, either that they do not get a sufficient amount of milk or that the milk is deficient in its vitamin content. Heating milk diminishes or destroys the antiscorbutic vitamin. Boiling it for a short time has less effect upon the antiscorbutic vitamin than pasteurization for a longer time. This is apparently because oxidization destroys this vitamin and there is more opportunity for oxidization during prolonged than during short heating. It is probable that the differences in the opportunities for oxidization during the heating of milk accounts very largely for the differences of opinion regarding the effect of the heating of milk on the development of scurvy. The antiscorbutic power of condensed and dried milks is less than that of raw milk. How much the antiscorbutic power is diminished depends on the methods used in condensing and drying the milk, that is, upon the amount of oxidization which occurs during the process. The antiscorbutic content of milk steadily diminishes with time, so that old milk contains less than fresh milk, although it may be sweet and suitable in every other way. Alkalinization diminishes and quickly destroys the antiscorbutic vitamin, especially if the food is at the same time heated. The alkalies which are present in most of the proprietary foods probably account, in part, at least, for the frequency with which scurvy develops in babies taking them.

Season and climate have nothing to do with the etiology of scurvy. It is not dependent upon hygienic surroundings and has no direct connection with other disturbances of nutrition. It is not caused by an insufficient amount of food. It is not a result of congenital or inherited debility, although apparently there may be a familial predisposition to it. Infection, however, does undoubtedly precipitate the development of acute symptoms, when they have been slight and unnoticed previously.

Pathology.—The changes in the bone marrow are characteristic. They are most marked at the ends of the diaphyses of the long bones and the anterior ends of the ribs. The bone marrow, which is normally rich in lymphoid cells, loses its lymphoid character and is converted into a

tissue poor in cellular elements and containing relatively few blood vessels. This tissue consists of a homogeneous ground substance containing spindle and stellate cells. There is still much calcified ground substance, but it has not been converted into true bone. As a result of the interference with the normal processes of ossification, the cortex of the bones is thinner and more brittle than normal and the density of the bones is materially diminished at the epiphyseal lines. Fractures of the shafts occur very readily, therefore, as the result of very slight injuries. These occur most often at or near the epiphyseal line. The epiphyses are often loosened and separated. Marked displacement of the epiphysis is, however, uncommon, because the periosteum usually remains intact. It is for this reason also that deformity almost never occurs after healing.

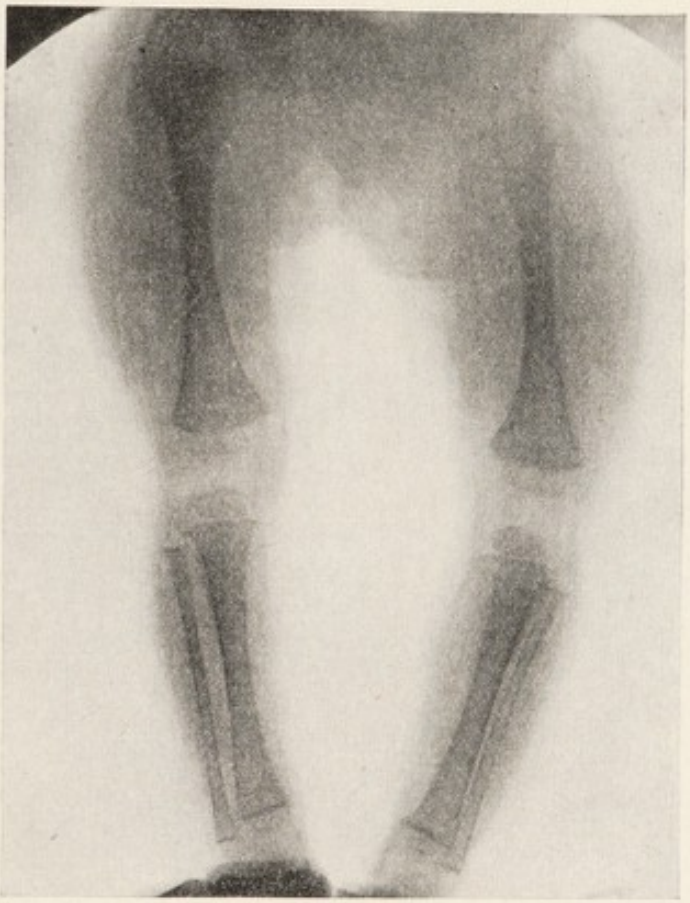

FIG. 42.—Scurvy. Early stage. Note thickening of periosteum.

The periosteum of the long bones is thickened and congested, but shows no excess of leucocytes or small round cells. Hemorrhages between the periosteum and the bone are very common and may be very extensive. They may break through the periosteum into the surrounding tissues. Small hemorrhages in the marrow of the bones are also not at all uncommon, but cannot be recognized clinically. Subperiosteal hemorrhages are much more common in the lower than in the upper extremities. Ossification may take place in the displaced periosteum. These changes are all easily perceptible with the aid of the Roentgen ray.

Hemorrhages may occur in any of the internal organs. They are common in the skin and are found at autopsy in most of the serous membranes. They may also occur in the intestinal mucosa. Hemorrhage into the intestine is, however, very unusual. Hematuria without

inflammatory processes in the kidney is common. A hemorrhagic condition of the gums is a common symptom when the teeth have erupted. It is, however, very uncommon before the teeth have erupted. Hemorrhage sometimes takes place in the orbit, pushing the eye forward. Sometimes it occurs also under the dura or into the joints.

There is not infrequently enlargement of the heart. It is most often of the right ventricle and is more often due to dilatation than to hypertrophy.

The blood shows the changes characteristic of mild or moderate anemia in infancy. The clotting power of the blood is, as a rule, slightly diminished, but this diminution is not constant and, therefore, cannot

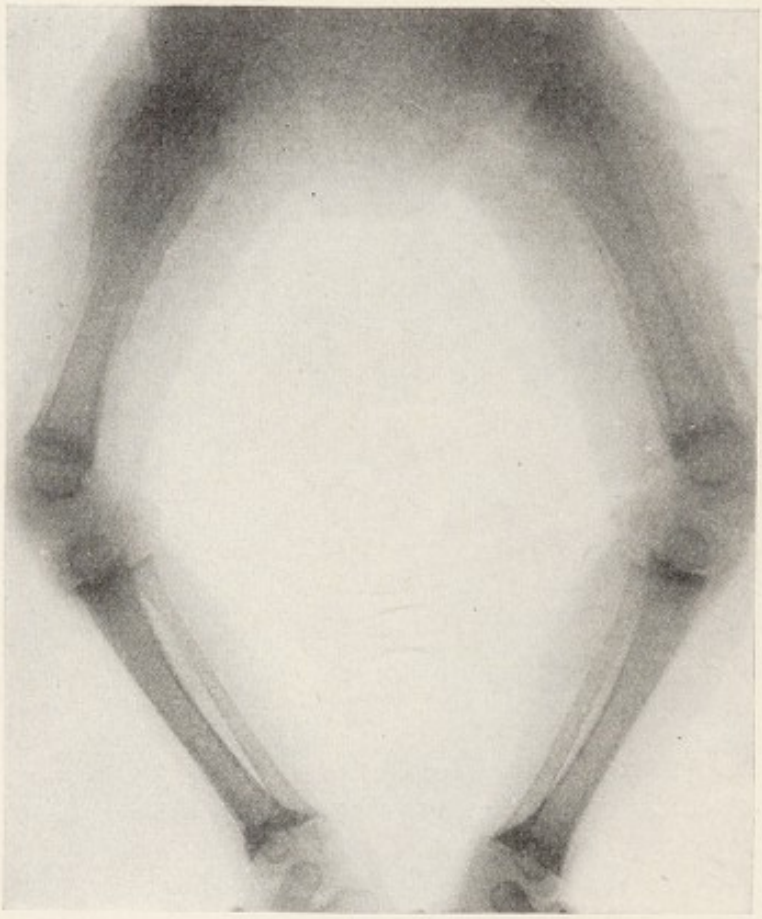

FIG. 43.—Scurvy. Large subperiosteal hemorrhages.

account for the hemorrhagic tendency in the disease. There is no deficiency of calcium or of blood platelets and no excess of antithrombin. The resistance of the capillary walls is diminished. This is, however, not pathognomonic of scurvy. It is probable that the hemorrhagic tendency in scurvy is due to some change in the vessel walls rather than in the blood. A form of edema infiltrating the skin and muscles, which does not pit on pressure and is most marked in the lower extremities, is not uncommon. It also is probably due to a nutritional disturbance of the smaller vessels.

The increase in the rate of the pulse and respiration, the exaggerated knee-jerks and the edema of the optic discs, which has been found in some cases, suggest very strongly that the nervous system is also involved. The peripheral nerves are frequently the site of hemorrhages. In a fatal case, described by Hess, there was focal degeneration of the lumbar

cord, the most marked lesion being a loss of cells in the lateral groups of the left anterior horn.

Little is known as to the metabolism in scurvy, either in adults or infants. The results obtained suggest that there is a disturbance in the balance of the mineral salts, but they are not constant. It is evident, however, that these changes are secondary and have no relation to the etiology of the disease.

Symptomatology.—The early symptoms of scurvy are almost invariably overlooked. If not overlooked, they are usually misinterpreted. One of the earliest symptoms is pallor. Another is failure to increase properly in weight and length. Another quite characteristic symptom is loss of appetite. The improvement in the appetite, which occurs so frequently when orange juice is given, although scurvy has not been suspected, shows how often anorexia is really a symptom of scurvy instead of indigestion or of some other disease. The rapid gain in weight and length after orange juice is given, also shows how often disturbances of this sort may be unrecognized symptoms of scurvy. Slight edema of the extremities or of the eyelids, usually more marked in the eyelids, is usually a somewhat later symptom. An increase in the rate of the pulse and of the respiration, usually proportional, without evident cause, is another early symptom. It is to these early symptoms that the term "latent scurvy" is sometimes applied.

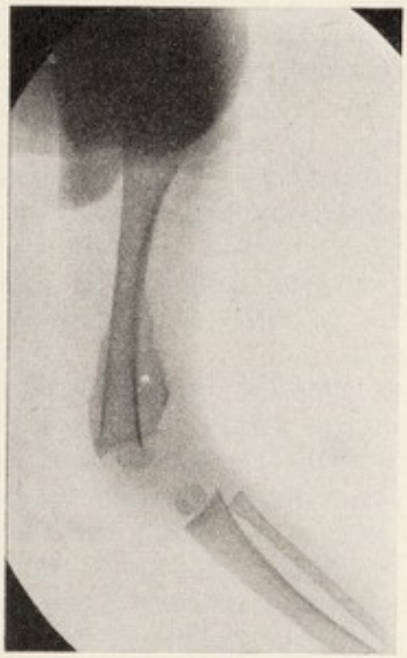

FIG. 44.—Scurvy. Subperiosteal hemorrhage and dislocation of epiphysis.

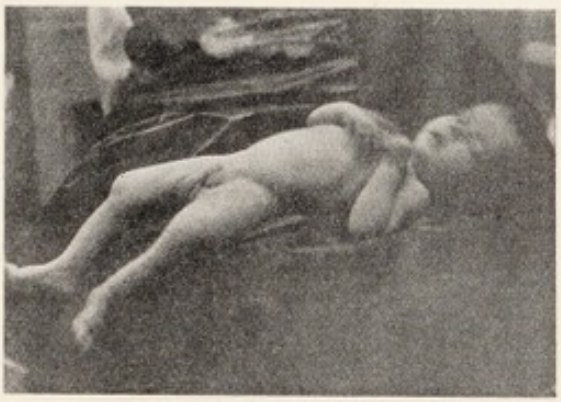

FIG. 45.—Characteristic position in scurvy.

When the symptoms become definite enough to be recognizable as characteristic of scurvy, they may be roughly divided into three main types, which, of course, may be combined. In the first, or usual, type, tenderness of the legs and back on handling is the first thing noticed. Mothers usually say that it hurts the baby when its diapers are changed or when it is having its bath. The tenderness and pain on motion gradually increase, and finally the baby ceases to use its legs. It usually holds them with the thighs partially flexed on the abdomen and rotated outward and the legs partially flexed on the thighs. Sometimes the baby ceases to use the legs before tenderness is noted. Sometimes the tenderness and loss of power develop very quickly. The onset is so sudden in some instances that the trouble is attributed to some coincident fall or other minor injury. Swelling usually develops soon after the tenderness. This swelling is

usually located about the diaphyses of the long bones of the extremities, more often about their lower ends. The lower extremities are involved very much more frequently than the upper. The swelling in the beginning is often due to a hard infiltration of the muscles and tissues over the bones, but may be due from the first to hemorrhage under the periosteum which lifts it from the bone. This is almost always the cause, later. In many instances both conditions are present. There is no hemorrhage into the joints and usually no swelling about the joints. If the hemorrhage breaks through the periosteum, the swelling may be due to blood between the muscles and many extend over the joint. There may also be separation of the epiphyses, most often of the lower epiphyses of the femurs. These changes are all beautifully shown by the Roentgen ray. The tenderness of the extremities is extreme and the babies are very much afraid of being touched. I have never seen such abject terror in any human being as is shown by some of these babies with scurvy. Although the extremities are not moved, there is no paralysis. The cessation of use is due to the pain which motion causes, not to inability. Passive motions are not limited, except by the swelling. The knee-jerks are present and usually exaggerated.

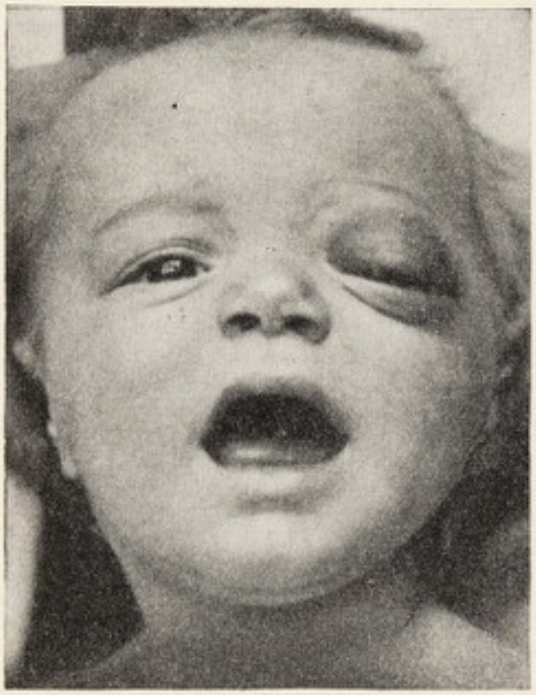

FIG. 46.—Exophthalmos in scurvy.

In the second type, the earliest pathognomonic symptom is a purplish swelling of the gums about the teeth. This swelling sometimes occurs in the gums when the teeth are just ready to erupt, but never otherwise, unless there are teeth. It usually appears about the upper incisors before it does the lower incisors. It sometimes develops behind the teeth before it does in front. These changes in the gums may persist for weeks before any other symptoms develop. The swelling may be so great as to almost cover up the teeth. It is often soft and spongy. There may be bleeding from the gums, but there usually is not.

In the third type the first symptom is hematuria. Attention is usually called to it because the urine stains the diapers brown or red. There are no other urinary symptoms. The urine is usually bright red, sometimes brownish. Microscopically it almost never shows anything but normal red blood corpuscles. Occasionally there may be an excess of cells and a rare cast.

The symptoms of these various types are, as already stated, likely to be combined, and also to be associated with other symptoms. The most common of the other symptoms is marked pallor. This is often associated with an ordinary edema, pitting on pressure, different from the firm infiltration in the extremities which has already been described. Hemorrhages are very common in the severe cases. They are most often into the skin. The single lesions are usually not large. Bleeding from the nose and bowels is very unusual. Hemorrhages into the internal

organs, while pathologically common, are almost never large enough to cause any symptoms. The most characteristic seat of hemorrhage is into the orbits. This results, of course, in exophthalmus, which may be either unilateral or bilateral. It is usually associated with ecchymosis of the lids and sometimes into the conjunctiva. It is said that there is not infrequently edema of the optic discs. I do not know about this, as I have never looked for it. Enlargement of the heart, especially to the right, is not uncommon. The rate of the heart is increased more than would be expected from the degree of anemia present. The rate of the inspiration increases parallel with that of the pulse. Murmurs in the heart are common, but are due to the anemia, not to a cardiac lesion. As a rule, there is no elevation of the temperature. In certain cases, however, there may be, and sometimes it may be considerable, even as high as 104° F. It is usually stated that the rise in temperature is due to some complication, not to the scurvy itself. This statement is probably true in most instances. I believe, however, that it may be due entirely to the scurvy. The reason for this belief is that in a considerable number of instances, in which no evidences of complications could be found, I have seen the temperature drop to normal within two or three days after orange juice was begun. A rosary, indistinguishable clinically from that of rickets, is said to develop not infrequently in scurvy. I have never been sure of it. If scorbutic, it disappears quickly with antiscorbutic treatment. The rosary of rickets is, of course, not modified by it.

Diagnosis.—Scurvy should always be suspected when babies lose their appetite, fail to gain in weight and are losing color, especially if they are taking a pasteurized, boiled or dried milk or one of the proprietary foods containing an alkali. It should also be considered as a possibility, even when babies are on the breast or taking raw milk. If there is slight edema of the extremities or eyelids or an increase in the rate of the pulse and respiration with, perhaps, slight exaggeration of the knee-jerks, the diagnosis of scurvy is still more probable. It can be made positively, however, only by giving an antiscorbutic and observing the results. Improvement is immediate, if the underlying trouble is scurvy.

In the type in which there is pain, swelling and diminution in the use of the extremities, the most common mistake is attributing the symptoms to rheumatism. Rheumatism, however, almost never occurs in infancy. If it does, it is almost never accompanied by swelling or localized tenderness. If it is accompanied by these signs, they are located about the joints, not over the shafts of the bones, and are likely to be associated with heat and redness. Rheumatism is usually accompanied by fever, scurvy usually is not. Rheumatism is not accompanied by swelling and discoloration of the gums, hematuria and ecchymoses. One or more of these signs is usually present in scurvy.

Periosteitis and osteomyelitis are also sometimes thought of, because of the swelling and tenderness in the extremities. In these conditions the trouble is almost always limited to one extremity and usually to one bone. The tenderness is more sharply localized. In periosteitis there is often superficial heat and redness. In scurvy the swelling and tenderness are almost never limited to one extremity and are usually present over several bones. There is no superficial heat or redness. Edema is more common in these conditions than in scurvy, but may occur in both. The temperature is always high in these diseases, but a high temperature does not rule out scurvy, as it may also be present in this disease.

The general condition is much worse in these conditions than in scurvy. They always have a marked leukocytosis and are never accompanied by other signs of scurvy, such as swollen and purple gums, hematuria and ecchymoses. The onset in them is usually much more acute.

When there is much swelling and it is, as sometimes happens, more marked in one extremity than in the other, scurvy may be mistaken for sarcoma of one of the long bones. Such a mistake should not be made, however, because there are always other evidences of scurvy in the extremities or elsewhere in the body. Examination with the Roentgen ray will settle the diagnosis at once in this, as in many other of the conditions which are mistaken for scurvy.

Hip disease is also often thought of, because of the apparent tenderness about the hip and the failure to use the legs. Tuberculous hip disease is very unusual at this age. In it the limitation of motion is in the hip joint and the swelling, if present, is in or about the joint, not over the lower portion of the diaphysis of the femur. It is usually unilateral and is not accompanied by other signs of scurvy. Acute infectious arthritis of the hip can be distinguished from scurvy as are osteomyelitis and periosteitis.

Scurvy is often mistaken for Pott's disease, because of the apparent tenderness in the back on handling and the failure to use the extremities. Tuberculosis of the spine is however, very unusual in infancy. Passive motions of the spine are limited, there is usually deformity of the spine, and almost invariably psoas contraction. There are, of course, no other signs of scurvy.

Scurvy is often mistaken for an injury, because of the pain on handling and motion and the failure to use the extremities. This mistake is especially likely to be made when the onset of the symptoms is acute. Too much importance is attributed to the history of a fall, strain or injury of some sort, which can be elicited by suggestion and cross questioning in every case. No confusion should arise, however, if the symptomatology of scurvy and that of the various injuries under consideration are carefully borne in mind.

Various forms of paralysis, especially infantile, are often considered on account of the apparent loss of power in the extremities. The failure to use the extremities in infantile paralysis is due to a real loss of power. The extremity is flaccid and usually not tender. In scurvy, the failure to use the extremity is due to the pain which motion causes. It is held rigidly, is tender and passive motions cause pain. There is never swelling in paralysis. There often in in scurvy. Other signs of scurvy are never present is infantile paralysis. The onset of infantile paralysis is sudden, that of scurvy, slow. Multiple neuritis has a more general distribution, almost never occurs at this age, is accompanied by wasting, never by swelling, and never by other signs of scurvy.

In the type of scurvy with swelling and discoloration of the gums, the most common mistake is to attribute the symptoms to difficult dentition. It is difficult to understand how this mistake can arise, when the swelling and discoloration develop, as they almost always do, about teeth that are already erupted. It is easier to understand, however, when they develop about teeth which are only partially erupted, and still easier when they develop before the eruption of the teeth. This, however, very rarely happens. The color of the swelling and the peculiar sponginess of the gums in scurvy are entirely different from the conditions seen in difficult dentition, in which the gums are tightly swollen over an

unerupted tooth and bright red in color, while in scurvy the gums are swollen about a tooth, the swelling is soft and spongy and the color dark purple. There ought, therefore, never be any confusion, if the gums are carefully examined.

The type beginning with hematuria is almost never recognized, because most physicians are unware that hematuria may be the earliest and only symptom of scurvy. Hematuria is, however, a very rare condition in infancy, except as a manifestation of scurvy. If the urine contains only blood and shows none of the other evidences of acute nephritis, the difficulty is almost always scurvy. Even if there are a few cells and casts, the chances are that, unless the condition has immediately followed some acute infection, it is scurvy. It is usually impossible to make the diagnosis between hematuria due to scurvy, if it is the only symptom, and that due to other conditions, however, without the use of the therapeutic test, that is, the administration of an antiscorbutic. This will cure the condition promptly, if it is due to scurvy.

Scurvy is sometimes mistaken for other conditions, when special symptoms are unusually prominent. When there is exophthalmos as the result of a hemorrhage into the orbit, the other evidences of scurvy are often overlooked and a diagnosis of sarcoma made. When there is hemorrhage from the gums or the ecchymoses are unusually marked, scurvy is often mistaken for purpura. Occasionally, when the anemia is especially prominent, the characteristic scorbutic manifestations are overlooked and a diagnosis of some severe type of anemia made. All of these mistakes are avoidable, if a careful physical examination is made and the possibility of scurvy as an explanation is kept in mind.

Prognosis.—The prognosis in scurvy is invariably good, if it is properly treated. Death occasionally occurs, if it is not. The acute manifestations begin to improve almost immediately. In those cases in which the symptoms are most marked in the extremities, pain and tenderness are usually the first symptoms to yield to treatment and are soon followed by the return of power in the extremities. Improvement in the condition of the gums usually begins later and progresses more slowly. The disappearance of the swellings about the bones is, as would be expected, always slow. Marked improvement in the swelling, even if it is extreme, is, however, usually noticeable in a week. The mildest cases are usually well in two days, and many cases are entirely well in five days. Almost all show marked improvement in three or four days. Pain and tenderness are almost always gone in one week, while the gums are rarely normal before one or two weeks. Most cases are well, except for the remains of swelling and hemorrhages, in two weeks, while recovery is almost always complete in three weeks. It takes longer, of course, for the disappearance of the secondary anemia and the general disturbance of the nutrition. Even when there has been a displacement of an epiphysis, it comes back into position and the extremity becomes entirely normal. In one neglected case, in which the hemorrhages beneath the periosteum had been excessive and in which there had been bony formation in the periosteum, the periosteum could not be entirely returned to its normal position even with an operation. Such a happening is, however, almost unique.

Treatment. *Prophylactic.*—The development of scurvy can always be prevented by the administration of an antiscorbutic. It hardly seems necessary, however, to give an antiscorbutic as a preventive of scurvy to babies that are on the breast or taking raw milk mixtures.

There is no objection to doing so, however, if desired. An antiscorbutic should always be given, however, to babies that are fed on pasteurized, boiled or dried milk or on the proprietary foods containing an alkali, if these foods are given continuously for more than a month.

An antiscorbutic should always be given when babies begin to become pale, lose their appetites, or cease to gain in weight unless there is some other evident cause for these symptoms.

Curative.—When definite symptoms of scurvy have developed, an antiscorbutic should, of course, always be given at once. Orange juice and lemon juice are the easiest antiscorbutics to use. Orange juice is preferable, because babies take it better. Lime juice has very little antiscorbutic power. One half ounce of orange juice daily is almost always sufficient to prevent the development of scurvy. An ounce daily will

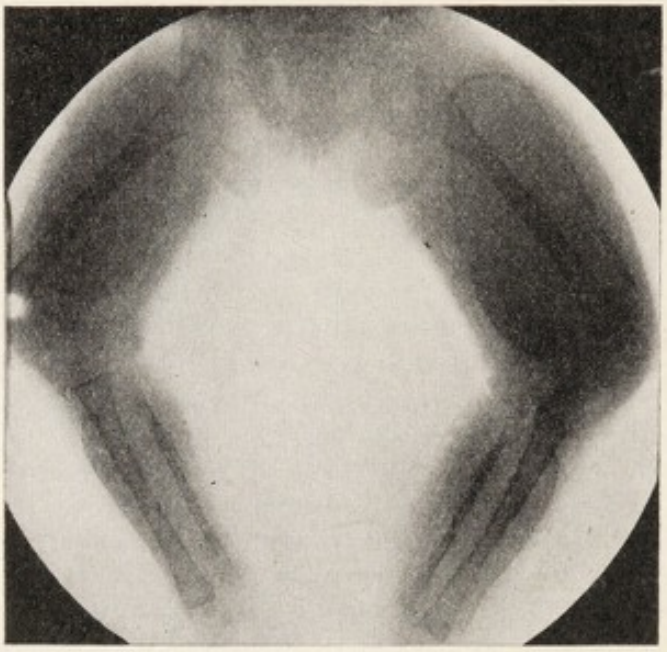

FIG. 47.—Scurvy. Bony formation in periosteum.

always cure it, less may or may not cure it. It may be given either plain or diluted with water. There is no objection to sweetening it with cane sugar. I prefer to give it all at one dose, rather than in several small doses. Orange juice does not lose its antiscorbutic power by being boiled or dried. Aging has no effect on it, but alkalization destroys it. It is active when given intravenously. Orange peel also contains the antiscorbutic vitamin, and may be used instead of orange juice on account of its cheapness. Hess and Fish used it by adding one ounce of finely grated orange peel to two ounces of water and sweetening it. Orange peel retains some of its antiscorbutic power for months after being dried.

The water of strained canned tomatoes can be used in place of orange juice as an antiscorbutic. The dose is the same as that of orange juice. Aging of at least a year in the can does not impair its value. There is no advantage in the use of the juice of canned tomatoes over orange juice, except that it is cheaper, and occasionally, when babies are disturbed by orange juice.

Boiled and mashed potatoes also have considerable antiscorbutic power. A potato water can be made by adding one tablespoonful of boiled and mashed potato to a pint of water, using it in the same way as

barley and other cereal waters. Potato flour and dessicated potatoes have no antiscorbutic action.

Some of the vegetables also have considerable antiscorbutic power, especially the swede. Carrots and beets have much less. It seems hardly necessary to consider these things, however, in the treatment of infantile scurvy when orange juice, lemon juice and canned tomatoes can be so easily procured. Incidentally, germinating grains have considerable antiscorbutic power. Bananas and prunes have none. Beef juice has a little. Canned vegetables have practically none.

There are no drugs which have any preventive or curative action in scurvy. Cod liver oil and olive oil, like the others, are useless.

Immobilization of the extremities by light splints for a few days, before the symptoms are relieved by the antiscorbutic treatment, often affords a great deal of comfort. Under no circumstances should subperiosteal hemorrhages be tapped or operated upon. They always go away of themselves under antiscorbutic treatment. No attempt should ever be made to draw off the blood when there is a hemorrhage into the orbit.

Scurvy can often be cured by a change in the character of the food, that is, by giving a raw instead of a heated food or by omitting the addition of an alkali. It can sometimes be cured by changing the milk, giving that of cows which have more of the antiscorbutic elements in their food. A great many cases of early and mild scurvy undoubtedly recover, without being recognized, as the result of some change in the food. In general, however, it is wiser to keep on with the food which a baby is taking, provided it is satisfactory in other ways, and give an antiscorbutic than to change it.

RICKETS

Rickets is a constitutional disease of nutrition. All the organs and tissues of the body are affected, but the chief lesions are in the bones. These lesions are pathognomonic. Their chief characteristic is a local or general disturbance of the normal processes of ossification. Rickets is most common between the sixth and eighteenth months. It seldom occurs earlier and very rarely begins after the third year. Late rickets is very uncommon, except in times of war or great national distress. It develops, therefore, at a time when the bones are in process of rapid development. It is generally stated that about 50% of all babies show clinical evidences of rickets. I am inclined to think that this proportion is altogether too low. In an investigation which I made in Boston twenty-five years ago, I found that 80% of the babies of the hospital class showed clinical evidences in the bones of this disease. My experience leads me to believe that it is no less frequent now than it was then, although I am quite certain that severe rickets is not as common as it was at that time. Finding slight bony changes of rickets does not mean, however, that the condition is a serious one or that it should cause any anxiety. The importance of these minor changes has, it seems to me, been greatly exaggerated in recent years and more attention paid to them than they really deserve.

Pathology.—The bones grow in length through the formation of bone tissue in the cartilage between the epiphysis and diaphysis. They grow in thickness as the result of the growth of bone from the inner layers of the periosteum. As the bone increases in circumference, the medullary canal is enlarged proportionately by the absorption of the inner layer of bone. Under normal condition these processes progress in regular order

and in clearly defined lines. In rickets there is an overgrowth of the cartilaginous layer between the epiphysis and diaphysis both in width and thickness and it is markedly hyperemic. In this area the zone of proliferation is much enlarged and the cells are arranged irregularly instead of symmetrically, as in normal conditions. The deposition of lime salts and the amount of calcification is, nevertheless, much less than under normal conditions. The epiphyseal centers of ossification are larger, softer and more vascular than normal. There is a similar disturbance in the subperiosteal formation of the shaft. The outer layers of the shaft are thickened, but soft. The medulla of the bone is more hyperemic than normal and the inner layers of the bone also become softened through lack of lime salts.

The visible results of these abnormalities in the growth of the bones are enlargement of the bones at the epiphyseal lines and at the centers of ossification and unnatural flexibility of the bones. On account of this increased flexibility, deformities and sometimes fractures are produced as the result of pressure or weight bearing.

The blood shows a diminution in either calcium or phosphorus, most often in phosphorus. The normal calcium content of the serum varies between 10 and 11 mg. per 100 c.cm. of serum. The inorganic phosphorus averages normally about 5.4 mg. per 100 c.cm. In rickets the product of the concentration in mg. of calcium and inorganic phosphorus per 100 c.cm. of serum is below 40 and often below 30. There is some question as to whether there really are two types of rickets, one characterized by a normal or nearly normal blood calcium and a low phosphorus and the other by a normal or nearly normal blood phosphorus and a low blood calcium, or not. It is certain, however, that in most instances, the blood calcium is normal or nearly so and the phosphorus low.

Etiology.—Much has been learned in the past five years as to the etiology of rickets, but the real etiologic factor has not yet been discovered. It seems almost certain, however, that rickets is due to a lack of something, not to too much of anything. Much of the evidence which has been discovered is important chiefly in that it is negative. It has been shown conclusively that rickets is neither hereditary nor congenital. It has been proved that it is not due to bacterial infection nor to endocrine insufficiency. It is not the result of a lack of calcium in the food. It is not due to a deficiency of vitamin A. It is not due to syphilis.

It seems evident, as it has been for many years, that rickets may develop either as the result of poor environment or of some defect in the food. That is, there is a basis for both of the divergent views which have been so long held as to its origin. The results of experiments on animals and of treatment of both children and animals with the ultraviolet rays and direct sunlight, show that lack of sunlight is the most important, if not the only, etiologic element in poor environment. Lack of sunlight explains the greater frequency of rickets in winter, in cities and in large families living in crowded surroundings, all of which prevent exposure to sunlight. It also explains the greater frequency of rickets in the temperate zone than in tropical and subtropical climates. The reason that Esquimaux babies do not have rickets is probably because they get so much fat in their food.

The etiologic defect in the diet is apparently neither an excess nor a deficiency of any one of the food elements—fat, carbohydrates or protein. It is not a lack of vitamin A, but it is barely possible that there may be some relation between this vitamin and the real cause. A deficiency of

phosphorus or an abnormal relation between the phosphorus and calcium may perhaps be the cause. Excessive amounts of calcium in the diet tend to impair phosphorus retention, while excessive amounts of phosphorus in the diet have an unfavorable influence on the calcium metabolism. It is probable that the retention of one element in the intestine by an excessive amount of the other in the diet is best explained by the formation of insoluble phosphates of calcium which cannot be absorbed (Orr and others). It is probable that defective absorption from the intestines is the cause of the low concentration of calcium and phosphorus found in the serum and the ultimate cause of the defective calcification of the bones. It is very difficult to understand, however, how cod liver oil can increase the absorption of these elements to such an extent as to cure the disease. It is equally difficult to understand how exposure of the body to the direct rays of the sun or to the ultraviolet rays can also increase the absorption sufficiently to cure the abnormalities in the blood. It is still more difficult to understand how two such radically different methods of treatment can accomplish the same end. It seems possible that they both may, in some way, stimulate some unknown factor or factors which regulate the metabolic processes especially concerned with the salts of calcium and phosphorus. The results of the investigations of Hess, Weinstock and others, who have been able to confer antirachitic properties on inactive oils, vegetables, grains and milk by irradiation with the mercury vapor quartz lamp, suggest such an explanation. It has also been found that it is the cholesterol in animal foods and the phytosterol in vegetable foods which are activated and acquire antirachitic properties. Why the activating of these bodies prevents and cures rickets, is nevertheless, just as obscure as ever.

There has been much discussion as to the relationship between rickets and spasmophilia. The explanation seems simple enough. If the blood calcium is low in rickets, spasmophilia develops; if it is not, it does not. Both may, perhaps, be due to the same cause, but neither is dependent upon the other. Spasmophilia may develop when there is no rickets, if the blood calcium is low.

Symptomatology and Physical Examination.—It should never be forgotten that, although rickets is due to a disturbance of nutrition, babies that have it are often fat and heavy. In such cases, however, the normal relation between the fat and muscles is disturbed, the muscles suffering at the expense of the fat tissue. Furthermore, atrophic babies are seldom rachitic and, if so, only to a slight degree. Rickets is more active and more severe when growth is rapid than when it is not. The only pathognomonic symptoms of rickets being the changes in the bones, it is very difficult to know whether other symptoms which are present are really to be attributed to rickets or to other conditions. Early symptoms which suggest rickets, but are in no way pathognomonic, are fretfulness, languor, and sweating, especially of the head. Delay in dentition or in development are also rather suggestive, but may as well be due to other causes. Symptoms referable to the digestive and respiratory tracts are probably really complications rather than evidences of the disease. The best way to deal with the physical examination and symptomatology is probably to take up the different organs separately.

Skeletal Changes. *Head.*—There is delay in the closure of the anterior fontanelle, sometimes even an increase in size, and the edges of the bones about the fontanelle are often soft. There is also not infrequently softening of the bones at the sutures. In my experience,

craniotabes is rather uncommon. By this term, I mean a softening of the bones of the skull, most often of the occipital and parietal, sufficiently marked so that they can be easily depressed locally or soft places can be felt in them. The best way to examine for craniotabes is to place the heels of the hands on the baby's forehead and then to feel over the rest of the head with the finger tips. It should go without saying that it is unwise to exert much pressure. As the result of the abnormal changes at the centers of ossification in the cranial bones, enlargements develop, which are often spoken of as *bosses*. These are most marked in the frontal and parietal bones. When they are very large, there is often a depression between them in the neighborhood of the sutures. The

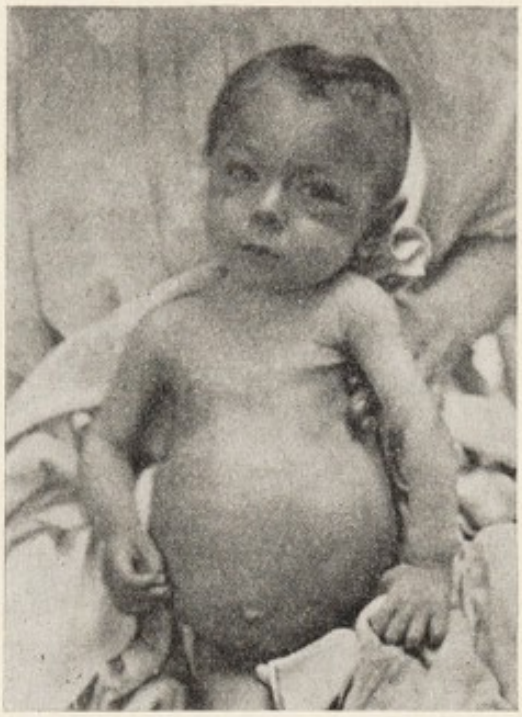

FIG. 48.—Rachitic head.

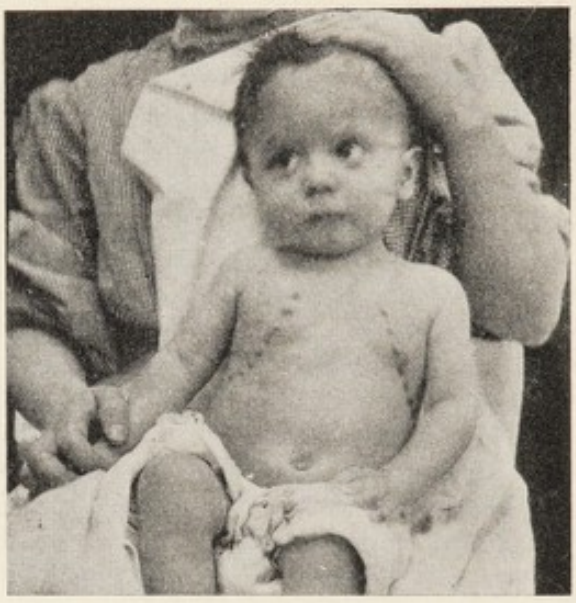

FIG. 49.—Black dots mark location of rosary.

deformity which results is often spoken of as the *square head*. The enlargements of the head from rickets and from chronic internal hydrocephalus are sometimes confused. The enlargement in rickets is due to a deformity of the bones of the cranium, that in hydrocephalus to a pressing outward of the bones as the result of increased pressure on the inside. The enlargement is, therefore, irregular in rickets, symmetrical in hydrocephalus. The fontanelle is level or depressed in rickets, bulging in hydrocephalus. As the result of the increase in intracranial pressure in hydrocephalus the orbital plates are often pushed downward, so that the eyes are pushed forward and the conjunctivae are visible above the irides. This never happens in rickets. Dentition is not infrequently delayed in rickets. It may be delayed, however, from other causes and, in many instances, the appearance of the teeth is normally later than the average. Both the first and the second teeth may be imperfect and decay easily as the result of the changes caused by rickets.

Thorax.—The most common deformity of the chest in rickets is the *rosary*. This is due to an enlargement at the junction of the ribs and their cartilages which forms a round bunch, something like a bead. Hence, the name. Owing to the comparative shortness of the ribs the rosary is

situated farther out from the median line than would be the case in an adult. It is often overlooked on this account. It is most marked from the fourth to the seventh ribs, but varies very much in size. It is important in thin babies not to mistake the normal junction of the ribs and

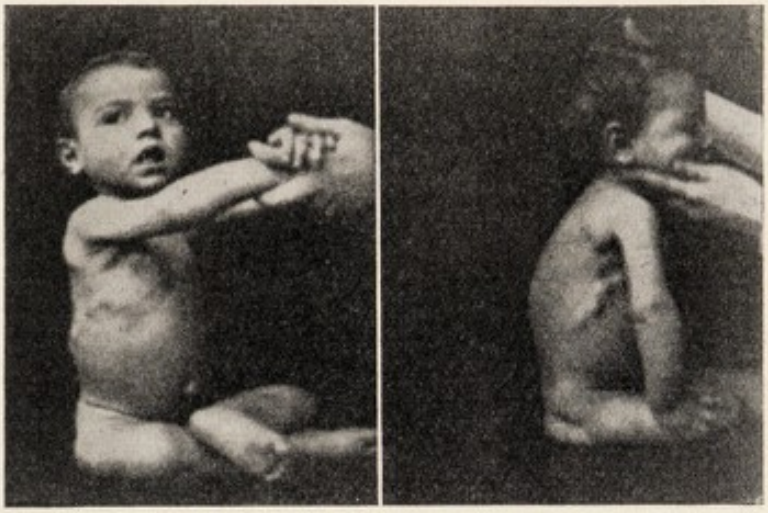

FIG. 50.—Rachitic rosaries.

cartilages for a rosary. It is not safe to make a diagnosis of a rosary unless a definite bulging can be felt at the edges of the ribs as well as on the anterior surface. It is said that a similar enlargement may develop as the result of scurvy. I have never seen it. It is almost always safe

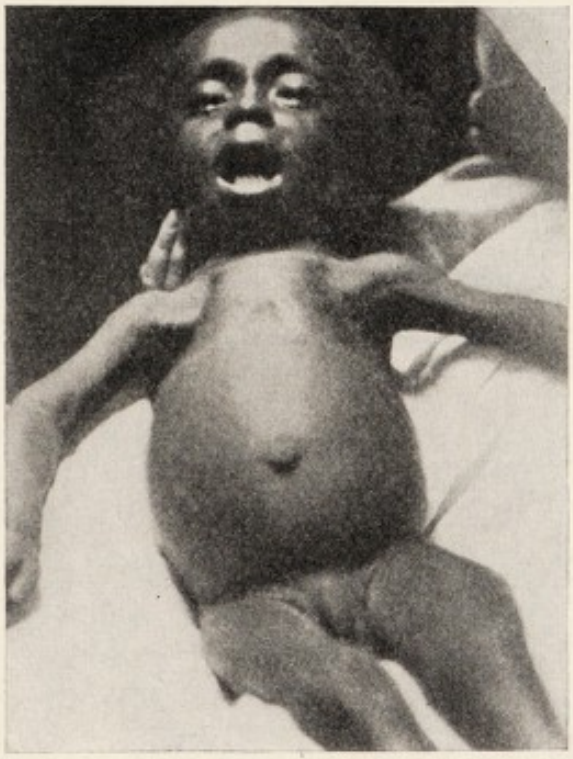

FIG. 51.—Deformity of chest in rickets. Shows also large abdomen and umbilical hernia.

to conclude that a rosary is rachitic in origin. If it is due to scurvy, it quickly disappears when orange juice is given. Orange juice has no effect on the rachitic rosary. In my experience, the rosary is the earliest recognizable sign of rickets. It is present in every instance and in about 40% is the only sign recognizable clinically.

As the result of atmospheric pressure and of the pull of the muscles on the soft bones, protrusion of the sternum often develops. It is known as *pigeon breast* or *chicken breast*. It must be remembered in this connection, however, that some individuals, even in infancy, have more prominent sternums than others. When there is obstruction from adenoids or enlarged tonsils, in connection with rickets, a depression of the sternum instead of a protrusion may develop, as a result of the interference with the entrance of air in respiration. This depression must not be mistaken for congenital funnel breast, which is, of course, present at birth and is usually more marked. As the result of the pull of the diaphragm and intercostal muscles on the soft bones of the thorax and atmospheric pressure, a horizontal depression often develops at the sides

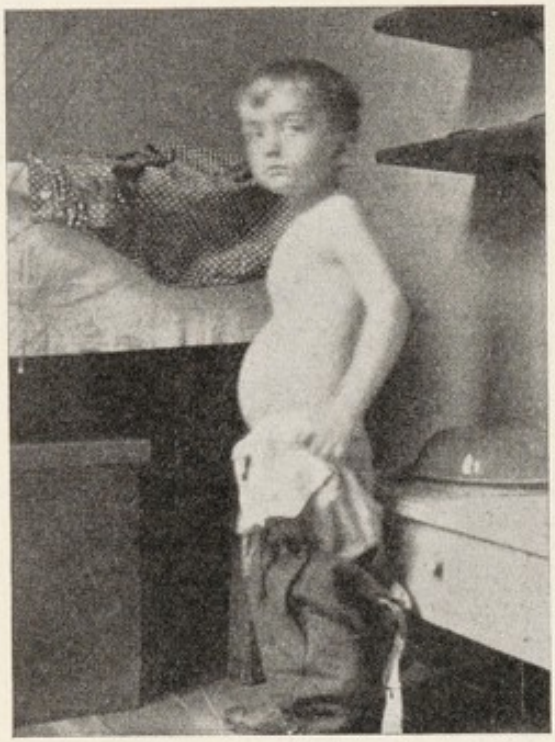

FIG. 52.—Deformity of chest from rickets. Note prominent abdomen.

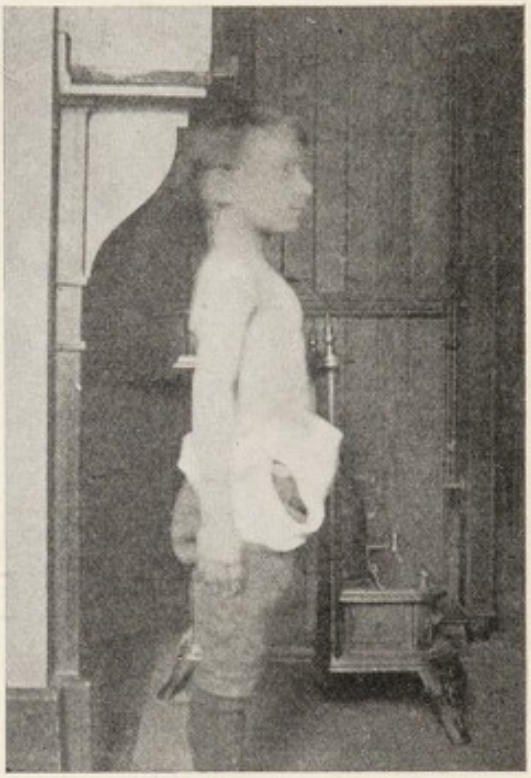

FIG. 53.—"Chicken breast" from rickets.

and front of the chest in the neighborhood of the junction of the middle and lower thirds. This depression is known as Harrison's groove. It is often accompanied by marked bulging of the portion of the thorax below it.

Extremities.—Enlargement at the epiphyseal lines and of the epiphyses of the long bones of the extremities are very common. They occur most often at the wrists and ankles, usually being more marked at the wrists, but may occur in other places. These enlargements may be slight or quite marked. The softening of the bones at the upper ends of the femurs results in the deformity known as coxa vara. The enlargement at the ends of the bones from rickets are sometimes mistaken for those caused by syphilis and scurvy. The enlargement in syphilis is at the lower end of the diaphysis and at the epiphyseal line instead of at the epiphyseal line and in the epiphysis. The enlargement in scurvy is at the lower end of the diaphysis and does not involve the epiphyseal line. It is, moreover, tender, while those due to syphilis and rickets are not tender. As

the result of the pull of the muscles on the softened bones of the extremities and also of pressure from above, if the babies stand or walk, deformities of these bones, which result in bow-legs and knock-knees, develop. Bowing of the legs may be of three varieties: an anterior bowing of the bones of the lower leg, an outward bowing of the bones of either or both the lower legs and thighs and a general outward bowing of the whole extremity. The best way to determine whether there is bowing of the legs or not is to bring the ankles together when the legs are extended with the baby lying on its back. It is important not to mistake the normal, slight outward bowing of the lower portions of the legs which is present in young babies, because of outward rotation at the hips, for bow-legs. This mistake can be avoided by being sure that the legs are not rotated outward. Knock-knees should be looked for in the same way as bow-legs. The knees should just touch, when the ankles are brought together with the legs extended. It is important to remember that knock-knees are not infrequently due to weak muscles, not to bony deformity. The deformity of the long bones of the legs is not infrequently the cause of flatfeet. Permanent shortening of the extremities sometimes results when the rachitic deformities are marked.

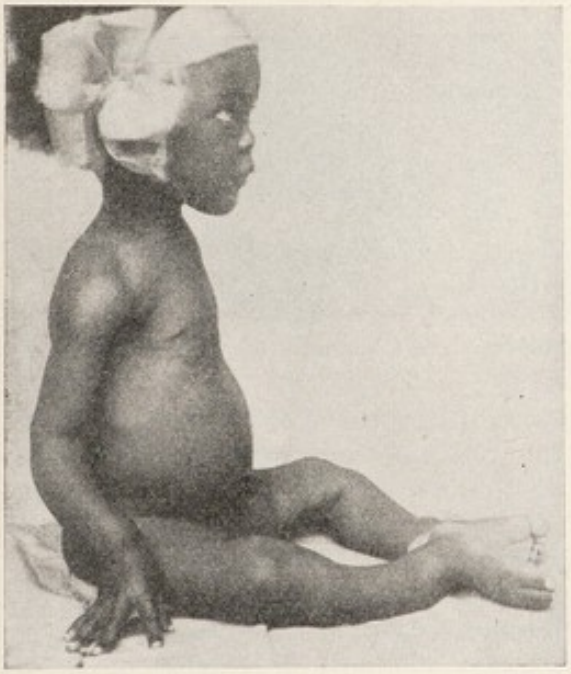

FIG. 54.—Bow-legs and enlarged epiphyses in rickets. Note also deformity of chest and enlargement of abdomen.

FIG. 55.—Curve of weakness in rickets.

Greenstick fractures of the bones of the extremities are not at all uncommon. I have frequently seen several fractures in each of the long bones. When this occurs, the extremities are likely to be tender. Tenderness may sometimes occur in rickets, even when there are no fractures. In many instances, tenderness of the extremities is due to a complicating scurvy. If this is so, it disappears quickly when an anti-scorbutic is given. It does not, of course, when it is due to rickets. This tenderness is sometimes attributed to rheumatism. It ought not be, of course, because rheumatism almost never occurs at this age and there are, moreover, always definite bony evidences of rickets.

MUSCLES.—The muscles, except in the mild cases, are flabby, or underdeveloped from lack of use. They show no essential anatomical changes. One of the most characteristic results of the muscular weakness is shown in the spine. In addition to delay in sitting up, the spine shows,

when babies with rickets sit up, a general kyphosis. This is usually spoken of as the *curve of weakness*. It disappears entirely when the baby lies on its face. There is no limitation of the motions of the spine on passive motion, except in very rare instances in which there is a slight spasm of the spinal muscles. This curve of weakness is occasionally mistaken for the deformity of Pott's disease. The deformity in Pott's disease is localized and angular. The deformity of the curve of weakness disappears when the baby lies on its face; that of Pott's disease does not change.

Weakness of the abdominal muscles is shown by the "*pot belly*." The development of the enlargement of the abdomen is favored by the dilation of the stomach and of the intestines, together with the elongation of the intestines, which are so common in rickets as the result of a coincident disturbance of the digestion. This enlargement of the abdomen, if there are liquid feces in the intestines, is sometimes mistaken for that

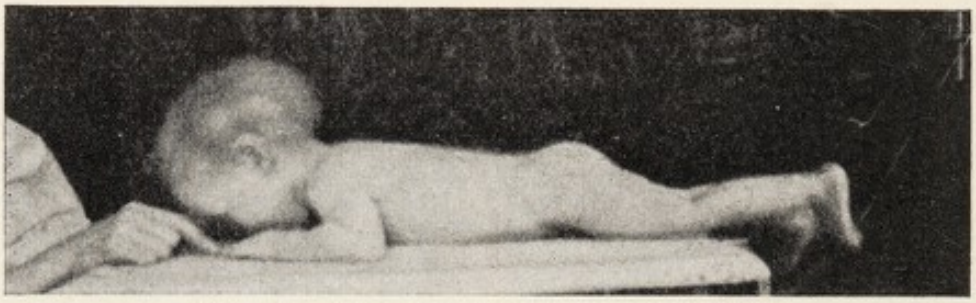

FIG. 56.—Disappearance of curve of weakness.

of tuberculous peritonitis. The differential diagnosis is taken up under tuberculous peritonitis. Herniae, both inguinal and umbilical, are not uncommon in rickets as the result of the weakness of the abdominal muscles. Diastasis of the recti muscles is also not uncommon. This is not an uncommon condition, however, when there is no rickets.

Delay in walking is another sign of muscular weakness. This delay is sometimes attributed to backwardness in mental development or to some form of paralysis. The diagnosis is not difficult, if it is remembered that the mental condition is normal in rickets and abnormal in feeble-mindedness. The reflexes are diminished or absent in infantile paralysis and exaggerated in cerebral paralysis while they are practically normal in rickets. There is, moreover, spasm of the muscles in cerebral paralysis.

NERVOUS SYSTEM.—Poorly nourished nerve tissue is unduly irritable and, therefore, responds too vigorously to minor stimuli. Symptoms of increased nervous irritability, such as fussiness and crying are, therefore, not uncommon. The marked symptoms of nervous irritability which have in the past been attributed to rickets are, however, not manifestations of that disease, but of a complicating or coincident spasmophilia. The relation between rickets and spasmophilia has already been discussed.

SKIN.—The nervous control of the sweat glands is impaired because of the lowered tone of the nervous system. Sweating is, therefore, a common manifestation. It is especially common about the head, and occurs most often at night. There is nothing pathognomonic about the sweating of rickets, however, even of the head. It occurs in all disturbances of nutrition in which the tone of the nervous system is lowered. On account of the maceration of the skin from sweating, it is very susceptible to infection and less resistant to other forms of irritation. Erup-

tions of various sorts are, therefore, very common in rickets, but in no way characteristic or pathognomonic.

Glandular System.—Although it is impossible to prove it, I feel quite confident that enlargement of the tonsils and adenoids is more likely to occur in rachitic than in normal babies. Enlarged tonsils and adenoids are likely to do more harm than in normal babies, because of the diminished resistance to local and general infections in this disease and because, on account of the soft bones and weak muscles, interference with the free entrance of air causes greater deformities of the chest. General enlargement of the peripheral lymph nodes is a common accompaniment. It is not, however, characteristic of rickets and occurs in all disturbances of nutrition in infancy.

Respiratory Tract.—The resistance of the mucous membranes of the respiratory tract, both upper and lower, to infection is much less than under normal conditions. Babies with rickets have, therefore, more than their share of infections of the nose and nasopharynx and of the bronchi. On account of the deformities of the chest and the weakness of the thoracic muscles, bronchitis is a far more serious condition in babies with rickets than in others. Atelectasis develops more easily and bronchitis more often goes on to bronchopneumonia. The prognosis of bronchopneumonia is always grave in babies with marked rickets. In babies with marked deformities of the chest, there is, not infrequently, a strip of partially atelectatic lung along both sides of the vertebral column which gives the signs of partial or complete solidification. These strips are often mistaken for tuberculosis of the lungs. The differential diagnosis is difficult. When these signs are found, however, in combination with a deformity of the chest, they are almost always due to atelectasis rather than to tuberculosis. The tuberculin test will settle the diagnosis.

Circulatory System.—There are no important changes in the heart or great vessels, although in marked cases the cardiac muscle may be affected in the same way as the other muscles. Murmurs of various sorts are not uncommon, but are not due directly to the rickets. They are manifestations of weakness of the cardiac muscle or are connected with anemia.

Secondary anemia is very common in rickets. It is usually of a mild type, but may be severe. It may or may not be associated with leucocytosis or enlargement of the liver and spleen. The anemia, whatever its grade, is, however, not a symptom of rickets, but merely another manifestation of malnutrition. The relation between the enlargement of the liver and spleen and the changes in the blood have been discussed under secondary anemia.

Liver and Spleen.—Enlargement of the liver is not uncommon in rickets. It is due to fatty change. It is not pathognomonic of rickets, but is simply another manifestation of the disturbance of the nutrition. Enlargement of the spleen is also not uncommon in rickets. It may or may not be associated with enlargement of the liver and may or may not be associated with changes in the blood. The pathologic condition is a simple hyperplasia. The enlargement of the spleen is again not pathognomonic of rickets, but simply one of the manifestations of disturbance of nutrition. There is no direct relation between the severity of the rickets and the enlargement of the liver or spleen.

Gastro-enteric Tract.—The digestive powers of the gastro-enteric tract are diminished in rickets, as is also the resistance to infection.

Disturbances of digestion and infections of the tract are, therefore, more common in babies with rickets than in normal babies. There is, however, nothing characteristic about these disturbances. Constipation is not uncommon and is due largely to the weakness of the abdominal and intestinal muscles, perhaps in part to elongation of the intestines.

Diagnosis.—The clinical diagnosis of rickets has been discussed in considering the symptomatology and physical examination. The radiographic changes in the bones are very characteristic and are most useful in confirming the diagnosis of rickets. They often justify a diagnosis of rickets which cannot otherwise be made and also make it possible to determine whether the rachitic process is progressing or healing. In the early stages there is a generalized diminution in the density of the

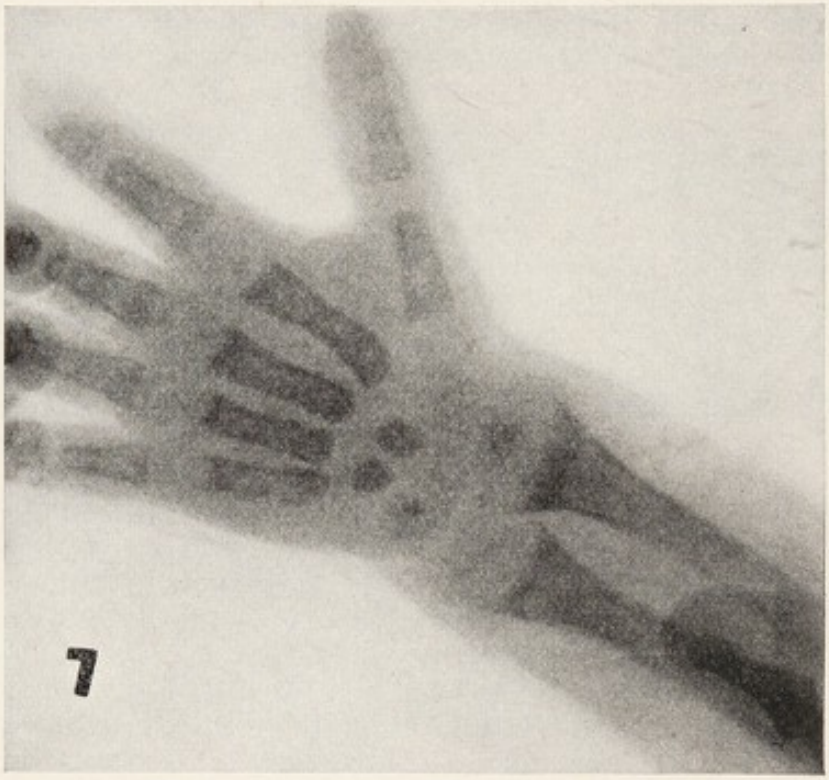

FIG. 57.—Rickets. Shows marked flaring of the ends of the radius and ulna and the distal ends of the metacarpal bones, as well as marked diminution in calcium content.

long bones and the outlines of the ends of the diaphyses are irregular and somewhat indistinct. The calcification of the shafts is also irregular. As the process progresses the shafts of the bones appear more atrophic, the structure is evident in too great detail and irregular calcification is more marked. The ends of the diaphyses become flattened and broadened and the surfaces are no longer sharp and straight, but concave and irregular with a hazy appearance. With further progress, there is marked flaring of the ends of the diaphyses with a ragged, hazy appearance, due to the irregular deposition of calcium. Periosteitis may be present and give an appearance of unevenness and irregularity. The centers of ossification of the epiphyses are indistinct or invisible. These changes are also evident in the centers of the carpal bones. Fractures of the shafts of the bones, complete or partial, are not uncommon and are often multiple.

When healing beings there is an increase in the density of the shafts of the bones, particularly marked at the lower ends of the diaphyses.

The irregularity of the epiphyseal line becomes more clearly defined and the line of calcification soon becomes a band. The centers of ossification in the epiphyses and in the carpal bones become visible or more marked. Evidences of periosteitis, which may not have shown in the florid stage, sometimes become visible. There may be one or more linear bands along the shafts of the bones, parallel to the shafts, and short transverse striations between these longitudinal bands.

After healing has taken place the flaring at the epiphyseal line persists, but the epiphyseal line is regular and a broad band of increased density appears. There is also a thickening of the cortex along the convex side of the curve of the shaft of the bone and the bones show a general increase in density.

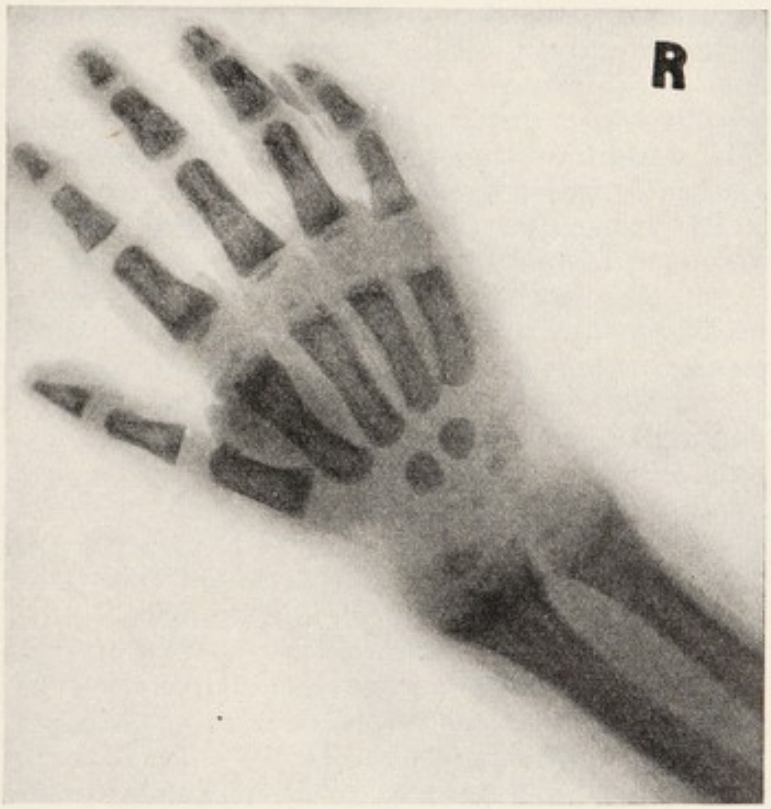

FIG. 58.—Rickets. Same case after one month of treatment. Note marked increase in calcification at ends of radius and ulna and at distal ends of metacarpal bones.

With improvement in the rachitic condition, the phosphorus content of the blood returns to normal. So also does the calcium content, if it has been diminished.

Prognosis.—Rickets is, of course, not of itself a fatal disease. Many babies with rickets die, however, as the result of complications of rickets, who would not otherwise. Complications in the respiratory tract are especially dangerous.

The deformities of rickets almost always disappear with time and growth. The rosary always eventually disappears. Minor deformities of the chest are always outgrown. It is surprising how much even marked deformities of the chest improve as the years go by and the chest increases in size. Traces of marked deformities are, however, likely to persist throughout life. Prominences and depressions of the sternum are especially likely to remain. Most of the deformities of the extremities are also overcome by growth. Slight degrees of knock-knees

are likely to persist, however, and marked bowing of the legs usually does not entirely disappear spontaneously. By proper treatment, however, permanent deformities of the lower extremities can be prevented.

Treatment.—A very important part of the treatment of rickets is the preventive. Babies that are nursed are much less likely to develop rickets and, if they do, it is usually of a mild type. Babies should, therefore, be nursed, if possible. If not, the best substitute is some modification of cow's milk, along the lines already described. Babies should be given all the sunlight possible. It is not necessary to expose the skin directly to the sun's rays, although they are more effective in this way. The scattered diffuse radiations contain a large number of the ultraviolet waves and are, therefore, both preventive and curative. These waves are, moreover, able to penetrate clothing to a certain extent, if it is not too thick or closely woven. White clothes are more permeable than dark. Window glass, however, shuts out the ultraviolet rays. Having the baby in a sun room or putting it in the sunlight which comes through a closed window does not either prevent or cure rickets.

Cod liver oil is a preventive of rickets. Five drops of the pure oil three times daily is usually sufficient in the beginning, but as much as one half a teaspoonful should be given three times a day by the time a baby is three months old. This amount is sufficient during the rest of the first year. Egg yolk is also a preventive against rickets. One egg daily is sufficient. It may be given mixed with the food or may be hard boiled, grated and fed with a spoon.

The development of rickets can also be prevented by exposure to the radiations of the mercury vapor quartz lamp or to those of the carbon arc lamp. It is usually easier, however, to give cod liver oil than to take a baby to a physician's office regularly or to have a lamp in the house.

There is much difference of opinion as to whether it is advisable to give all babies cod liver oil from the time they are a few months old or to wait until some evidences of rickets develop. I am rather inclined to see that they are properly fed and get as much sunlight as possible and not to give cod liver oil until some signs of rickets develop. It is useless to give either cod liver oil or calcium salts to the mother either during pregnancy or lactation. They have no action either in the prevention or cure of rickets. The addition of calcium salts to the infant's food neither prevents nor cures rickets.

Rickets, when it has developed, can be cured either by the administration of cod liver oil or by exposure to the ultraviolet rays. The antirachitic action of egg yolk is so much less than that of cod liver oil that it is better to use cod liver oil after the disease has developed. Sunlight will cure rickets, but not as quickly as exposure to the ultraviolet rays. It is advisable, therefore, to use the ultraviolet rays in addition to sunlight, if possible. One teaspoonful of cod liver oil three times a day is sufficient for a cure. The mercury vapor quartz lamp should be twenty inches from the surface of the body. The baby should be stripped and the whole body exposed. The initial treatment should be two minutes to the front and two minutes to the back of the body, a total of four minutes. The exposure should be increased one minute to the front and one minute to the back, a total of two minutes, each successive treatment until an exposure of fifteen minutes to the back and fifteen minutes to the front, a total of thirty minutes, is given. It is usually advisable to combine the treatment with cod liver oil with that by the ultraviolet rays.

When possible, it is advisable to take Roentgenograms of the wrists before beginning treatment and to check up the results of the treatment by successive pictures. Otherwise, it is rather difficult to know just how much improvement is taking place. While interesting, it is not necessary to follow the calcium and phosphorus content of the blood.

There is still a difference of opinion as to the efficacy of phosphorus in the prevention and cure of rickets. It is certain, however, that rickets can be cured without phosphorus. Phosphorus is, to a certain extent, a dangerous drug and it seems to me, therefore, that, as rickets can be quickly and easily cured without it, it is better not to use it. If it is desired to use it, the best preparation is the phosphorated oil of the pharmacopeia. One minim of this oil contains grain $\frac{1}{115}$ of phosphorus. The dose of phosphorus is between $\frac{1}{100}$ and $\frac{1}{200}$ of a grain three times a day. If phosphorated oil is given, it is advisable to mix it with cod liver oil or some other oil, in order that the variations in the dose may be less, a larger quantity being given.

The treatment of the complications and symptoms of rickets must be along general lines. The development of deformities of the chest can often be prevented by the removal of adenoids and sometimes of tonsils. The development of deformities of the extremities can be prevented to a considerable extent by keeping babies off their feet and, therefore, diminishing the pressure on the bones. Much can also be done to prevent the development of marked deformities by proper orthopedic treatment. If they have developed, they can almost always be overcome by proper orthopedic treatment or operation.

SPASMOPHILIA

Spasmophilia is a condition in which there is a recognizable mechanical and electrical overexcitability of the nervous system which produces a pathologic predisposition to certain peculiar local and general clonic and tonic spasms.

Symptomatology.—Exaggerated mechanical irritability may be shown by tapping the nerves at Erb's points. The most characteristic manifestations are those obtained by tapping the facial and peroneal nerves. The response to tapping the facial nerve is known as Chvostek's sign, or the *facial phenomenon*. When the trunk of the facial nerve is tapped near the ear, there may be a response from any or all of the branches of the nerve. The most common is the drawing of the mouth to that side. The same reaction results when the lower branch of the facial is tapped in the cheek. When the upper branch is tapped about half an inch outside of the external canthus of the eye, there is a spasmodic contraction of the eyelids. When the peroneal nerve is tapped just behind the head of the fibula, the foot turns outward and a little upward. These reactions are almost never found in infancy, except in spasmophilia. After infancy their importance diminishes directly with increasing age.

Another sign, presumably due also to increased mechanical irritability, is *Trousseau's sign*. This sign is the development of the typical spasm of the muscles of the hand of tetany, not only on the compressed side, but also on the other. It is elicited by exerting firm pressure with the hand about the upper portion of the upper arm. The compression must be sufficient to obliterate the radial pulse and cause cyanosis of the hand. This procedure is distinctly painful and is, therefore, hardly justifiable when information of equal value can be obtained by simply tapping the nerve trunks.

The exaggerated electrical irritability is shown by changes in the reaction to the galvanic current—*Erb's phenomenon*. A definite contraction law was established many years ago by Thiemich and Mann. The important points to be remembered, however, are that, in infancy, the appearance of cathodal opening contractions under 5 ma. is pathognomonic of spasmophilia and that the appearance of anodal opening contractions with less current than that causing anodal closing contractions is very strong evidence in favor of it. After infancy these peculiarities are of less importance. After five years, certainly, they are usually not indicative of spasmophilia.

There are two characteristic clinical manifestations: tetany, or carpopedal spasm, and laryngismus stridulus. A third manifestation is eclampsia. There is, however, nothing characteristic about the convulsions of spasmophilia and it is impossible, from the convulsions alone, to know whether or not they are manifestations of this disease.

Fig. 59.—Tetany. Characteristic position of hands and feet.

Tetany.—The most characteristic symptom of tetany is muscular rigidity, this rigidity occurring as intermittent paroxysmal contractions. The duration and intensity of the contractions vary, in some cases being long and remittent, in others fleeting and occurring at long intervals. The most typical of these contractions is carpopedal spasm. In these spasms the fingers are flexed at the metacarpophalangeal joints, while the phalanges are extended. The thumb is strongly adducted in the extended position. The wrist is acutely flexed and the hand turned to the ulnar side. The elbow and shoulder joints are not involved in the milder cases. The feet are strongly extended, sometimes in the position of pure equinus, but more often in that of equinovarus. The first phalanges of the toes are strongly flexed, the others extended. The knee and hip joints are usually free. In severe cases these contractions are not infrequently associated with opisthotonos. These spasms are likely to be associated with pain. Incidentally, pain on motion and edema of the wrists and ankles between the paroxysms is not uncommon. The intelligence is always unimpaired, even during the paroxysmal contractions.

The paroxysms of tetany are so characteristic that they ought not be confused with anything else. The *persistent spasm of the new-born*, which is also called *myotonia of the new-born*, is sometimes mistaken for it. This, however, occurs during the first few weeks or months, while tetany seldom occurs before six months. The spasm is constant, never intermittent, involves either the flexors or the extensors, and lasts continuously for days or weeks, while the contractions in tetany are intermittent, characteristic and of short duration. A similar condition of *general muscular hypertonia* sometimes develops in marked disturbances of nutrition in infancy. Its characteristics are the same as those of persistent spasm of the new-born. Tetany and tetanus are sometimes confused. Tetanus is usually shown first by trismus and difficulty in crying and taking food.

When general contractions occur, they are entirely different from the characteristic localized spasms of tetany. Tetanus of the new-born develops within the first two weeks of life, a time at which tetany never occurs. Tetanus in infancy is very unusual and is always secondary to a wound of some sort. Tetany is always accompanied by some of the other evidences of exaggerated mechanical irritability and by changes in the electrical reactions.

Laryngismus Stridulus.—In typical attacks of *laryngospasm*, the baby makes several short inspirations in rapid succession, each one accompanied by a crowing sound. It then stops breathing with the chest in full inspiration. It quickly becomes cyanotic. After it becomes sufficiently narcotized, the spasm relaxes and it begins to breathe again. In rare instances, however, it does not breathe again and dies. Occasionally, an attack terminates in a convulsion. In many instances the crowing sound is made at the beginning of inspiration, but the breath is not held. Slight crowing sounds may be made intermittently for hours, or even for several days. Laryngismus stridulus and *congenital laryngeal stridor* are sometimes confused. In congenital stridor, the crowing sound is heard at or within a few days after birth and is continuous. It is never present at birth in laryngismus stridulus and is always intermittent. In congenital stridor there is no discomfort, no cyanosis and never any cessation of the respiration, while in laryngismus stridulus there is discomfort, cyanosis and cessation of the respiration. Moreover, in laryngismus stridulus there are always other evidences of spasmophilia, which are absent in congenital stridor.

Attacks of laryngismus stridulus are sometimes confused with "*breath-holding.*" This condition is more common in older children than in infants. The holding of the breath is not preceded by inspiratory crowing, but is almost invariably precipitated by crying or anger. There are, moreover, no other manifestations of spasmophilia. It is hardly possible to understand how laryngismus stridulus can be mistaken for either catarrhal or diphtheritic laryngitis, when all the characteristics of these conditions are borne in mind. I have known these mistakes to be made, however. It hardly seems necessary to go into the differential diagnosis.

Eclampsia.—There is nothing whatever characteristic about the convulsions of spasmophilia. It is impossible, therefore, to make a diagnosis between convulsions from this and from other causes. There is always a fair chance, however, that convulsions in infancy are spasmophilic. If they are, some of the evidences of the increased mechanical irritability of the nervous system are almost invariably present. If they are not, the characteristic electrical reactions are always present, so that the diagnosis is always possible.

Many other special manifestations of spasmophilia relating to various organs have been described. It is very doubtful whether these conditions are really manifestations of spasmophilia, as the diagnoses have not been corroborated by changes in the electrical reactions or examinations of the blood.

Pathology.—There are no characteristic pathologic lesions in spasmophilia. Various lesions of the parathyroids have been found in some instances, but it is extremely doubtful that these lesions have any direct connection with the disease.

Etiology.—Spasmophilia very seldom develops during the first six months of life, is most common during the second six months and uncom-

mon after two years. There is very little evidence to show that the conditions which have been attributed to spasmophilia in childhood are really due to this disease. It often occurs in successive children in the same family. There is, however, no evidence to show that it is hereditary and its occurrence in several members of the same family is probably due simply to a common environment and food. It is very uncommon in the breast-fed. None of these things, however, show anything of any real importance as to the etiology of the disease.

It is generally believed that calcium and magnesium salts tend to lower nervous irritability and that sodium and potassium salts tend to increase it. That is, these salts act antagonistically to each other. Reiss has expressed this proposition by the following formula: $\frac{Ca + Mg}{Na + K}$ Theoretically, therefore, spasmophilia might equally well result from a diminution in the calcium and magnesium salts or from an increase in the sodium and potassium salts in the tissues. There is an abundance of evidence to show that the average amount of calcium in the blood in infancy is normally about 10.5 mg. per 100 c.cm. It has been found that evidences of spasmophilia are almost invariably present when the calcium content drops below 7.5 mg., and that these evidences disappear when the calcium content is brought back within normal limits. It has also been found that in these cases with lowered calcium, the sodium of the blood is normal, the potassium slightly increased and the magnesium slightly diminished, while there is a marked variation in the inorganic phosphorus. The variations in the phosphorus are, however, so marked that it seems evident that the phosphorus concentration has no bearing on the etiology of the disease. It seems evident, therefore, that the increased irritability of the nervous system is due to the diminution in the concentration of calcium in the serum, not to variations in that of the other elements.

Unfortunately, however, this explanation apparently does not entirely solve the problem, because there is a considerable amount of clinical and experimental evidence to show that an excess of sodium salts may cause spasmophilia. I do not feel competent to discuss the question of alkalosis, but I do know from personal experience that the administration of sodium salts, either intravenously or by the mouth, is at times followed by the development of the symptoms of spasmophilia.

Because of the fact that tetany develops after parathyroidectomy and because there is an increased elimination of calcium in the urine and feces and a diminution of calcium in the tissues after this operation, it has been suggested that the diminution in the calcium content of the blood in spasmophilia, and hence the condition itself, may be due to an insufficiency of these glands. Various observers have found changes in the parathyroids in spasmophilia. Others, however, have found normal parathyroids in spasmophilia and precisely similar pathological changes in babies that had not had spasmophilia. It is safe to conclude, therefore, that no connection between the parathyroids and infantile spasmophilia has been proved. Furthermore, the administration of parathyroid extract has no effect on the course of the disease.

Clinically, it is a well-known fact that the symptoms of spasmophilia often appear coincidently with or immediately after some infection. The gastric acidity is low in spasmophilia and a low gastric acidity is apparently associated with a diminished calcium concentration of the blood. It has been suggested that the connection between the develop-

ment of the symptoms of spasmophilia and infection is that the infection causes fever, that fever and illness diminish gastric acidity, and that the lowered gastric acidity results in a diminution of the calcium concentration of the blood. The facts that the administration of acids increases the calcium content of the blood and that spasmophilia seldom develops in babies that are taking human milk, with its low buffer value, or in babies that are taking acidified milks make this explanation seem probable.

There is, and has been for many years, much difference of opinion as to the connection between rickets and spasmophilia. It seems apparent now, in the light of the recent findings in the blood in spasmophilia and rickets, that they are independent of each other. It is possible, however, that the same causes may produce in the blood both those changes which cause rickets and those which cause spasmophilia. At present there is nothing to show why they produce one disease in one instance, the other in another and both in a third.

Prognosis.—Death is unusual in spasmophilia. When it occurs, it is almost always as the result of convulsions or laryngismus stridulus. Before as much was known about the condition as there is at present, its course was almost invariably long. Recovery almost always came, however, with warm weather. With the present methods of treatment, the clinical manifestations can easily be relieved in a few days and the blood findings restored to normal within a few weeks.

Treatment.—In the past, the clinical manifestations of spasmophilia were almost invariably promptly relieved, except in the rare instances in which the baby was on the breast, by giving it human milk. I have, for example, known a baby that was having repeated convulsions never to have another after its first feeding of human milk. When it was impossible to get human milk, the symptoms almost always disappeared and always diminished when babies were taken off cow's milk and put on the cereal waters. Sometimes they returned, sometimes they did not, when cow's milk was given again. I never saw any improvement in the symptoms of spasmophilia in past years when babies were given cod liver oil and phosphorus, as most of them were, because there seemed to be nothing else to do. I am, therefore, rather skeptical as to the favorable results which are now attributed to this form of medication. Parathyroid extract is useless.

The calcium salts unquestionably have a favorable action in spasmophilia. Calcium chloride is much more efficient than calcium lactate. The best form to use is dessicated calcium chloride. It should be given in doses of ten grains six or seven times daily. It has been found that ammonium chloride and hydrochloric acid relieve the symptoms of spasmophilia as well as calcium chloride. Gamble has shown that they all have the same acid effect in the body. This consists in a reduction of the plasma bicarbonate, due directly to an increase in the chlorides, and an elevation of the hydrogen-ion concentration of the plasma. Both of these alterations appear to produce an increased ionization of the calcium. In consequence, a physiologically increased concentration of ionized calcium is obtained from the lowered total calcium content. It has been found, however, that calcium chloride and hydrochloric acid cause an increase in the total calcium of the plasma, which ammonium chloride does not. Calcium chloride and hydrochloric acid are, therefore, probably more efficient agents than ammonium chloride. On account of this action of hydrochloric acid, it seems reasonable to suppose

that acidified milk may perhaps be a preventive of spasmophilia. Calcium chloride seems to be the best drug to use, however, because it not only supplies calcium but also increases the ionization of the calcium already present in the blood.

The ultraviolet rays have a most remarkable curative action in spasmophilia. Improvement with their use is almost immediate, as is shown not only by the relief of the symptoms but by an increase in the calcium in the blood. In the cases treated by Hoag, the symptoms of mechanical hyperirritability disappeared in from four to eight days. He found that the total length of exposure necessary to cause an increase of 1 mg. of calcium in 100 c.cm. of serum varied from 17 to 65 minutes, with an average of 41 minutes. The rise of calcium was proportionately more rapid in those patients who had the lowest concentration in the serum at the beginning of the treatment. He used an all-mercury electrode, quartz-encased burner, using four amperes on a 110-volt direct circuit. The lamp was placed 50 cm. from the surface of the body. The initial treatment was two minutes to the front and two minutes to the back of the body, and this was increased one minute to the front and one minute to the back daily. The average number of days under treatment was thirteen.

To sum up, the development of spasmophilia can, in most instances, be prevented by breast feeding and direct exposure to sunlight. It is very probable that it may be prevented by the addition of hydrochloric acid to artificial foods. It can be cured quickly by the administration of calcium chloride and even more quickly by exposure to the ultraviolet rays.

ACID INTOXICATION

(ACIDOSIS)

True acid intoxication is a very rare condition. The diagnosis of acidosis is often made clinically on insufficient grounds and is almost invariably wrong. The symptoms attributed to acid intoxication are usually due to something else. Not every child that vomits, has a sweet breath or acetone bodies in the urine has acidosis. The only pathognomonic symptom is hyperpnea.

The reaction of the blood plasma and of the tissues of the body is slightly alkaline. The alkalinity is due principally to bicarbonate of sodium. The alkaline reaction is maintained at a constant level even under adverse conditions. Normally, the acids which are being constantly formed in the body are removed from the body in the carbon dioxide given off by the lungs, in the acids excreted in the urine and by combination with ammonia formed from urea, the product being also excreted in the urine. Acidosis results only when the eliminating mechanism is overcome.

Carbon dioxide is formed as the result of metabolic activity. When an increased amount is formed, as during exercise, it stimulates the respiratory center and the excess is removed by deep and rapid breathing, that is, there is an increase in the depth and rapidity of the respiration. The acetone bodies—beta-oxybutyric and diacetic acids—neutralize part of the bicarbonate of soda and set free carbon dioxide. Other acids, which may be ingested, formed in the body or not eliminated by the kidneys, act in the same way. The carbon dioxide which is formed is eliminated through the lungs. The elimination in this way is, however, not sufficient to neutralize a large excess of acid. Consequently, there is a diminution

in the amount of bicarbonate of soda in the blood plasma. It is, therefore, not able to carry as much carbon dioxide and hyperpnea persists. The air leaving the lungs contains less carbon dioxide on this account and serves as a guide to the amount of acidosis. The carbon dioxide tension of the alveolar air is normally between 40 and 45 mm. Thirty to thirty-five means a mild acidosis and twenty or less a severe acidosis.

When the increased rate and depth of the respiration is not sufficient to prevent a diminution in the bicarbonate of soda of the plasma, ammonia is formed from the tissues of the body, which combines with the acids and protects the bicarbonate of soda of the plasma. The ammonia salts which are formed are eliminated by the kidneys.

The alkaline phosphates in the plasma are excreted in the form of acid phosphates by the kidneys. As the result of the chemical change which takes place, there is an increase in the bicarbonate of soda in the blood. The acidity of the urine increases.

Acid intoxication develops when the equilibrium between the production of acids and the powers of elimination is disturbed. This may be the result of an overwhelmingly rapid production of acids or due to the inability of the kidneys to excrete a normal amount of acid. In diabetes, the excessive amount of acid is due to the inability of the organism to break down carbohydrates and thus prevent the formation of acids of the acetone group from fat. The inability of the kidneys to eliminate properly may be the result of disease of the kidney tissue or because sufficient blood is not brought to them.

Symptomatology and Diagnosis.—The only pathognomonic symptom of acidosis is a peculiar type of dyspnea or, rather, hyperpnea, without cyanosis. The peculiarities of this type of respiration are that the rate of the respiration is increased, and that both inspiration and expiration are prolonged, the normal relation between the two being maintained. Flushing of the cheeks, cherry red lips and a white line about the mouth may be either present or absent. None of these symptoms are pathognomonic and their absence does not militate in any way against acid intoxication. When acid intoxication is due to the acetone bodies, the breath not infrequently has a peculiar aromatic odor. This odor is, however, not always present and is decidedly difficult to recognize. Acid intoxication is not infrequently associated with vomiting. Vomiting occurs in many other conditions, however, is not infrequently absent in acid intoxication and, even when associated with it, is not necessarily a manifestation of the intoxication. Diarrhea often precedes or is associated with acid intoxication, especially in infancy. In such cases, however, the diarrhea is not a symptom of the acid intoxication but the acid intoxication is a result of the diarrhea.

The urine is almost always diminished in amount. This diminution is usually the result of vomiting or of the diminished ingestion of liquids. It may be the result of diarrhea. In certain instances, however, the diminution in the amount of urine, which is a sign of impaired elimination through the kidneys, may be the cause of the acid intoxication. The acidity of the urine is almost always increased. This increase in acidity is not pathognomonic, however, as it always occurs with vomiting and in febrile conditions. The presence of acetone bodies in the urine does not justify a diagnosis of acid intoxication. Under normal conditions, the urine contains a minute amount of acetone. The acetone bodies appear in the urine in children as the result of starvation by the third or fourth day. Considerable quantities of acetone and diacetic acid

are regularly found in the urine in a large number of diseases accompanied by high fever both during the course of the disease and during convalescence. They are found so commonly that their presence cannot be considered as of importance unless other evidences of acidosis are present. When the amount of these bodies is very large, however, additional evidences of acidosis should be sought for. It must also never be forgotten that acid intoxication is not necessarily associated with an overproduction of the acetone bodies.

In the test for acetone a crystal of sodium nitroprusside is added to urine in a test tube. The urine is then made strongly alkaline with sodium hydrate. If acetone is present, the color of the solution changes to purple when glacial acetic acid is added. The purple color is often more marked in the foam. In the test for diacetic acid a strong aqueous solution of ferric chloride is added to urine in a test tube. A Burgundy red color shows the presence of diacetic acid. If the reaction takes place after the urine has been previously boiled, it is not due to diacetic acid. If the change in color is very marked, beta-oxybutyric acid is probably present.

The presence of acetone in the breath can be determined by blowing the expired air through Scott-Wilson's reagent, which is a mixture of ten grains of mercuric cyanide and one hundred and eighty grains of sodium hydroxide in 1200 c.cm. of water. If acetone is present, a white cloud is formed. The maximum density is reached in five minutes. The greater the amount of acetone, the larger is the cloud. If the child is conscious, the air may be obtained by having it blow up a rubber bag. If it is unconscious, it must be made to breathe the air in and out of a bag by using a mask of some sort. It must be remembered, however, that this test simply shows whether there is or is not acetone in the breath. Even if there is a high acetone in the breath, it does not necessarily mean that there is a low alkali reserve, that is, acidosis.

There are several varieties of acidosis. The most common in childhood is that due to the excessive formation of the acetone bodies—beta-oxybutyric and diacetic acids and acetone. The acetone probably does no harm, the others do. Acid intoxication from the acetone bodies occurs in diabetes as the result of an incomplete breaking down of the fatty acids because of the faulty metabolism of the carbohydrates. The acid intoxication which occurs in the course of and after acute infectious diseases is due to the acetone bodies. Why it occurs is not known unless, perhaps, there is a disturbance of the metabolism of the fats and carbohydrates as the result of changes due to infection or to toxemia. The acid intoxication which occurs after operations is also due to the acetone bodies. Why it occurs is not known. Recurrent vomiting is often associated with the presence of acetone bodies in the urine and breath. In most instances they are probably not the cause, but one of the results of the cause of the vomiting. The presence of the acetone bodies in the urine before the onset of vomiting, as sometimes happens, and their appearance in large amounts soon after the onset of vomiting, before their appearance can be accounted for by starvation, suggest, however, that in some cases there may be a more intimate relation between them and the attacks.

The acidosis of renal origin is due to the inability of the kidneys to excrete acids, especially acid phosphate. The result of this inability is an accumulation of acids in the body, even though there is no increased production of acids. In such cases there are no acetone bodies in the urine and the ammonia excretion may be normal or even diminished.

The acid intoxication which sometimes occurs in infancy during the course of and after severe diarrhea is not due to an excessive production of the acetone bodies, but to failure of the kidneys to eliminate acids. The failure of the kidneys is not due to disease of the kidneys, but to functional inability because of the loss of fluid from the body through the intestines and a diminution in the amount of blood brought to them, because the blood volume and flow are diminished.

Acidosis may be due occasionally to lactic acid which is produced as the result of convulsions, asphyxia and phosphorus poisoning, to unknown acids developed in diseases like pneumonia, and to the failure of the blood to bring carbon dioxide to the lungs in cardio-respiratory diseases because of the poor circulation. Acidosis may sometimes be due to the ingestion of mineral acids or to poisons. It also sometimes develops in the course of burns and in surgical shock. All of these latter causes are, however, of relatively little importance in early life.

There are a number of laboratory procedures by which the diagnosis of acid intoxication can be positively made. They are, however, unfortunately not practicable for the average practitioner. Two of them, however, are not very difficult and can be carried out without much apparatus. The carbon dioxide tension in the alveolar air can be determined approximately by the use of the little apparatus devised by Marriott and sold by Hynson, Westcott and Dunning of Baltimore. This method is not difficult and works well with older children. It is hard to carry out, however, with infants and young children. It is a colorometric method, and the solutions fade quickly. The carbon dioxide tension is normally between 40 and 45 mm. Tensions between 30 and 35 mm. show a mild degree of acidosis and 20 a moderately severe one. Tensions below 20 mean a very serious condition.

The alkali tolerance test of Sellards is also a simple one. I have had no experience with it. The bicarbonate of soda in the blood is diminished in acid intoxication. When alkali is given by the mouth a certain amount is taken up by the blood before any is eliminated in the urine. Normally, fifteen to thirty grains of bicarbonate of soda makes the urine of infants alkaline to litmus, while a drachm will do it in children. Much larger amounts are required in acidosis. An alcoholic solution of cresol purple is said by Howland and Marriott to be a better indicator than litmus paper. When this indicator is added to urine, if the color changes to violet or purple, there is no acidosis. If it remains green, acidosis may or may not be present. Under normal conditions, the administration of a small amount of an alkali by the mouth causes the change to purple in the urine. In acidosis, much larger amounts are required.

The determination of the bicarbonate reserve of the plasma is too complicated for the average practitioner.

Prognosis.—Real acid intoxication is always a serious condition. The conditions which are so often mistaken for it may or may not be serious. The prognosis is somewhat better in those forms which are due to the excessive formation of the acetone bodies than in those which are due to other causes. The outlook is almost hopeless when it is the result of disease of the kidneys. It is not so bad in the type due to dehydration. It must be remembered also that, even if the acidosis is relieved, the child may die of the condition which caused it.

Treatment.—As always, preventive treatment is of the greatest importance. The development of acid intoxication in diabetes can be prevented, at any rate for a long time, by proper regulation of the diet

and the administration of insulin. That which develops after operations can usually be prevented by giving glucose before and after the operation. It is often advisable to give a glucose enema immediately after operation. The type which develops in the course of severe diarrheas can be prevented by proper feeding and care to prevent diarrhea and by preventing dehydration during diarrhea. That which develops during acute infections can be prevented by greater care in the avoidance of infection.

The treatment of acid intoxication in the course of diabetes is discussed in the treatment of that disease. That of acid intoxication due to an excessive formation of the acetone bodies in other conditions than diabetes is along the same lines. Glucose, or dextrose, should be given frequently both by the mouth and rectum. Either a 5% or a 10% solution may be given by the mouth. 5% is strong enough by the rectum. The glucose may be given in combination with orange juice or, in mild cases, orange juice may be given alone. It must not be forgotten, however, that large quantities of orange juice may cause both vomiting and diarrhea. The orange juice not only furnishes sugar, but also acts as an alkali. The glucose solution may be given by the rectum either as an enema or by the drip method. If the child is not too restless, the latter method is preferable. It is seldom that more than ten drops a minute can be absorbed, often not more than five. A 5% solution may be used subcutaneously and a 10% solution intravenously. If there is no improvement when the glucose solution is given intravenously and the urine shows sugar, insulin, in the proportion of one unit of insulin to three grams of glucose, should be given subcutaneously at the same time.

When acid intoxication occurs in the course of severe diarrhea as the result of dehydration, it is useless to give glucose. The object of the treatment is to restore the blood volume and replace the water lost by the tissues, so that the kidneys can resume their functions. Water should be pushed by mouth. It may be given through a tube if necessary. It is useless to give it by rectum when there is a severe diarrhea, because it will not be retained. Physiologic salt solution should be given subcutaneously or intraperitoneally. It may also be given intravenously. From two to eight ounces should be given subcutaneously and from four to eight ounces intraperitoneally, according to the size and age of the patient. The subcutaneous injections should be repeated as soon as they are absorbed. Salt solution should not be given again intraperitoneally until at least twelve or, probably better, twenty-four hours have elapsed. Improvement begins coincidently with an increase in the amount of urine.

When acid intoxication is due to disease of the kidneys, little can be done outside of the measures ordinarily used to increase the secretion of urine. These are not likely to be of much use. When due to other causes, there is not much to do except to give large amounts of water and possibly bicarbonate of soda, with the object of restoring the alkali reserve. If given by the mouth, it may be given in orange juice or grape juice. The concentration of the solution ought not be more than one to thirty or one to sixty. When given by the rectum, it may be given either by enema or by the drip method, as in the case of glucose. It is difficult to get in enough soda by these methods to control the symptoms in severe cases. It should then be given intravenously. The amount to be given in this way should theoretically depend on how much the reserve has been depleted, as shown by the carbon dioxide tension

of the alveolar air. Practically, the top limit at a single injection is from fifteen to twenty c.cm. of a 4% solution per kilogram of body weight. This may be repeated several times daily. It is doubtful, however, if it is advisable to use bicarbonate of soda when the intoxication is due either to the acetone bodies or to dehydration. It must always be remembered, moreover, that an excess of bicarbonate of soda, even by the mouth, may do great harm.

SECTION VI

DISEASES OF THE MOUTH

CATARRHAL STOMATITIS

Catarrhal stomatitis may be due to traumatism, which may be either mechanical or chemical. It may also be the result of bacterial infection. It is not unlikely, moreover, that a secondary bacterial infection occurs when the primary cause is some form of traumatism. The catarrhal stomatitis which is so common in the course of other diseases is presumably also due to bacterial infection. In babies the injury which leads to infection is not infrequently inflicted in washing or cleaning the mouth. Babies mouths should not be washed. A swallow or two of water after feeding is all that is necessary.

Symptomatology.—The appearances in the mouth vary according to the severity of the inflammation in the individual instance. Most of the mouth is usually involved. The mucous membrane is reddened and injected. It bleeds easily when touched. In all but the mildest cases there is some swelling of the mucous membrane. This is usually most marked over the gums and about the teeth, if there are any. Occasionally there are superficial ulcerations. The lips are sometimes swollen, but usually not. The mouth feels hot both subjectively and objectively. There is an increased secretion of saliva. In severe cases there is much drooling. The skin about the mouth and of the chin may be irritated by it. The tongue is coated. The edges are often reddened and the papillae distinct. The breath is usually not particularly foul. There is seldom any enlargement of the lymph nodes in the neck. There is marked anorexia. In the mild cases there is no rise in temperature. There may be a moderate elevation in the more severe cases. The constitutional symptoms are usually not marked, but may be. They are usually chiefly digestive. There is almost always some loss of weight, which is sometimes considerable, as the result of lack of food. The course is usually less than a week, but may be considerably longer.

Treatment.—The mouth, gums and teeth must be kept clean. Older children can swash a mouth wash about very well and keep the mouth clean in this way. The mouths of babies and young children must be washed. This should be done every few hours with a swab of absorbent cotton on a stick or a piece of cotton or soft cloth on the finger. Great care must be taken not to injure the mucous membrane further and in this way do harm. A good mouth wash is a saturated solution of boric acid. Another is a mixture of thirty grains of borax and two drachms of glycerine with water enough to make four ounces. Any of the simple alkaline washes are satisfactory. One of the best is the officinal liquor antisepticus alkalinus. Another good mouth wash is that made by adding two teaspoonfuls of tincture of myrrh to a cup of water. It is important not to use too strong solutions of any kind. If ulcers have formed they should be touched with a nitrate of silver stick.

Ice to suck often affords much comfort. The food should be liquid. It is usually taken best when cold. If babies and young children will

not take enough food, it should be given through a tube. There are no drugs which, taken internally, have any effect on the catarrhal process in the mouth. It is important to look after the general condition as far as possible, because catarrhal stomatitis not only occurs most often in children who are below par, but, of itself, also materially disturbs their nutrition.

APHTHOUS STOMATITIS

(HERPETIC STOMATITIS)

The term "herpetic stomatitis" ought not be applied to this condition, but should be limited to that very rare form of stomatitis in which the primary lesion is a herpes of the mucous membrane of the mouth. The two forms are indistinguishable, however, after ulceration has taken place. There is much difference of opinion as to the relationship between aphthous stomatitis and impetigo contagiosa. Whatever this may be, they are both due to local bacterial infection with either staphylococci or streptococci. In aphthous stomatitis a streptococcus is probably more often the cause. Aphthous stomatitis seldom occurs before the teeth have erupted. It is most common in children under ten years of age. It is more common among children of the hospital class than among those of the well-to-do. It is probably somewhat contagious and not infrequently occurs in small epidemics.

Symptomatology.—The appearance of the typical picture in the mouth is usually preceded by one or two days of slight indisposition, during which there may be evidences of a slight catarrhal stomatitis. The first characteristic manifestation is the appearance of slightly raised red spots, varying in diameter from one eighth to one quarter of an inch, on the mucous membrane of the mouth. They are most numerous along the edges and at the tip of the tongue, on the inner surface of the lips and on the outer surface of the gums. Within a day or two these reddish spots become yellowish, as the result of a deposit of fibrin in the mucous membrane. There is a bright red areola about the spots. The epithelial layer over the fibrinous deposit becomes softened and desquamates, leaving a superficial ulcer. The red areola is rather more marked at this time. Healing occurs from the periphery toward the center. The whole process usually takes from one week to ten days. There may be only a few of these characteristic areas, or many. They may, at times, appear in crops. Not infrequently the spots coalesce. There is a catarrhal inflammation of the mucous membrane of the rest of the mouth, and the edges of the gums are often soft and bleed easily. There is always profuse salivation, which is likely to cause irritation of the skin about the mouth and of the chin. The odor of the breath is quite foul, but not as foul as in ulcerative stomatitis. The temperature is always elevated. It usually varies between 100° F. and 102° F., but not infrequently goes as high as 103° F. and 104° F. There is almost always some swelling of the submaxillary lymph nodes and sometimes of those in the neck. There is always marked anorexia and not infrequently considerable disturbance of the nutrition. It is usually not a very serious disease. I have never seen a child die of aphthous stomatitis, but I have seen a number that were very dangerously ill.

Treatment.—The treatment is essentially the same as that of catarrhal stomatitis. A 1-1000 solution of permanganate of potash is sometimes useful. It is usually not necessary in mild cases to touch the ulcers with the nitrate of silver stick. This should always be done, however, in the

more serious cases. It was formerly believed that chlorate of potash had a specific action in this condition and that it not only acted locally when used as a mouth wash, but that it was excreted into the mouth in the saliva and thus exerted a continuous action. It has been conclusively proved, however, that chlorate of potash has no action whatever in the mouth. It is not advisable to use it, therefore, because there is a possibility that it may cause irritation of the kidneys.

ULCERATIVE AND GANGRENOUS STOMATITIS AND NOMA

These three conditions are taken up together because the present belief is that they are all caused by Vincent's bacillus. This is a fusiform bacillus which also often appears in a degenerated form as a spirillum. Both forms are usually seen together, although the fusiform bacillus may be the only one present. The spirillum is never seen alone. It is very easy to miss this organism, however, unless the smear is taken from deep down in the ulcerated area. It is cultivated only with great difficulty.

Ulcerative stomatitis is not infrequently the result of mineral poisoning, most often of mercury. It also develops in the course of severe cases of scurvy. It is probable that scurvy and the mineral poisons are not the real cause of the ulcerative condition, but merely produce such changes in the mucous membrane that infection with Vincent's bacillus becomes possible. Gangrenous stomatitis may follow ulcerative stomatitis or the process may be gangrenous from the first. Noma may develop in connection with ulcerative or gangrenous stomatitis, or may be the first manifestation of infection. Disturbance of the nutrition from any cause, especially if a catarrhal inflammation of the mucous membrane of the mouth is associated with it, predisposes to the development of both gangrenous stomatitis and noma. Measles is more often followed by these conditions than any other disease.

Symptomatology.—Ulcerative and gangrenous stomatitis never develop until after the teeth have erupted. In *ulcerative stomatitis* the gums are much swollen, reddish purple in color and bleed easily. A distinct yellow line develops at the junction of the gums and the teeth. The ulcerative process extends downward about the teeth. These become loosened and not infrequently fall out. If the periosteum and bone of the jaw are also involved, abscesses may be formed and parts of the jaws destroyed. There is, of course, a profuse, very foul, somewhat purulent discharge from the mouth. The odor from the mouth is very foul. The temperature is usually only moderate. The constitutional symptoms vary materially, depending upon the amount of absorption and also upon how much interference there is with the taking of food. The lymph nodes in the neck are almost always swollen. The tendency in ulcerative stomatitis is toward recovery. The course is usually from one to three weeks. It may, however, be much longer and sometimes ends in death.

In *gangrenous stomatitis* the early appearances are not very different from those in ulcerative stomatitis. Sometimes the typical ulcerative process becomes gangrenous. More often, however, the gangrenous process begins at the junction of the gums and teeth without there having been any previous ulceration. The gangrenous process usually extends rapidly. The teeth become loosened and drop out. Death usually occurs before there has been much involvement of the periosteum and

bones. There is a profuse, very foul discharge. The odor of the breath is dreadful. The temperature is usually not much elevated. Pain is usually not very marked. Toxemia quickly develops with prostration and often stupor. This is so marked that the children usually do not suffer much. Death usually occurs in a week or ten days, but recovery is possible.

Noma is really a variety of gangrenous stomatitis. The same name is applied to a similar gangrenous condition in other parts of the body. It most often begins in one cheek as an ulceration with a grayish necrotic covering. The whole side of the face is swollen and has a waxy appearance. A central area of infiltration can be felt extending somewhat beyond the edges of the ulcer. The odor of the breath is foul, even at this time. The indurated area spreads rapidly and a reddened area appears on the outside of the cheek corresponding to the ulcerated area within. A slough is formed, which soon results in perforation of the cheek, although this does not always occur. The gangrenous process extends very rapidly, so that within a few days a part or the whole of the cheek may have sloughed away. The gangrenous process may begin at the corner of the mouth or even about the nose. The gums are almost always involved. The process is usually limited to one cheek. The discharge is not as marked as would be expected. It is often bloodstained. The odor from the discharge and from the mouth is horrible. Words can hardly describe it.

The temperature is at first not very high, but becomes higher after a few days. There is usually not much suffering, but profound prostration soon develops from toxemia. There may be vomiting and diarrhea. Bronchopneumonia almost always develops as the child becomes weaker and is likely to be the cause of death, unless the child dies sooner of sepsis.

Death usually occurs in a week or ten days, although the fatal termination is sometimes delayed much longer. In rare instances, the disease terminates spontaneously. In other instances recovery occurs after surgical intervention. The outlook is probably somewhat better now than in the past with a treatment which is directly destructive to the causative organism.

Treatment.—All of these conditions are almost entirely preventable by proper care and treatment. There is no excuse for the development of lesions of the mucous membrane of the mouth and gums from mineral poisons, when they are given medicinally. Scurvy should be recognized and cured long before the appearance of ulcerative lesions in the mouth. Proper care of the mouth during serious illnesses and during the course of the eruptive diseases ought to prevent the development of those mild catarrhal lesions which open the doors for infection with Vincent's bacillus. Babies that are properly fed and cared for will never get into a condition in which they will develop gangrenous stomatitis or noma.

It goes without saying that in all of these conditions the mouth should be kept as clean as possible with mild, non-irritating, alkaline washes. Chlorate of potash, whether used as a mouth wash or taken internally, has no curative action. A 1-1000 solution of permanganate of potassium is of some advantage in the milder ulcerative form. Nitrate of silver is of little use in these severe types. Tincture of iodine is supposed to destroy Vincent's bacillus and a 5% or 10% aqueous solution of chromic acid is also said to have a specific action. One or both of these should, therefore, be applied thoroughly and frequently in all of these conditions.

It was believed up to a few years ago that in every case of noma not only the affected area, but the tissues for a short distance beyond this area should be cut out, and the wound treated with the actual cautery. The present feeling is that less radical surgical procedures are advisable and that they should be combined with treatment with tincture of iodine and chromic acid. Local applications of arsphenamin or neoarsphenamin have been recommended. It is also recommended to inject them as in the treatment of syphilis.

I have had no personal experience with gangrenous stomatitis and noma since the introduction of the new methods of local treatment and of the new preparations of arsenic. I have never seen a case recover which was treated by the older methods or by surgery.

THRUSH

Thrush is a form of stomatitis produced by the growth of a fungus. It is generally believed that this fungus is the oidium albicans, but there is some doubt as to whether it really is or not.

The thrush fungus is widely distributed in nature, the spores being in air and dust. The fungus grows in sweet or acid substances and, therefore, may be present in the food. Contagion is, therefore, not necessary, although it is directly or indirectly transmitted from one infant to another in the majority of instances. It does not attach itself readily to a healthy mucous membrane. It finds it difficult to develop even on a wounded mucous membrane, if an infant is vigorous and healthy. Catarrhal stomatitis is a predisposing cause. Injury to the mucous membrane also opens the door to infection, while a debilitated condition from any cause predisposes to it. It very seldom occurs after infancy.

Symptomatology.—The fungus usually develops first upon the edges of the tongue or on the cheeks, sometimes on the gums or on the inside of the lips. Unless checked, it usually spreads rapidly. It appears first as discrete white spots the size of a pinhead or less. These spots increase in size and coalesce. Where they have coalesced, the surface looks rough. It does not resemble a pseudomembrane. It is slightly raised above the surface. It is rather hard to rub off. When rubbed off, it does not leave a bleeding surface. It is likely to come off of itself in one place while it spreads in others.

There is a moderate increase in the amount of saliva, but not nearly as much as in other forms of stomatitis. Thrush apparently causes but little discomfort and, as a rule, does not interfere much with the taking of food. There is ordinarily no fever and usually not much constitutional disturbance. It is said that thrush may extend backward and downward into the glottis. I have never seen this happen.

The duration of the disease, when untreated, is very indefinite. It may last but a few days or may run on for weeks. The duration depends very largely on the general condition of the baby.

Diagnosis.—The only thing with which thrush is likely to be confused, or the only thing which is likely to be mistaken for thrush, is curds of milk temporarily adherent to the mucous membrane. The diagnosis is easy. The curds of milk are easily wiped off, while it is difficult to wipe off the deposits of thrush. Even when the deposit of thrush is large, it ought not be confused with membrane. The surface of thrush is rough, while that of membrane is smooth. There is always some inflammation about a membrane, while there is none about thrush. The thrush

can be wiped off and does not leave a raw surface; membrane is dislodged with great difficulty and leaves a raw surface. There are fever, symptoms of constitutional disturbance, foul breath and enlargement of the cervical or submaxillary lymph nodes with membrane. None of these symptoms are present with thrush. If there is any question, the diagnosis of thrush is easily made by putting a little of the growth under the microscope, where the characteristic mycelia and spores are easily seen.

Treatment.—In spite of the wide distribution of the thrush fungus, its development in the mouth can be easily prevented by leaving the mouth alone, by cleanliness and by proper feeding and care. In the vast majority of instances, thrush develops because of lack of care of the nipples. It should be regarded as a reflection on whoever takes care of the baby, whether it is in its own home or in an institution.

If thrush has developed and the baby is on the breast, the mother's nipples should be washed with a saturated solution of boric acid before and after each nursing. If the baby is on the bottle, the nipples and bottles should be boiled after each nursing and the nipples should be kept in a saturated solution of boric acid.

After each feeding the mouth should be thoroughly cleaned with a saturated solution of boric acid, either with a swab of cotton on a stick or with the finger wrapped with cotton or soft cloth. There is much difference of opinion as to whether the spots should be rubbed off or not. In my experience, recovery occurs much more quickly when they are thoroughly removed each time. There is no other solution which works as well as boric acid. Under no conditions should a mouth wash prepared with syrup be used, as the fungus grows in syrups.

It is also very important to regulate the feeding and the infant's life so that its nutrition will be improved. If it will not suck, it should be fed with a spoon or a medicine dropper. It is seldom necessary to use a tube.

DISTURBANCES OF DENTITION

Difficult Dentition.—In spite of all that has been said and written regarding the infrequency of disturbances in infancy as the result of dentition, the tendency of the laity and also of many physicians, who ought to know better, is still to attribute to dentition all sorts of symptoms and diseases which have no relation to dentition, except that they occur at the same time. It is evident how foolish this is, when it is remembered that dentition is a process which begins *in utero* and is not completed until the eruption of the "wisdom teeth," which seldom occurs before the seventeenth year. Nevertheless, babies and children are well most of the time. No one attributes miscarriages and congenital deformities or repeated colds and indigestion in late childhood to dentition. It would seem as reasonable to do this as to attribute many of the ills of infancy to it. The diseases and disturbances which are laid to dentition are almost invariably due to something else. It is a safe rule to follow not to think of dentition as a cause, unless every other possible cause has been ruled out, and then to feel quite certain, not that the cause is dentition, but that it has not been found. Nevertheless, in rare instances certain symptoms are apparently connected with dentition. In my experience these never occur except when the gums are much reddened and swollen over a tooth almost ready to erupt. I have seen occasionally such minor manifestations as restlessness, discomfort, nausea and slight elevation of temperature, and very rarely more marked

symptoms, such as vomiting, diarrhea and high fever, which I have believed to be due to dentition. The reason that I have believed them to be due to it is because they have ceased immediately when a tooth has erupted. In a few instances, moreover, I have seen the same set of symptoms in an individual baby each time that the gums were distended by a tooth. I have occasionally seen similar symptoms in older children, which persisted until a tooth erupted.

If the gum is much swollen and reddened over a tooth and there is no other evident cause for the symptoms which the baby has, it is advisable to make an incision in the gum down to the tooth. The incision should be as long as the tooth is wide. If it is a molar tooth, two diagonal incisions, crossing each other, should be made. If the incision of the gum is justified, the tooth will immediately pop into sight. It seems to me far more rational to cut the gum with a clean knife than to attempt to rub it through with a finger or a thimble both of which are almost always more or less dirty. No one would think of using a finger or a thimble anywhere else than in the mouth in place of a knife and I see no reason why they should be used in the mouth. If the incision has been necessary, the formation of a cicatrix which might interfere with the eruption of the tooth later need not be feared. It seems to me that it need not be feared, even when an unnecessary incision has been made, because, under normal conditions, a tooth does not push its way up through the gum, but the gum is absorbed above it. It is unreasonable to think that rubbing paregoric on the gums will have any effect on any discomfort which may come from them. It is doubtful if the paregoric can be absorbed in sufficient amounts to effect the discomfort and, moreover, it is certain to be washed away by the saliva before it has time to act. If enough is rubbed on, it will be swallowed and absorbed lower down and will, therefore, quiet the baby.

Imperfect Teeth.—The nutrition of the teeth and consequently their normal development may be disturbed not only during infancy, but also *in utero*. When the disturbance occurs *in utero* it is usually due to syphilis, but may sometimes be the result of disturbances of the mother's nutrition. No one knows what the disturbance of nutrition in the mother is which causes these disturbances in the nutrition of the teeth of the fetus. It hardly seems reasonable to believe that it is due to a lack of calcium in the mother's diet. It is barely possible, however, that, in certain instances, the mother's calcium metabolism may be abnormal and that that of the baby may be disturbed at the same time. What we know regarding the connection between the calcification of the bones and the intake of calcium in infancy suggests that the difficulty is not due to lack of calcium. It is evident, therefore, that it is irrational to give calcium salts to the mother during pregnancy and that nothing is really known as to how a woman should be fed or how she should live, while she is pregnant, in order to prevent disturbances in the formation of her infant's teeth. All that a woman can do is to lead as normal a life in every way and to keep herself in as good condition as possible during pregnancy.

Disturbances in the nutrition and development of the teeth during infancy, judging from what we know of the amount of calcium in babies' foods and the effect of a deficiency of calcium on the development of other bones, are not due to a lack of calcium. Arguing from rickets, in which the changes in the bones are due to a disturbance of the absorption and metabolism of calcium, it is probable that the disturbances in the nutri-

tion of the teeth which occur in connection with rickets are due to the same cause. It is evident that those measures which prevent the development of rickets will prevent disturbances of nutrition in the teeth from the same cause. Much has been said recently about the relation between disturbances in the nutrition and development of the teeth and a deficiency of vitamins, the chief argument in favor of these disturbances being due to a deficiency of vitamins being the changes which develop in animals when they are deprived of the antiscorbutic vitamin. There is no proof, however, that such changes occur in the teeth in scurvy in infancy or that the changes which occur in the bones of animals are the same as those which occur in disturbed development of the teeth.

The development of the teeth is apparently seldom affected by disturbances of the nutrition which result in emaciation or by starvation. The nutrition of the teeth is, however, not infrequently affected by serious illnesses of any sort occurring during infancy and childhood.

Knowing, as we do, practically nothing as to the real etiology of the disturbances of the nutrition of the teeth and of their development during infancy and childhood, all that can be done is to feed babies and children as well as possible, keep them in good general condition and prevent them from having serious illnesses. There is no justification for giving babies and children calcium as a drug with the idea of improving the teeth, because there is always more calcium in their food than they can utilize. Babies and children that are properly cared for will never lack vitamins. It is unnecessary, therefore, to think of them in connection with the teeth. There are no indications for any special form of diet. As already stated, all that can be done is to keep infants and children well.

Deformities of the Jaw.—Deformities of the jaw are unfortunately not uncommon in childhood. It is difficult to know what proportion of them are due to inheritance, what to adenoids and what to thumb-sucking. In my experience very few of them are due to thumb-sucking and most of them to adenoids. These deformities not infrequently cause marked disturbance of the general nutrition. They may interfere almost as much with respiration as did the adenoids which caused them. When marked, they prevent proper mastication of the food and cause disturbances of the digestion. The disturbances of nutrition which result from oral respiration and indigestion are often considerable.

The treatment consists, of course, in correcting the deformities of the jaws by mechanical means. This correction always takes a long time. It is not as troublesome and does not cause as much nervous disturbance as would be expected. The expense of orthodontic treatment is, however, prohibitive for all but the well-to-do and a strain on most of them. In my opinion the charges which the orthodontists make are excessive and entirely out of proportion to the charges which dentists make for other work and which physicians charge for their services. Unfortunately the opportunities for free treatment of these deformities in Boston are extremely limited. I am inclined to think that they are little, if any, better in other communities.

SECTION VII

DISEASES OF THE GASTRO-ENTERIC TRACT

The classification of the diseases of the gastro-enteric tract which follows is the one which I used last when at the head of the Department of Pediatrics of the Harvard Medical School. It is open to many objections, but on the whole it seems to me to be more satisfactory than any other. At any rate, it serves as a reasonable basis for the consideration of the various diseases and conditions of this tract. It is based, as far as possible, on etiology. Many exceptions may be taken to it, however, even on this basis.

The various malpositions and malformations of the stomach and intestines classed as developmental, as well as pyloric stenosis, are discussed where they more properly belong, under the head of Congenital Malformations. Hernia has been discussed among the diseases of the newborn.

CLASSIFICATION
OF
DISEASES OF THE GASTRO-ENTERIC TRACT

Developmental

- Malpositions
- Malformations

Mechanical

- Contraction of the Stomach
- Dilatation of the Stomach
- Pyloric Stenosis
- Dilatation of the Colon
- Volvulus
- Intussusception
- Hernia
- Fissure of Anus
- Prolapse of Rectum
- Hemorrhoids

New Growths

Traumatic

- Foreign Bodies
- Corrosive Gastritis
- Ulcer of Stomach and Duodenum

Nervous

- Vomiting (Spasm of the Pylorus)
- Diarrhea

Disturbances of Digestion

1. Overfeeding as a whole
2. Overfeeding with various food elements
   a. Fat
   b. Carbohydrates
   c. Protein
   d. Salts
3. Indigestion with Fermentation

Infections

Gastritis
Infectious Diarrhea
a. Dystentery Bacillus
b. Gas Bacillus
c. Other Organisms
Cholera Infantum
Chronic Colitis
Proctitis
Appendicitis
Fistulae

Unclassified

Recurrent Vomiting
Constipation
Incontinence of Feces
Intestinal Parasites

CONTRACTION AND DILATATION OF THE STOMACH

Much less attention is paid to either contraction or dilatation of the stomach than used to be before it was known that the food begins to leave the stomach as soon as it is taken and that there is no fixed relation between the size of the stomach and the amount taken at a feeding. I doubt very much if there is any pathologic evidence proving that contraction of the stomach ever occurs. Nevertheless, clinically there is no doubt that babies that have had only small amounts of food for considerable lengths of time or that have been vomiting constantly and those with marked disturbances of the general nutrition are unable to take and retain as much food at a feeding as they would be expected to take from their age and size.

It is now known that dilatation of the stomach in infancy is almost always the result of organic stenosis or spasm of the pylorus. Nevertheless, dilatation of the stomach does occasionally develop as the result of gross overfeeding, of excessive ferementation in the stomach or as one of the manifestations of the general lowering of the muscular tone in rickets. Dilatation of the stomach may also occasionally occur acutely. Dilatation of the stomach after infancy is extremely uncommon.

Symptomatology.—The only symptom of contraction of the stomach is vomiting after the ingestion of small amounts of food. The main symptom of dilatation of the stomach is also vomiting. There is nothing characteristic about the vomiting in dilatation of the stomach, except when several feedings are retained and then vomited all together. When this happens, it is very important, however, to be certain that the cause of the retention is not obstruction at the pylorus. Associated with the vomiting are likely to be discomfort from distention of the stomach by food or gas, hunger as the result of the vomiting, and constipation, also secondary to the vomiting and the lack of the normal amount of intestinal contents. Malnutrition also develops as the result of an insufficient retention of food.

Physical Signs.—The epigastrium is likely to be distended. Occasionally the outline of the lower border of the stomach is visible. The percussion note may be either flat or tympanitic, according to whether the stomach is filled with fluid or with gas. If the dilated stomach is empty, none of these signs are present. It is very difficult, however, to determine from either inspection or percussion whether the stomach is dilated or not, because the lower border of the stomach may normally reach to the navel and it is almost impossible to separate the percussion

sounds coming from the transverse colon from those coming from the stomach. If there is visible peristalsis it is easier to determine the outline of the stomach. If visible peristalsis is present, however, it signifies that the dilatation of the stomach is secondary to obstruction at the pylorus and a relatively unimportant symptom.

Diagnosis.—When the diagnosis of dilatation of the stomach is not evident from the symptoms and physical signs, it can usually be determined with the stomach tube, either by finding how much there is in the stomach or by determining how much fluid can be introduced into it without much discomfort. The area of the stomach after it is filled is then easily determined by inspection or percussion. It may also be determined with the help of the Roentgen ray after the introduction of an opaque meal.

Prognosis and Treatment.—When dilatation of the stomach is due to obstruction at the pylorus, the treatment consists of that for the relief of the causative condition, and the prognosis depends on whether this condition can or cannot be relieved. When the dilatation is not due to obstruction at the pylorus, the prognosis is almost always good, because the causative condition can almost always be removed. The treatment consists primarily in regulation of the quantity of food given at a time and of the quality of the food, so that it is suited to the individual baby's digestion. When the dilatation is one of the manifestations of rickets, the rickets should, of course, be treated. In the meantime, much relief is often afforded by washing out the stomach once or twice daily just before some feeding time. In my experience, plain water does as well as anything else. Sometimes, however, the addition of a small amount of bicarbonate of soda to it seems to be of advantage.

DILATATION OF THE COLON

Moderate dilatation of the colon in association with dilatation of the small intestine is not uncommon in chronic disturbances of digestion both in infancy and childhood, and in infancy as the result of rickets. Dilatation of the colon may also develop immediately after birth as the result of a congenital defect in the development of the colon. The term "Hirschsprung's Disease" should be limited to this form. It may also develop later in infancy, and occasionally in childhood, as the result of a congenital weakness of the colonic walls. It is much more often due, however, to interference with the emptying of the bowel. This interference may occasionally be due to an increase in the length of the normally relatively long colon, especially at the sigmoid flexure. This results in a bending of the intestine on itself, which mechanically interferes with the passage of the intestinal contents. It is more often due, however, to a localized narrowing of the colon low down in its course, either as the result of a congenital malformation or of pressure from the outside, as by bands or adhesions. It is possible that in some instances the interference with the passage of feces may be due to localized paralysis or spasm of the colonic wall as the result of some abnormality in the nervous control. In later childhood it is also possible that the obstruction may be due to localized contractures of the intestine as the result of previous inflammation of the bowel.

Hirschsprung's Disease.—Babies with this malformation are apparently perfectly normal at birth. The symptoms appear, however, as soon as food is taken. The abdomen quickly becomes distended,

often so much as to interfere with the movement of the diaphragm, causing disturbance of the respiration and of the heart action. Cyanosis develops and death not infrequently occurs within a short time, unless the distension is relieved by emptying the bowels. The bowels do not move spontaneously and vomiting quickly appears. If the colon is emptied, the distention and other symptoms disappear, only to recur as soon as food is again taken. Death usually occurs in a few days or weeks. If the babies live more than a few days, the symptoms of toxemia from absorption from the intestines are added to those already present.

The treatment is most unsatisfactory. Nothing can be done except to wash out the colon each time that it becomes distended. Operative measures are certain to be fatal at this age.

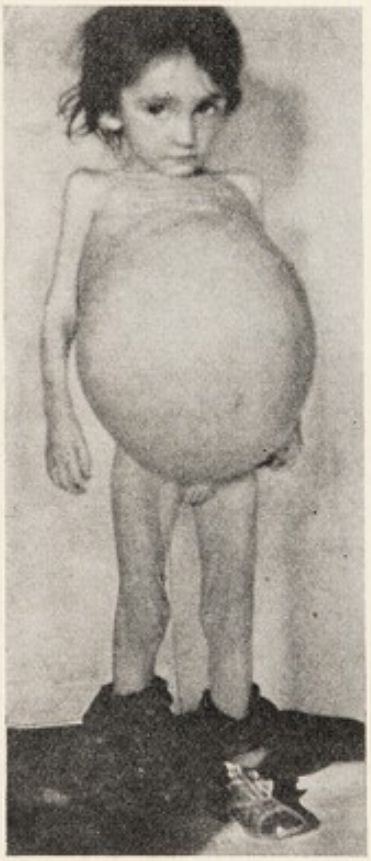

FIG. 60.—Dilatation of colon.

FIG. 61.—Dilatation of colon.

MEGACOLON.—The earliest symptom of dilatation of the colon developing after the first days of life is constipation. This symptom persists, but is occasionally replaced temporarily by diarrhea. Distention of the abdomen gradually develops until it becomes extreme. As the result of the coincident disturbance of nutrition the rest of the body and the extremities are emaciated, so that finally the child seems to be almost all abdomen, with a head and extremities attached to it. The separate coils of the intestine are often distinguishable through the thin abdominal wall and peristaltic movements are not infrequently visible. When the distention is extreme, vomiting is not infrequent. The diaphragm is pressed up so that respiration and the heart action is hampered. Edema of the legs not infrequently develops as the result of pressure on the vena cava. Ulceration of the mucous membrane of the

colon usually develops after a time. This is shown by the presence of large amounts of mucus, pus, blood and sometimes membrane in the stools. Absorption of the products of the decomposition of the colonic contents also occur. As the result of the absorption of these products and of the lesions of the intestinal mucosa, there are often fever and other symptoms of toxemia. Death finally occurs after months or years. Recovery without operative interference is impossible. Pathologically, there is marked hypertrophy of the muscular coils of the large intestine in addition to dilatation and lesions of the mucous membrane.

It is conceivable that megacolon may be confused with distention of the abdomen from fluid from other causes than peritonitis or with distention from tumors of various sort. Careful physical examination will, however, always make a correct diagnosis possible. It can always be

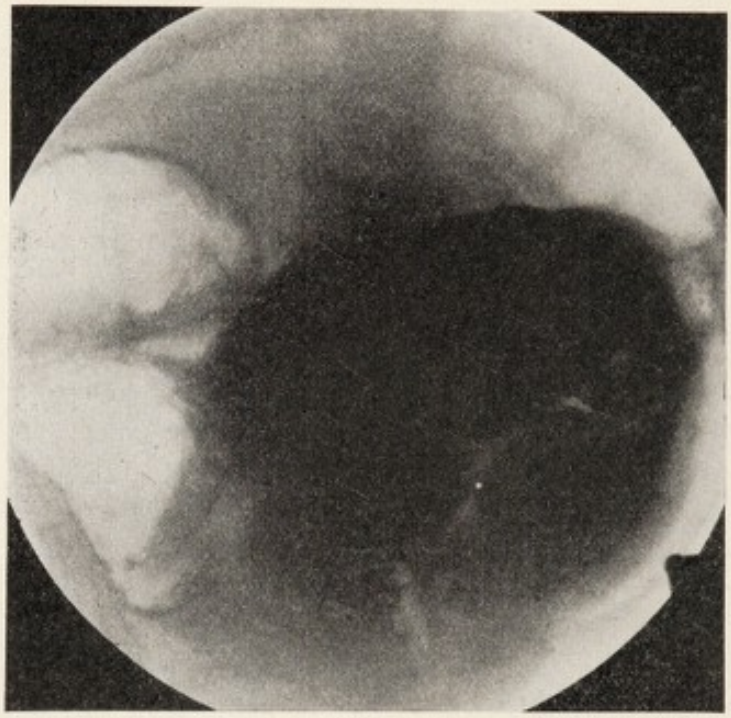

FIG. 62.—Dilatation of colon. Anteroposterior view.

corroborated, moreover, by examination with the Roentgen ray after an opaque enema.

Diagnosis.—The diagnosis of megacolon is usually very easy, the history and picture being so characteristic. It may sometimes be confused with general dilatation of the intestine as the result of chronic disturbances of digestion and rickets. When the enlargement of the abdomen is due to a general dilatation of the intestines, constipation is usually not a marked symptom. In fact, there is usually diarrhea. Individual coils of intestine and peristalsis are seldom visible through the abdominal wall and when they are, they are much less marked than in megacolon. The diagnosis is easily made by giving an opaque enema and examining the abdomen with the fluoroscope or by taking Roentgenograms.

Megacolon may also be confused with the ascitic form of tuberculous peritonitis. It ought not be, however, because in tuberculous peritonitis the distention of the abdomen is uniform, neither peristalsis nor coils of intestine are visible and all the signs of free fluid in the abdominal cavity

are present, physical signs which are diametrically opposite to those found with megacolon.

Treatment.—The treatment consists in giving food which is nutritious but which has relatively little residue and in emptying the colon from below. It is inadvisable in these cases to give coarse foods or foods which have much residue with the idea of overcoming the constipation, because the more bulk there is in the colon, the worse is the condition. Large amounts of milk are, therefore, contraindicated. The carbohydrates should make up a large part of the diet, with a considerable amount of the easily absorbable fats and enough protein to cover the protein needs.

Mineral oil is useful in these cases because it prevents the feces from becoming hard and dry. Laxatives which act on the small intestine are not indicated because the constipation is not due to trouble in the small

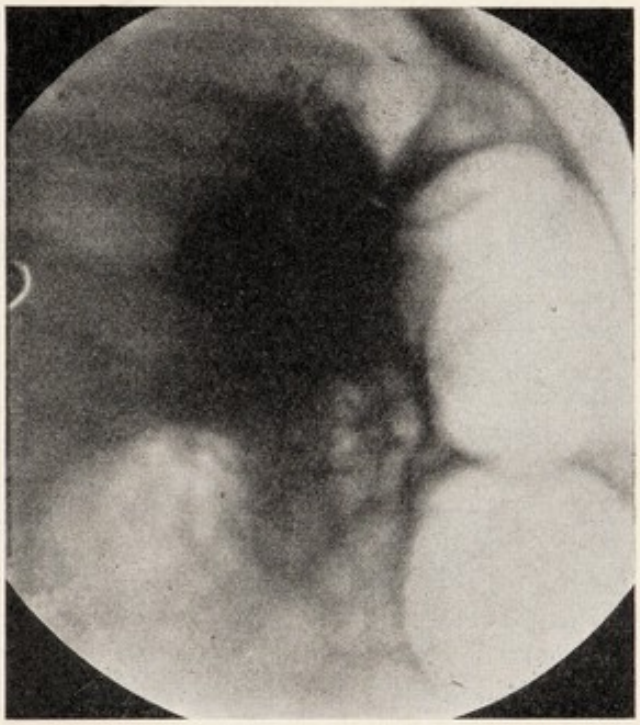

FIG. 63.—Dilatation of colon. Lateral view.

intestine. It is rather doubtful also whether laxatives which act mainly on the large intestine can do much good, because the colon is not able to respond to stimuli to empty itself. The bowels must be emptied, therefore, from below by enemas and by irrigation. Daily irrigations are usually necessary. Water is probably as good as anything. When the feces are very hard, oil enemas are often useful. It is sometimes necessary to empty the rectum mechanically.

Recovery being impossible under medical treatment, it is advisable to operate in this condition while the general condition of the child is good enough to stand the operation. An artificial anus should first be made and sometime later the colon removed and an intestinal anastomosis done. The operation is a very serious one and quite likely to result in death either at the time or soon after. Nevertheless, some chance is better than none. Unfortunately, even when the operation is successful and the child survives, dilatation occasionally recurs higher up in the bowel.

INTUSSUSCEPTION

Intussusception is the name given to the invagination of one portion of the intestine into another. It may occur at any age. Cases have been reported in babies five days old and in late childhood. The vast majority, however, occur in infancy, most often in the second and third quarters of the first year. For some unknown reason it is more common in males than in females. Intussusception is just as likely to occur in the breast-fed as in the artificially-fed, in the strong as in the weak and in the well as in the sick.

The usual location of the intussusception is at the ileocecal valve, the ileum slipping through the valve into the colon. The location of the intussusception may, however, be anywhere in either the small or large intestines. When it is in the small, it is usually low down. The inversion of a Meckel's diverticulum or a polyp may be the cause of an intussusception.

Intussusception is due primarily to an irregular action of the muscular walls of the intestine. The fixed point which causes the intussusception may be due either to localized spasm or paralysis of the wall. Experimentally, the part above the fixed point does not slide down into the lower portion, but the lower portion gradually comes up above the fixed point around the upper portion. This fact is, however, of no great clinical importance except during reduction. Invagination of the gut would not be so serious, if the intestine was not attached to the mesentery on one side and the mesentery did not contain blood vessels. As the invagination progresses the mesentery is drawn in with the gut and the circulation through the blood vessels which it contains is interfered with or cut off. Interference with the circulation results in edema and stoppage of the circulation results in the death of the part involved. The interference with the circulation and the disturbance of the nutrition of the intestinal wall result in infection with bacteria, inflammation and the formation of adhesions. It is evident that in the beginning the resistance to reduction is due to swelling from edema. Later it is due to the formation of adhesions. How early marked edema develops and how early adhesions are formed and gangrene occurs varies markedly in individual cases. It is evident also that how long reduction is possible depends on the rapidity with which these changes develop.

Symptomatology.—The onset is most characteristic. The baby suddenly cries out from severe pain and shows the symptoms of shock—pallor, sweating, rapid pulse, collapse. The pain is always paroxysmal. Between the attacks of pain, which may occur every few minutes or at as long intervals as one or two hours, the baby is perfectly comfortable. Symptoms of shock occur with each paroxysm of pain, but are usually not as marked as with the first paroxysm. Reflex vomiting is not uncommon with the attacks of pain.

In most instances the bowels move soon. At first the stools are made up simply of the intestinal contents. Mucus quickly appears in them, however, as the result of the congestion of the intussuscepted bowel. The mucus soon becomes blood stained as the result of the interference with the circulation. The movements then consist entirely of mucus and blood. Not infrequently there is much more blood than mucus. The blood may be bright or brownish. In some instances there is but little mucus and the stools are composed almost entirely of blood. In rare instances the appearance of these stools is delayed for some time. I have seen a number of cases in which there had been no bloody stools,

but in which there was a profuse discharge of dark blood after a rectal examination.

After obstruction develops, the symptoms change in character. Vomiting begins or increases in severity. The vomitus becomes "fecal" in character. The symptoms of toxemia from absorption from the contents of the obstructed bowel appear and often develop very rapidly. They are fever, rapid pulse and the general symptoms of intoxication. Before the obstruction has developed there is usually but little elevation of the temperature. Distention of the abdomen almost never develops before there is obstruction. It is more often absent than present after the development of obstruction.

Physical Signs.—The only pathognomonic physical signs of intussusception are the presence of a tumor in the abdomen and in the rectum. The tumor in the abdomen is usually described as sausage shaped. I have, however, never felt of any sausages which felt like the tumor in an intussusception. Early in an intussusception, if it is of the ileocolic variety, as it usually is, the tumor is situated in the lower two thirds of the right abdomen. It is small and easily palpable. A little later the mass is at the hepatic flexure under the liver, where it is very difficult to feel. In many instances it is impossible to feel it. Still later it is in the left half of the abdomen, is larger and again fairly easy to feel. When the intussusception is in the small intestine, the tumor may, of course, be situated anywhere. When it originates in the large intestine below the cecum it is usually situated in the left half of the abdomen. It is, of course, impossible to feel the lower portion of the intussusception by the rectum in intussusceptions in the small intestine or in ileocecal intussusceptions until the mass has reached the rectum. When it has got that far it makes very little difference to the baby whether it is felt or not, as by this time the condition has gone too far to be likely to be relieved by any form of treatment.

Diagnosis.—The onset and symptomatology of intussusception are so characteristic that an error in diagnosis should never be made in any but the most atypical cases. Failures in diagnosis probably occur most often because the physician prescribes over the telephone for a colic or fails to examine the baby properly if he sees it. There is no disease of infancy in which it is more important to make an early diagnosis than in intussusception because, when the diagnosis is made early, recovery is the rule. When it is not made early, death is almost inevitable. The diagnosis can often be confirmed by the Roentgen ray after an opaque enema. This procedure is seldom necessary, however, it wastes time and it is a disadvantage at the operation to have the bowel filled with the enema.

An intussusception may be confused with colic. It ought not be, however, because colic is not associated with the symptoms of shock as intussusception always is. Furthermore, the pain in colic, while it may be paroxysmal, is almost always more persistent. None of the later manifestations of intussusception develop, of course, with colic. Intussusception may also be confused with infectious diarrhea. The onset of intussusception is acute, that of infectious diarrhea is relatively slow. There are no symptoms of shock at the beginning of infectious diarrhea. The stools in infectious diarrhea contain fecal matter as well as mucus and blood. There is no fecal matter in the stools in intussusception, after the beginning. There are, of course, no fecal vomiting nor palpable tumor in infectious diarrhea. Intussusception may sometimes be confused with Henoch's purpura. The onset with pain is the same in

purpura as in intussusception. The symptoms of shock are, however, absent. There may be blood and mucus in the stools in Henoch's purpura, but there is also some fecal matter. The pain is usually more persistent. There are almost always other evidences of purpura present. In rare instances, there may be a very small tumor in purpura, never a large one as in intussusception. In very rare instances the purpuric infiltration of the intestinal wall serves as the fixed point for the development of an intussusception. I have seen this several times. The diagnosis in these cases is very difficult and depends on the development of the characteristic symptoms of shock in connection with pain, the absence of fecal matter in the stools and the presence of a tumor in the abdomen. In one case of purpura which I saw, two intussusceptions, both of which were operated on, occurred inside of ten days. Occasionally the bloody discharge from the rectum from a polyp suggests the possibility of intussusception, especially, if there is some pain in the abdomen. The diagnosis is not difficult, however, because there is no sudden onset of pain with symptoms of shock, the stools contain fecal matter, there is no tumor in the abdomen and the polyp can usually be felt with the finger.

Occasionally other forms of acute intestinal obstruction, such as *volvulus* or acute obstruction from bands or an adherent Meckel's diverticulum, may cause symptoms very similar to those in intussusception. The differences in the symptoms are only in degree. A tumor is less often palpable in these conditions and there is more often distension of the abdomen. The diagnosis is easily made, however, at the time of operation.

Prognosis and Treatment.—The almost invariable result of an intussusception in the hands of Nature is death. Occasionally spontaneous reduction occurs. Still more rarely a portion of the gut sloughs off and is passed through the anus, the portions of the intestines above and below the intussuscepted portion having become adherent and joined together. These things happen so seldom, however, that it ought to be taken for granted in an individual case that they will not and that death is certain unless something is done. The prognosis is almost as bad under medical treatment as in the hands of Nature. There are no drugs which can do any good. It is possible that in rare instances at the very beginning of an intussusception, before much edema has developed and before there has been any marked congestion, the introduction of air or water by the rectum may push the bowel back into place. This cannot happen, however, if there is much edema or any inflammation. The chances of the reduction of an intussusception by such measures are so slight, however, that they ought never be used, because much time is usually lost in using them, the infant's general condition is impaired by them, it is less likely to survive an operation after them, and it is impossible to tell, without waiting and losing more time, whether the intussusception has been reduced or not. The rational treatment for intussusception is operation immediately after the diagnosis is made. No delay is justifiable. When the diagnosis is made early and the operation is performed at once, the prognosis is fairly good. The majority of cases recover. When it is made late and the operation is done late, death almost always results. When the operation is done before there is edema, the intussusception can be easily reduced. When there is much edema, reduction is usually possible, but very difficult. When adhesions have formed and the vitality of the gut is impaired, reduction is impossible and an

intestinal anastomosis must then be done. The result of an intestinal anastomosis in infancy is almost invariably fatal. An artificial anus carries almost as bad a prognosis. It is impossible to operate too early on a case of intussusception. The progress of the disease is so rapid in many instances that, even when the operation is done on the first day, reduction is impossible and death certain.

Subacute and Chronic Intussusception

In rare instances an intussusception may be small and not cause enough interference with the circulation in the mesentery to cause complete obstruction. In such cases, the diagnosis is very difficult. It is impossible to describe the symptoms accurately. They are those of intussusception modified, as they must be, by the fact that the obstruction of the gut is only partial. The prognosis is, of course, better. There is more chance of spontaneous reduction. When the diagnosis is made, however, operation should always be done.

FISSURE OF THE ANUS

Fissure of the anus is not very uncommon in late infancy and early childhood. It is almost invariably due to stretching of the anus as the result of the passage of large, hard stools. In acute cases there is merely a linear longitudinal crack of the mucous membrane, which bleeds a little. In cases of longer duration the crack is deeper and there is often a little grayish membrane at the bottom or a slight purulent discharge.

The symptoms are pain on defecation and the presence of a little bright blood on the outside of the stool. When the fissure persists for a considerable time, or there are repeated fissures, spasmodic constipation often results because of the fear of the child of the pain which a movement of the bowel causes and because of a secondary spasm of the sphincter. The diagnosis is easily made by spreading the rectum open and finding the fissure.

The treatment consists in the first place of regulation of the diet and the administration of mild laxatives to prevent the formation of large, hard stools. Enemas may be necessary for a time. The anus should be carefully cleaned after each movement and thoroughly greased with boric acid ointment. In most instances, recovery is prompt with these simple measures. If it is not, the fissure should be touched with the nitrate of silver stick. I have never found it necessary to stretch the sphincter, as is sometimes necessary in adults, to cure the condition.

PROLAPSE OF THE RECTUM

Prolapse of the rectum is not very uncommon during infancy and very early childhood. It seldom develops after the fourth year. Its development is favored by the feebleness of the levator ani muscles at this age, and in thin babies by a deficiency in the ischiorectal fat. The immediate cause is straining at stool. This may be the result of constipation, but is more often of diarrhea. I have never seen it develop as the result of phimosis, pin-worms or stone in the bladder, which are said to be common causes.

Symptomatology.—The bowel seldom protrudes except during and after defecation. It may protrude simply as a small ring of mucous membrane about the anus or may stick out as much as two inches. The protruded gut is purplish red and bleeds easily. If it has been down repeatedly, it shows the evidences of inflammation of the mucous mem-

brane. The prolapse does not cause as much discomfort as would be expected.

Prognosis and Treatment.—As always, the preventive treatment is the most important. Babies and young children that are not constipated and do not have diarrhea never have prolapse of the rectum. The tendency is to rapid recovery when the cause is removed.

Whenever prolapse occurs the bowel should be reduced at once. The patient should be placed on its face across the lap with its legs hanging down, and the tumor pushed back with the fingers which have been oiled. When the protrusion is considerable it must be remembered that the part which is farthest out is that which came down last and must, therefore, be pushed back first. It is impossible to reduce the prolapse, if the attempt is made to push it all in together. It must be turned in gradually in the same way as the finger of a glove which is inside out. When the tumor is not easily reduced, the application of cold for a few minutes will sometimes make the reduction easier. After the tumor is reduced, a pad should be placed over the anus and held in place by a T bandage.

The patient should lie on its back whenever the bowels move in order to prevent straining. It is sometimes advisable, with small babies, to hold the buttocks lightly together while the bowels are moving. With older children, who cannot or will not have a movement of the bowels when lying on the back, a special toilet seat, with a narrow slit in place of the usual oval opening, should be used. It is impossible to strain when sitting on a flat board. It is also advisable to have the seat so high that the child cannot rest its feet on the floor. It is advisable to have the patient lie down for one half hour after the bowels have moved. I have never found suppositories of any sort of any use in this condition and have never seen cases severe enough to need strapping of the buttocks, as is sometimes recommended. I have never seen any cases which did not yield to simple treatment. If they do not, the prolapsing bowel may be touched with the actual cautery in lines parallel with the long diameter of the gut or various operations may be done.

It goes without saying that in addition to reducing the prolapse when it occurs and taking measures to make the prolapse less easy, everything should be done to remove the cause. Unless the cause is removed, all other measures are of little use. When the cause is removed, the prolapse usually stops of itself.

HEMORRHOIDS

Small external piles are not very uncommon in childhood. They seldom cause any discomfort, however, except to the minds of the mothers after they have discovered them. They almost always disappear with time.

Internal piles are very uncommon in early life. When present they are almost always the result of constipation, except in rare instances in which they are the result of venous congestion from some pathologic condition in the abdomen. Except when they are secondary to some other disease, they usually disappear rapidly after the causative constipation is relieved. The treatment of both internal and external piles is the same in childhood as in adult life.

GASTRIC AND DUODENAL ULCERS

Both gastric and duodenal ulcers occur occasionally in the first week of life as manifestations of hemorrhagic disease of the newborn. They may or may not be the cause of bleeding. If they are, there is vomiting of

blood and the passage of blood in the stools. It makes no difference in the prognosis of hemorrhagic disease of the newborn whether the hemorrhage comes from an ulcer of the stomach or duodenum or somewhere else. The treatment is that of hemorrhagic disease of the newborn without regard to whether there is an ulcer of the stomach or duodenum or not.

Small ulcers of the stomach, usually multiple, are occasionally found in connection with tuberculosis in infancy and early childhood. They are, however, merely pathologic curiosities and almost never cause any recognizable symptoms. Hemorrhage and perforation are possibilities, but occur so seldom that they may safely be forgotten.

Small ulcers of the duodenum are apparently not uncommon in early infancy in connection with marked disturbances of the nutrition. They are, however, of no clinical importance and give rise to no demonstrable symptoms or physical signs. They are simply pathologic curiosities. It is probable that these ulcers are often present and heal in babies that recover from these marked disturbances of nutrition.

Ulcers of the stomach and duodenum similar to those found in adult life sometimes occur in early life. They are, however, very uncommon. It is presumable that the symptoms are the same as in adults, but, owing to the inability of children to describe their feelings accurately, the real condition is not only seldom recognized, but almost never suspected. Hemorrhage and perforation may occur in childhood as in adult life. When they occur, the symptoms are the same as in adults.

The treatment of ulcer, whether of the stomach or duodenum is essentially the same in children as in adults. Since there is seldom much induration about the ulcers in early life and since there is practically no chance of the development of malignant disease at the seat of an ulcer in childhood, the advisability of an operation for the removal of an ulcer is much less in childhood than in adult life. Operation for hemorrhage and perforation should be done in children for the same indications as in adults.

NERVOUS DISORDERS OF THE GASTRO-ENTERIC TRACT

Symptoms pointing to disturbance in the gastro-enteric tract develop occasionally as the result of causes or conditions acting directly on the nervous system. The symptoms referable to the digestive tract are due to disturbance of the functions of this tract as the result of abnormal influences transmitted to it from unduly irritable or exhausted nerve centers. The most characteristic symptoms are those due to the disturbance of the mechanical functions of the stomach and intestines. When the symptoms are due to disturbance of the secretory functions of the digestive tract, they are indistinguishable from those due to disturbance from other causes. The condition is then really an indigestion. Only those symptoms which are due to disturbance of the mechanical functions will, therefore, be described. These are more common in infancy than in childhood and more common in early than in late childhood.

Extremes of temperature are the most common of the causes acting through the central nervous system. Heat is a more common cause than cold. Diarrhea results more often than vomiting. If there is vomiting, the vomitus consists simply of the contents of the stomach and shows no signs of indigestion. The diarrhea is due simply to increased peristalsis. The stools, therefore, show no evidences of indigestion, the intestinal contents simply being hurried through the bowels. They are,

however, increased in number and diminished in consistency. The temperature may be slightly increased, if the cause is extremely hot weather, otherwise it is not. There is not infrequently some abdominal discomfort and general constitutional depression.

Excitement and fear may have the same effect as extremes of temperature. They are more likely to cause symptoms in young children than in infants. Vomiting and diarrhea are about equally common. The characteristics of the vomitus and of the stools are the same as when the trouble is due to extremes of temperature. Anger may also cause disturbances. Vomiting is much more often the result of anger than is diarrhea. The temperature is normal and there is usually no abdominal discomfort or general depression.

Theoretically it is possible that unsuitable and indigestible food, by acting as a foreign body, may, by irritation of the stomach and intestines, reflexly cause vomiting and diarrhea without producing any disturbance of digestion. If it does, it certainly happens very seldom. The vomitus contains, of course, the food which is the cause of the symptoms. The stools do also, but in addition are increased in number and diminished in consistency, without showing other evidences of indigestion.

It is doubtful if any of the chronic disorders of the digestive tract are ever due entirely to abnormal nervous influences without indigestion. The primary cause of many of the chronic disturbances of digestion in infancy and childhood is, however, not infrequently some error in the care and routine. This is more often the case in childhood than in infancy. Nevertheless, even in infancy, overexcitement, too much attention, noisy surroundings and lack of rest and sleep are often at the bottom of intractable cases of indigestion in infancy. Overwork at school, too busy or exciting a life, late hours and improper home surroundings are also often at the basis of chronic disturbances of digestion in childhood. No change in diet and no amount of medicine will help these babies or children at all. They will improve only when the undue strain on the nervous system is removed.

Treatment.—It is evident that the first thing to be done in the treatment of disorders of the gastro-enteric tract resulting from causes acting through the nervous system is to remove the cause. Recovery is almost always rapid in acute conditions when the cause is removed. If the cause is excessive heat, the baby must be kept cool. If it is excessive cold, it must be kept warm. If it is excitement or fear, the cause of the excitement or fear must be removed and its recurrence prevented. It is usually advisable to omit one or two feedings and then to begin the usual food weakened for one or two days. If the patient is a breast-fed baby, the duration of the nursing should be shortened and boiled water given before or during the nursing in order to dilute the milk. If the baby is taking an artificial food, it should be diluted with water. If the patient is older and on a mixed diet, the less easily digestible articles should be temporarily omitted. If the cause is anger, it is advisable to cut out one or two feedings. The cure, however, when this is the cause, consists in proper training of the child.

If the cause of the trouble is unsuitable or indigestible food, it is advisable to give a laxative to hurry it out of the bowels before it has had time to set up a disturbance of the digestion. Milk of magnesia and phosphate of soda are suitable for babies, phenolphthalein or one of the preparations of senna for children. It is hardly necessary to give castor oil or calomel. A laxative should not be given, unless the cause of the

trouble is improper food. If the cause is not improper food, and the baby is having a large number of stools, normal except that they are loose, it is allowable to diminish the excessive peristalsis by giving paregoric, in doses of from five to twenty drops every two or four hours, according to the age of the child and the severity of the diarrhea.

SPASM OF THE PYLORUS

It is somewhat doubtful whether spasm of the pylorus belongs with nervous disorders of the gastro-enteric tract or elsewhere, its etiology being very obscure. Many physicians do not believe in spasm of the pylorus as an entity, but think that it occurs only as a complication of hypertrophic pyloric stenosis. I believe that it increases the symptoms in true hypertrophic stenosis, often accounts for the variation in their intensity and is responsible for their sudden onset in some cases. I also believe that there may be spasm without hypertrophy and which does not cause hypertrophy. Pure spasm is, however, relatively uncommon in comparison with hypertrophic stenosis. Its occurrence is practically limited to infancy.

Etiology.—The etiology of spasm of the pylorus in infancy is, as already stated, very obscure. It apparently occurs most often in the excitable and nervous offspring of neurotic parents. It is possible that the normal muscular hyperirritability at this age predisposes to spasm of the pylorus and that, in consequence, the mechanical irritation of the food or of the chemical products of digestion directly or reflexly produce spasm, when they would not in later life. It is also possible that some disturbance of the gastric digestion precedes and causes the spasm. Evidence in favor of this possibility is that spasm of the pylorus is far more common in artificially-fed than in breast-fed babies. Moreover, a hypersecretion of the gastric juice has been found in some cases and hyperacidity of the gastric juice in others. Furthermore, treatment directed to neutralizing hyperacidity often gives favorable results. It is not certain, however, how many, if any, of the cases of spasm of the pylorus are due to this cause.

Symptomatology.—The first symptom is vomiting. It may begin soon after birth, but usually does not develop for several weeks or months. Vomiting may be the only symptom, but in the severe cases it is not infrequently preceded and accompanied by evidences of gastric pain and discomfort. It is at times explosive and at others not. The amount of the vomitus does not ordinarily exceed the amount of food taken at the last meal. It does not, as a rule, show any evidences of disturbance of the digestion, although it may be highly acid. In the severe cases, there is visible peristalsis running over the stomach from left to right, just as in hypertrophic pyloric stenosis. In some instances there is also a palpable tumor at the pylorus. This tumor is, however, not as large and hard as that felt in hypertrophic stenosis, is usually longer and thinner, and in typical cases can be felt to appear and disappear under the finger as the pylorus contracts and relaxes. In hypertrophic stenosis the tumor is, of course, when it is felt, always of the same size. There is a tendency to constipation. The degree of the constipation depends, of course, upon the amount of food which passes through the pylorus. As a rule, the constipation is not very marked, showing that, in spite of the vomiting, considerable food passes through the pylorus into the intestine. There is almost always some disturbance of the nutrition. How great this is depends, of course, upon the amount of vomiting.

Roentgenograms taken after an opaque meal snow interference with the passage of the stomach contents into the duodenum. Not infrequently nothing leaves the stomach for some time or only a little at long

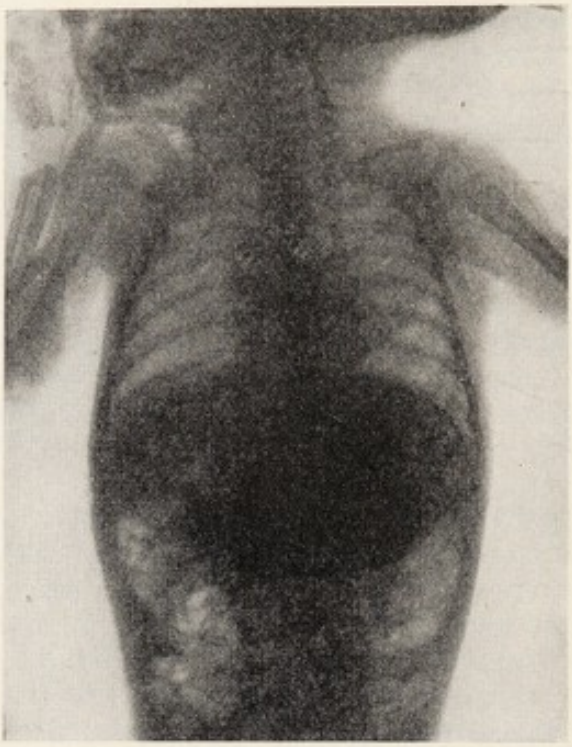

FIG. 64.—Spasm of the pylorus. Taken immediately after an opaque meal.

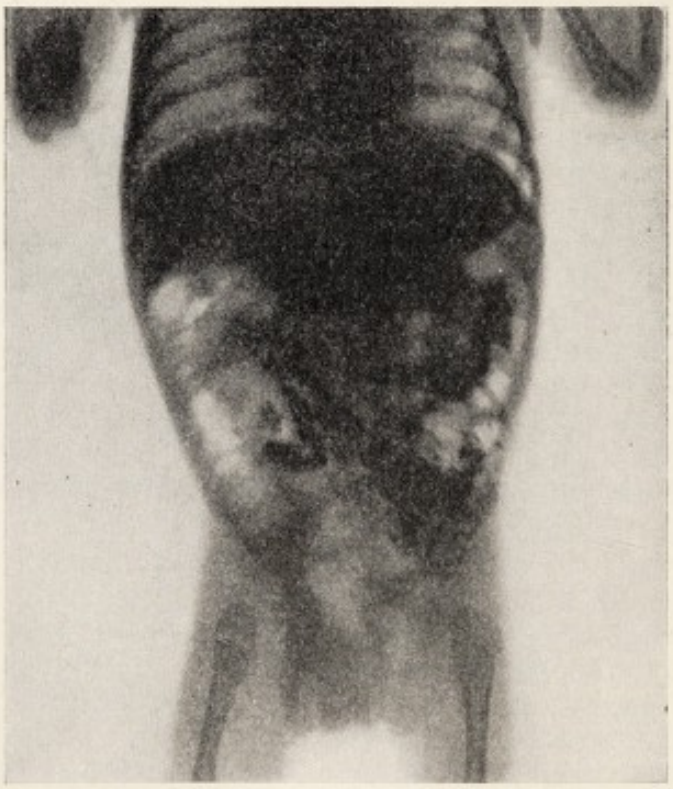

FIG. 65.—Spasm of the pylorus. Taken forty minutes after an opaque meal.

intervals. Eventually, if the meal is not all vomited, the pylorus relaxes and the food passes out, while in stenosis nothing leaves the stomach for

hours or, at best, only a little trickles through. The pylorus never relaxes and finally the meal is vomited.

Diagnosis.—The differential diagnosis between spasm and hypertrophic stenosis of the pylorus is taken up in the discussion of the diagnosis of that condition. The vomiting in spasm of the pylorus is sometimes confused with that which occurs as the result of lack of tone in the cardia. In this condition the vomiting usually occurs soon after the food is taken, but as a rule only when the baby moves or cries. The vomiting is never explosive, there is no visible peristalsis or tumor, the stools are usually sufficient in amount and the nutrition little, if at all, impaired. The Roentgen ray shows no signs of obstruction at the pyloric orifice. Chronic indigestion is also sometimes mistaken for spasm of the pylorus.

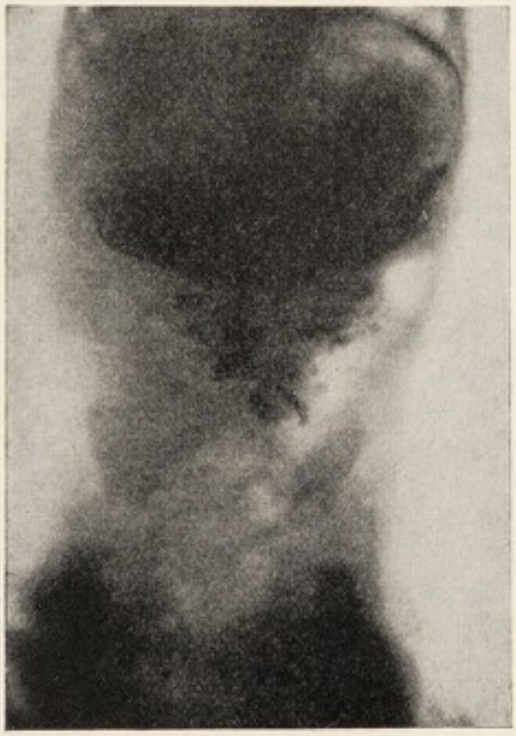

FIG. 66.—Indigestion with vomiting. Taken immediately after an opaque meal.

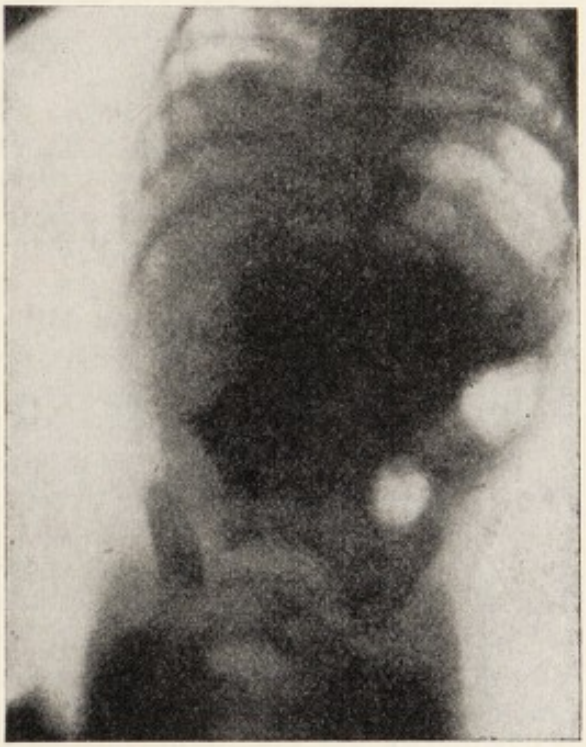

FIG. 67.—Indigestion with vomiting. Taken fifteen minutes after an opaque meal.

It should not be, however, as the symptomatology of the two conditions is quite different. The vomiting of indigestion may begin at any time, is not explosive, almost always occurs several times after the same feeding and varies with the character of the food. The vomitus almost always shows evidences of indigestion. The bowels may be constipated, but the stools are usually sufficient and almost always undigested. There is no visible peristalsis and no tumor. Examination with the Roentgen ray shows that food begins to leave the stomach at once.

Prognosis.—The prognosis is, in general, good. The symptoms persist for many weeks or months in the severe cases, however, even under the most careful treatment. Occasionally cases do not recover. If they do not do well on medical treatment, they are not likely to after an operation, although it would seem as if the splitting of the pylorus ought to relieve this condition entirely and at once.

Treatment.—The treatment of spasm of the pylorus consists almost entirely in regulation of the diet. The best food is good human milk.

If this is vomited, a part of the cream should be removed. If human milk cannot be obtained or is not retained, even after some of the cream is removed and the milk diluted, some modification of cow's milk must be tried. In general, it is advisable to keep the percentage of fat low, because fat tends to delay the emptying of the stomach. A percentage of 0.50 is none too low in the beginning. Carbohydrates, which leave the stomach readily and quickly, can usually be given freely. Lactose is probably the best of the sugars in this condition, but any of them may be tried. It is important to prevent the formation of large casein curds in the stomach, as they leave the stomach with difficulty. One of the best ways to do this is to give as large a portion of the protein in the form of whey proteins, which are not coagulated by rennin, as possible. Plain whey is often useful in beginning the treatment. All the measures detailed elsewhere to prevent the formation of large casein curds may be tried. While these are the general principles to be followed, they can, of course, serve only as a basis for the preparation of the food, which must be varied to suit the individual infant.

Another method of feeding which is worthy of trying and works very well in some instances is the thick cereal feeding recommended by Sauer in the treatment of hypertrophic stenosis of the pylorus. This method of feeding is described in the treatment of this disease.

There is no rule as to whether the food should be given at short or long intervals or as to the quantity which should be given at a feeding. The most rational way of regulating the interval between feedings is to determine how long it takes the stomach to empty itself in the individual instance, and to make the intervals somewhat longer than this. In general it is probably better to give small amounts at a feeding, although there are many exceptions to this rule.

Daily lavage with plain water or a weak solution of bicarbonate of soda is often helpful, although it is possible that, in some instances, it may tend to keep up the spasm. Warm applications to the epigastrium for half an hour before and half an hour after feeding are sometimes helpful. Flaxseed meal poultices are the most efficacious. Minute doses of some preparation of opium—for example, one fortieth of a minim of the tincture—at the beginning of or a short time before feedings sometimes seems to diminish the spasm. Atropin as recommended by Haas in the treatment of pyloric stenosis, ought, theoretically, to be useful in this condition. Haas' method is described in the treatment of hypertrophic stenosis. In my experience it has not been as helpful as might be expected.

Surgical intervention for the relief of pyloric spasm is seldom necessary and should only be tried when it seems certain that the baby will otherwise die. Theoretically, it would seem that splitting the pylorus would prevent its spasmodic contraction and cure the condition. Practically, however, it very often does not relieve the symptoms and, in certain instances, the babies are worse after than before the operation. It would seem also that posterior gastro-enterotomy would relieve the symptoms entirely. It does not do so, however, in the same way that it does in hypertrophic stenosis. Operation should, therefore, be performed only as a last resort.

Disturbances of Digestion

Disturbances of digestion are more common in infancy and early childhood than in middle and late childhood. The etiology of disturbances of digestion is the same at all ages. The relative importance of

the etiologic factors is, however, very different at different ages. The frequency of the different types of indigestion is also quite different in infancy and in childhood. While the symptoms are essentially the same at all ages, their relative severity changes materially with advance in age. The prognosis is also quite different in infancy and in childhood. It is advisable, therefore, to discuss indigestion in infancy and indigestion in childhood separately.

INDIGESTION IN INFANCY

Etiology.—Indigestion in infancy may be due to an excess of an otherwise suitable food, to a too rich, but otherwise well-balanced, food and to food containing an excessive amount of one or perhaps two of the individual food elements. It may also be caused indirectly by other diseases or by any extraneous causes which weaken the general resistance or lower the digestive powers. Indigestion due to causes of this sort is often spoken of as parenteral. The tendency in the past has been not to pay sufficient attention to these parenteral causes. The tendency at present seems to be to exaggerate their importance. It is very easy, for example, to go too far, as is now being done, in attributing so many of the disturbances of digestion to mastoid disease. Indigestion may be either acute or chronic.

Pathology.—The pathologic changes in the gastro-enteric tract are insignificant. In acute disturbances of digestion and also, but less often, in the chronic, there is reddening of the surface and an excessive secretion of mucus, while in some instances there is desquamation of the superficial epithelium. In other instances there are no macrosopic changes beyond thinning of the intestinal wall. The microscopic changes are slight and unimportant. In the more chronic cases there is a general wasting of all the tissues of the body and in both the acute and chronic cases there may be degenerative changes, frequently fatty, in the parenchymatous organs. The important changes, and those which have most to do with the disturbances of nutrition, are in the metabolic processes. These are not recognizable pathologically and are at present imperfectly understood. They vary according to which of the food elements is the cause of the indigestion.

Symptomatology.—Disturbances of the digestion are much less common in the breast-fed than in the artificially-fed. The symptoms are also, as a rule, less severe. All disturbances of digestion, regardless of their cause, have many symptoms in common. The other symptoms vary in accordance with the food element which is the cause of the disturbance. Disturbances of the digestion due to an excess of fat are likely to be more serious and more persistent than those due to the other food elements. Those due to an excess of sugar are more often acute and more often fatal. Those due to an excess of starch are more likely to be chronic than acute. The symptoms of indigestion are usually less marked than those due to the disturbance of the nutrition. Those due to an excess of protein are apparently less frequent and less serious than those due to the other food elements. It must be remembered, in this connection, however, that the symptoms of indigestion due to an excess of protein are less characteristic than those due to an excess of fat and sugar and that, therefore, this apparent infrequency may be due to failure to recognize the symptoms and that the protein may really be at fault, when the blame is attached to one of the other elements.

Simple disturbances of the digestion may be associated with fermentation of the improperly digested intestinal contents as the result of abnormal bacterial activity. This abnormal bacterial activity may take place in any of the food elements. It is probable that there is more or less fermentation in almost every case of indigestion. When these fermentative processes are marked, they often dominate the picture and the condition is then spoken of as indigestion with fermentation. The border line between indigestion with bacterial fermentation and without it is, however, a very indefinite one. In many instances it is impossible to determine in which class a given case belongs. I am in the habit of classifying the disturbances of digestion in infancy as follows:

1. Indigestion from an excess of food.
2. Indigestion from an excess of an individual food element.
   a. Fat.
   b. Carbohydrates.
   c. Protein.
   d. Salts.
3. Indigestion with fermentation.

Indigestion from an Excess of Food

Human Milk.—Indigestion from an excessive amount of human milk is comparatively uncommon for two reasons. In the first place, Nature tends to accommodate the supply of milk to the demand. In the next place, if an excessive amount is taken, the baby usually regurgitates it before it has time to cause any disturbance of the digestion. Indigestion from an excessively rich milk is somewhat more common, but still relatively uncommon. The chief symptoms of indigestion from an excessive amount of human milk or a very rich milk are vomiting, flatulence, colic and an increased number of stools. The babies may at first gain well, but after a time fail to gain properly in weight. These babies are, as a rule, fussy and do not sleep well. The symptoms are, however, seldom very marked. When the milk is very rich, they are likley to lose their appetites. There is nothing characteristic about the vomitus. The stools usually contain fat curds and more or less mucus. Indigestion from an excessive amount of human milk or from a very rich milk is seldom a serious condition. It is usually easily corrected.

When there is too much milk the duration of each nursing must be shortened. How much it must be shortened can usually be determined by observation of the baby's symptoms. More accurate results can be obtained by weighing the baby at intervals during the nursing and stopping it when the desired amount has been obtained. The failure to empty the breasts will usually quickly bring about a diminution in the supply of milk. Until this happens the mother should limit to a certain extent the amount of food which she takes.

When the milk is excessively rich, the intervals between the nursings should be lengthened, because lengthening the intervals tends to diminish the amount of the total solids of the milk. The mother should eat more simple food and should take more exercise. Water should be given at the time of the nursing to dilute the milk, until the strength of the milk has become normal. The amount of water to be given depends on the age of the baby and the strength of the milk. It may be given before or during nursing, with a spoon, in a bottle, or with a dropper put into the

mouth beside the nipple during the nursing. It is almost never necessary to wean a baby because the breast milk is too strong.

Artificial Food.—Indigestion from an artificial food suitable in every way, except that too much of it is given, is much less common than it used to be. Indigestion from an artificial food that is too strong in all its percentages is more common than it was a few years ago, because of the present tendency to feed whole milk dilutions. Indigestion from too much of a suitable artificial food or from one too strong in all its percentages is, however, much more common than that from an excessive amount of or too strong human milk, but very much less common than that from an artificial food containing an excessive amount of one or two of the food elements.

The symptoms of indigestion from an excessive amount of a suitable artificial food or of a food too strong in all its percentages, are loss of appetite, vomiting, an excessive number of stools, flatulence, and colic. For a time there may be regular or excessive gain in weight. In a short time, however, there is failure to gain in or loss of weight. The babies are almost always fussy and irritable and sleep poorly. The vomitus is not characteristic, but may show the evidences of disturbance of the digestion of any or all of the food elements. Evidences of disturbance of the digestion of fat are the most common. If there is much vomiting, the stools may be constipated because of the lack of sufficient food remains to form the normal amount of feces.

The prognosis of indigestion due to too much of a suitable food or to an excessively rich food is usually good. The trouble usually yields quickly and easily to proper treatment.

The treatment consists primarily in cutting down the amount of food or in weakening the food. It is usually sufficient to cut the amount of food down to that suitable for the age and weight of the individual infant. When the food is too strong, it is generally advisable to weaken it more than enough than to bring it to the strength which would be suitable for the average normal baby of the given age. This is necessary, because the digestive powers have usually been so weakened by the excessive demands made upon them that they are unequal to meet even the usual demands. As the digestive powers gradually recover, the food can be slowly strengthened. It is usually possible to cure indigestion from an excessive amount of a good food or from a food too strong in all its percentages without a wet-nurse.

When the disturbance is an acute one from a temporary indiscretion, all food should be stopped for from twelve to twenty-four hours and the intestines emptied with castor oil or milk of magnesia. When the disturbance is a chronic one, it is often advisable to begin treatment with a cathartic. It is not advisable to stop food, even temporarily.

Indigestion from an Excess of Fat

Human Milk.—Indigestion from an excessive amount of fat in human milk is comparatively uncommon. The percentage of fat is seldom very high and, even if it is, babies are usually able to accommodate themselves to it.

The chief symptoms of an excessive amount of fat in human milk are loss of appetite, vomiting and abnormal stools, with more or less flatulence and colic. Gain in weight may be normal or excessive for a time, but finally there is failure to gain in weight or loss of weight. The

symptoms are seldom serious. There is usually nothing abnormal about the vomitus. It may have, in the more severe cases, the odor of butyric and other fatty acids. The stools contain many small, soft curds and sometimes look oily. They are more acid than normal and may cause irritation of the buttocks. Soap stools are almost never seen as the result of an excess of fat in breast milk.

Indigestion from an excessive amount of fat in breast milk is seldom serious. It usually does not last long and is almost always easily corrected.

If the mother has been eating an excessive amount of fat, this should be cut down, although doing so seldom influences the amount of fat in the milk. In most instances the difficulty is that she has been eating too much in general, rather than too much fat. Cutting down her food as a whole, increasing the amount of exercise which she takes and getting her out of doors more usually promptly brings down the amount of fat to within normal limits. Shortening the duration of each nursing to prevent the complete emptying of the breast, because the fore-milk contains less fat than the last milk or "strippings," is theoretically indicated. Practically, however, there is danger of diminishing the amount of milk, when the breasts are not emptied, and it is necessary to diminish the intervals between the nursings in order that the baby may get enough food. Doing this tends to increase the amount of total solids in the milk. Water may be given at the time of the nursing in order to diminish the percentage of fat by diluting the milk. This is hardly a rational method, however, because the other food elements are diminished to the same extent as the fat.

Artificial Food.—Indigestion is more often due to an excess of fat in artificial food than to an excess of any other single element. The results of a disturbance of digestion from an excess of fat are, moreover, more serious, more permanent and more difficult to correct than those due to any other element.

The symptoms are, in general, loss of appetite, flatulence, colic, vomiting and abnormal stools. For a time there may be normal or excessive gain in weight, but soon there is failure to gain in weight or more often progressive loss of weight. The temperature is often elevated in acute disturbances of the digestion caused by fat, but is likely to be somewhat subnormal in chronic disturbances. The vomitus is acid in reaction and has a strong acid odor, usually of butyric and the other fatty acids. It sometimes has a creamy appearance. The most common abnormality in the stools is the presence of many small soft curds. These are often accompanied by mucus. In other instances the stools have a gray, shiny appearance. When there is an excess of neutral fat, the stools may be of a creamy consistency and are often about the color of cream. In other instances they look like curdled milk. More often, especially in the chronic cases, the stools are gray or grayish yellow, large, hard and dry. They may be so dry as to be crumbly. The fat in these stools is in combination with calcium and magnesium in the form of soap, that is, these are the typical "soap stools." In other instances the stools are watery, strongly acid and cause marked irritation of the buttocks. When this happens, the fat is in combination with the alkaline salts, especially sodium. When the disturbance of digestion resulting from an excess of fat in the food is acute, the temperature is not infrequently high. When there is diarrhea, as there often is in acute disturbances, there is not only an excessive loss of fat in the stools, but also a very

considerable loss of alkaline salts, especially sodium. The result is a diminution in the alkali reserve and a tendency to acidosis with an excess of ammonia in the urine. Symptoms of acid intoxication seldom develop, however, and, when they do, they are due mainly to dehydration with secondary functional inability of the kidneys.

When the disturbance of the digestion is a chronic one, there is a continuous loss of magnesium and calcium in the stools and a consequent disturbance of the metabolism. This shows itself chiefly by a chronic disturbance of the nutrition. There is probably some connection between rickets and spasmophilia and chronic indigestion from an excess of fat in the food. The nature of this disturbance is problematical. In severe and long continued cases the disturbance of the nutrition may be so marked that the babies present the characteristic picture of "marasmus" or "atrophy."

The prognosis in indigestion from an excess of fat in an artificial food depends on whether the condition is acute or chronic. If it is acute, it varies with the severity of the symptoms, but is, in general, good. If it is chronic, the prognosis depends on the severity of the symptoms, the duration of the trouble and the degree of the disturbance of nutrition. It is very grave in the marked cases. Recovery is always slow, even in the mild cases. A long time is always required to reëstablish a normal tolerance for fat. Relapses are frequent and of long duration. Even a very slight excess of fat in the food will almost always bring one on.

The treatment of indigestion from an excessive amount of fat in an artificial food consists primarily in diminishing the amount of fat in the food. The fat should be cut out entirely in acute cases. A small amount of fat can usually be put back in the food in a few days. How rapidly the amount can be increased can only be determined by observation of the symptoms and examination of the stools. It is inadvisable, however, to go back to as large an amount of fat as the baby was having before the acute attack, unless this attack was due to some mistake and the giving of an unusually large amount.

In the serious chronic cases all the fat should be immediately cut out. In the milder cases the amount of fat should always be greatly reduced at once. Time is saved, recovery is hastened and tolerance established much more quickly when the percentage of fat is cut down at once below the limit of tolerance than when this point is reached by several small reductions. It is never a mistake to reduce the percentage of fat more than is necessary. Time is always lost, if the reduction is insufficient. The percentage of fat should be reduced to at least two in mild cases, to one in the more severe cases, and to nothing in the most serious. If the stools continue to show an excessive amount of fat after the fat has been reduced, it should be reduced still further, until there are no evidences of fat indigestion in the stools. The percentage of fat may then be slowly and cautiously increased. Not more than 0.25% should be added at a time and several days should intervene between the changes. The stools should be examined after every change to determine whether the tolerance has been passed or not. No further increase should be made, if there is an excess of fat in the stools. It is very difficult to reëstablish a tolerance for fat after it has been weakened and very easy to break it down again. It must be remembered that the continuous use of a food low in fat tends to weaken the power of digesting fat and, therefore, to lower the tolerance for it. Another difficulty that arises when the percentage of fat is very low is that, on account of the high

caloric value of fat, the caloric value of the food may not be sufficient to meet the infant's caloric needs. It is necessary, therefore, when the percentage of fat is much diminished, to increase the percentages of the carbohydrates and protein in order to cover the caloric needs. It is sometimes very difficult to do this without causing a disturbance of the digestion from an excess of one of the other food elements, since the caloric value of fat is more than twice that of sugar, starch and protein. Babies with an almost complete intolerance for the fat of cow's milk can often take the fat of human milk without difficulty. When it is impossible, therefore, to increase the percentages of carbohydrates and protein sufficiently to cover the caloric needs in these cases, human milk should be tried. In some instances, however, these babies are unable to tolerate even the fat of human milk. They can, however, usually take skimmed human milk. I have not found that babies with a marked intolerance for the fat of cow's milk were able to take it much, if any, better, when it was homogenized and have not had good results with olive oil or other oils used in place of cow's milk fat, even when they were homogenized.

Indigestion from an Excess of Carbohydrates

Human Milk.—Indigestion from an excess of sugar in human milk is very uncommon. It is very seldom that the percentage of sugar in human milk is over ten, but increases of from 1 to 3%, although the normal average is 7%, almost never cause any disturbance. The symptoms, when they occur, are the same as those from an excess of milk sugar in an artificial food, but always much less marked. They are flatulence and colic, vomiting and abnormal stools. There is seldom enough disturbance of the digestion to cause any loss of weight. The vomitus sometimes has the odor of lactic acid or acetic acid, but these odors are not pathognomonic. The stools are usually not characteristic, but sometimes they are loose, light green in color, acid in odor and reaction and irritating to the buttocks. The indigestion is never serious and is not usually of long duration.

It is possible that, if the mother has been taking an excessively large amount of sugar, a reduction in the amount of sugar which she takes may result in a diminution in the percentage of sugar in the milk. If the amount of sugar ingested has been, however, within reasonable limits, cutting it down can not be expected to have any effect on the percentage of sugar in the milk. If the percentage of sugar in the milk is a little high, the cause is almost always eating too much food in general rather than too much sugar. It is also almost always associated with an increase in the amount of the other food elements in the milk. Cutting down the food as a whole, increasing the amount of exercise which the mother takes and getting her out of doors more will ordinarily quickly bring down the percentage of sugar, and of the other elements, to within normal limits.

Artificial Food.—Indigestion from an excess of carbohydrates in an artificial food may be due to an excessive amount of either starch or sugar. The sugar at fault may be any one of the sugars commonly used in infant feeding—milk sugar, cane sugar or one of the maltose-dextrins combinations. Many physicians seem to believe that milk sugar is the only one of the sugars which can disturb the digestion. This belief is wrong, however, as any and all of the sugars may cause serious disturbance of the digestion. The disturbances of digestion caused by the various forms

of carbohydrates have many symptoms in common. Each of them, however, also causes certain special symptoms or combinations of symptoms which are more or less characteristic.

Milk Sugar.—Milk sugar in an artificial food seldom causes any disturbance of the digestion, unless there is more than 7% of it in the mixture. Six per cent. or even as little as 5%, will, however, sometimes cause trouble in susceptible infants. It is not advisable to give more than 7% continuously, although many babies can take 10%, or even more, for months without having any disturbance from it. If more than 7% is given continuously, however, there is usually indigestion after a time. The disturbances caused by milk sugar may be either acute or chronic, but are more often acute. This is, of course, the case when a large amount of milk sugar is given suddenly and symptoms arise at once. The symptoms are also likely to come on acutely, even when an excessive amount of sugar has been given for some time. In a considerable proportion of the cases of indigestion due to an excess of milk sugar a part of the symptoms are caused by the products of the fermentation of the sugar as the result of bacterial activity. It is very difficult, and in many instances almost impossible, to determine what part of the symptoms are due to the disturbance of the digestion of the sugar and how much to the products of abnormal bacterial activity in the sugar.

The most prominent and characteristic symptom of a disturbance of the digestion from an excess of milk sugar is the passage of loose or watery, green, acid and irritating stools. They often contain more or less mucus as the result of the irritation of the mucous membrane of the intestine by the acid contents. The odor is distinctly acid. In some instances the characteristic odors of lactic, acetic and succinic acids may be distinguished. The buttocks and genitals are often much excoriated. Vomiting is a much less frequent symptom and usually occurs only when a very large amount of sugar has been taken. The vomitus is acid in reaction and may, like the stools, have the odor of lactic, acetic and succinic acids. The vomitus is usually watery. Flatulence and colic are common symptoms both in acute and chronic disturbances. Loss of weight is a constant and often a marked symptom in the acute cases. It is usually not very marked in the chronic disorders. The temperature often rises rapidly and is not infrequently very high in the more severe acute disturbances of digestion due to an excess of milk sugar. It is very doubtful, however, whether the rise in temperature is due directly to the absorption of the sugar. It is seldom of long duration. The temperature is but little or not at all elevated in the more chronic cases. The symptoms of intoxication may be very marked in the severe acute cases. Among them are restlessness and other evidences of disturbance of the nervous system, marked prostration and disturbance of the rhythm of the respiration.

Acute indigestion from milk sugar in an artificial food may be very serious and dangerous. If the babies survive for forty-eight hours after the onset of the symptoms, however, they usually get well. Improvement is rapid after it once begins. The outlook in chronic indigestion from milk sugar is good as to life, but some weeks or months are usually necessary before the tolerance for milk sugar is reëstablished. It is usually easier, however, to reëstablish a tolerance for milk sugar than for fat.

In acute disturbances of the digestion from an excess of milk sugar, milk sugar must be eliminated as far as possible from the food. It is impossible to eliminate it entirely, however, when milk is used, because

milk contains milk sugar. It must be remembered in this connection, moreover, that whey contains between 4.5% and 5% of milk sugar. Whey and whey mixtures are contraindicated, therefore, because they are almost never made with a diluent. The percentage of milk sugar in them is, therefore, always high. Fat is also usually not well tolerated. The indications are, therefore, for mixtures containing but little fat and milk sugar and a considerable amount of protein. Starch is usually tolerated in small amounts in this condition, probably because the starch is broken down slowly and its end-product, dextrose, quickly absorbed. Mixtures containing from 0.50% of 1% of fat and from 1% to 2%, or perhaps even more, of protein, with no additional milk sugar, are, therefore, suitable. Mixtures which contain 1% of protein must necessarily contain at least 1.25% of milk sugar and those which contain 2% of protein at least 2.50% of sugar, unless a part of the protein is added in the form of one of the powdered casein preparations. Starch, in the proportion of from 0.50% to 0.75%, may be added if desired. It is evident that mixtures of skimmed milk with cereal diluents and without added sugar also meet these indications. Some of the dried milks like Dryco, from which a part of the fat has been removed, or the skimmed milk Klim, when mixed with water, also contain a low fat and sugar with a high protein. Low percentages of fat and sugar with high percentages of protein can be obtained by adding precipitated casein to skimmed milk or even to whole milk mixtures. Albumin milk is also sometimes useful in these cases. It is difficult, however, with mixtures of this general character, to cover the caloric needs of the infants. It is advisable, therefore, after a few days to add one of the maltose-dextrins combinations to the mixture in order to bring up its caloric value. It is usually possible to return after a short time to milk sugar. Human milk is contraindicated in these cases because of the high percentage of milk sugar which it contains. It is usually well borne after the acute symptoms have subsided. In convalescence it is, as always, the best food.

In subacute and chronic disturbances of digestion from an excess of milk sugar, all the milk sugar which is being added to the food should be cut out at once, thus reducing the percentage of milk sugar to that which is necessarily put into the food in the cream and milk. If the mixture is in other respects a suitable one and there are no evidences of disturbance of the digestion of the fat and protein, their percentages may be left unchanged. It is advisable, if possible, however, to increase the percentage of protein a little in order to raise the caloric value of the food. It is usually inadvisable to increase the percentage of fat, at any rate at first, in order to bring up the caloric value, because there is very likely to be a diminished tolerance for fat when there is a disturbance of the digestion of milk sugar. In fact, it is often advisable to cut it down a little temporarily. Starch may be added to the amount of 0.75%, or even 1%, in order to increase the caloric value. If the caloric value of the food is still too low, it may be increased after a few days by the addition of one of the maltose-dextrins combinations or by the addition of corn syrup. Cane sugar should not be added, because it almost invariably is badly borne when milk sugar is not tolerated.

Whey and whey mixtures are contraindicated in these cases, because of the high percentage of milk sugar which they contain. The various preparations of dried milk, mixtures to which precipitated casein is added and albumin milk are all often useful temporarily in these, as in acute cases, because of the high percentage of protein and the low percentages of

milk sugar and fat which they contain. One of the maltose-dextrins preparations may be added to these mixtures if desired.

In mild chronic disturbances of the digestion from an excess of milk sugar it is often possible, after cutting out the milk sugar temporarily, to gradually increase it enough to cover the caloric needs without causing a recurrence of the symptoms. It should be added, however, slowly, increasing the percentage not more than 0.50 at a time. In these cases it is not necessary to make use of the maltose-dextrins preparations or starch. Milk sugar being on the whole the most suitable sugar for infants, it is advisable, but not necessary, to replace the maltose-dextrins preparations, if they have been added, by milk sugar, as soon as it is possible.

Cane Sugar.—The symptoms of disturbance of the digestion, whether acute or chronic, from an excess of cane sugar in the food are essentially the same as those from an excess of milk sugar. Extreme elevations of the temperature in acute disturbances are, however, somewhat less common. Babies that get a large amount of cane sugar in their food not infrequently show marked evidences of disturbance of the nutrition for some time before the appearance of indigestion. They may, in fact, get into a very serious condition before any evidences of indigestion develop. They become fat, flabby, and pale, while their resistance to infection and disease is materially diminished.

The prognosis in acute disturbances of the digestion as the result of an excess of cane sugar in the food is essentially the same as that in the disturbances caused by milk sugar. It is not quite as good in the chronic cases, however, because of the greater disturbance of the nutrition produced by the long continued use of large amounts of cane sugar.

The treatment of disturbances of the digestion due to an excessive amount of cane sugar is along the same lines as when the disturbance is caused by milk sugar. The cane sugar should be cut out entirely. The percentage of sugar in the food is then equal to that of the milk sugar which is contained in the cream and milk in the mixture. After the symptoms have ceased, the percentage of sugar can be gradually increased by the addition of milk sugar. If the symptoms recur, when milk sugar is added to the mixture, one of the maltose-dextrins preparations or corn syrup can be substituted for it. Starch may also be added in order to increase the caloric value of the food. If desired, the dried milks or albumin milk may be used, or powdered casein can be added to milk mixtures, as in the treatment of indigestion from an excess of milk sugar.

Maltose-Dextrins Preparations.—The symptoms of indigestion from an excess of one of the maltose-dextrins preparations are much like those caused by other sugars. The odor of the vomitus, while acid, is somewhat different from that caused by the other sugars. This difference is probably due to the presence in it, in many instances, of butyric acid. Flatulence and colic are usually more marked symptoms than when the indigestion is due to one of the other sugars. The stools are almost always loose or watery. They are usually dark brown in color, but are some times green. The odor is usually a peculiarly acrid one. Sometimes, however, it is that of butyric acid. The stools are strongly acid in reaction and cause very marked irritation of the buttocks, thighs and genitals. The temperature is usually less elevated in acute disturbances of digestion from the maltose-dextrins mixtures than when they are due to the other sugars. The prognosis in the disturbances of digestion caused by the maltose-dextrins preparations is usually somewhat better than in those

due to the other sugars. The acute disturbances are usually not as severe and both acute and chronic disturbances ordinarily yield more readily to treatment.

The treatment of disturbances of digestion due to an excess of the maltose-dextrins preparations is necessarily along the same lines as when the other sugars are at fault. It consists primarily in the immediate withdrawal of the preparation. What sugar there is left in the food is then milk sugar, its percentage depending upon the amount of milk and cream in the mixture. There is usually somewhat less diminution in the tolerance for fat in disturbances of the digestion caused by the maltose-dextrins mixtures than in those caused by milk sugar and cane sugar. It is, therefore, often possible to increase the caloric value of the food by increasing somewhat the percentage of fat in it. The percentage of casein may be increased as in disturbances of digestion caused by the other sugars. All the measures used in the treatment of these disturbances are applicable to disturbances caused by the maltose-dextrins preparations. It is usually possible, however, to continue the same mixture without the maltose-dextrins preparation which was at fault, provided it is otherwise suitable, and to gradually substitute milk sugar for it. In most instances milk sugar is well borne in these cases and can be quickly substituted for the maltose-dextrins preparations. When the disturbance of digestion is slight and it is inadvisable for some reason to use milk sugar, it is sometimes possible to correct the disturbance by substituting one of the other maltose-dextrins combinations for the one in use, because the greater the proportion of maltose in these preparations the greater in general is their laxative action. The substitution of another preparation containing a larger proportion of the dextrins and a smaller proportion of maltose will, therefore, sometimes relieve the symptoms.

Starch.—Even when starch is added in excess to a food of which milk forms the basis, it usually causes relatively little disturbance of the digestion and effects the nutrition but little. Starch is much less likely to disturb the digestion, if it is thoroughly cooked than when it is partially cooked. It almost never causes trouble unless there is 1% or more of starch in the mixture. The most common symptoms are flatulence and colic. Vomiting is unusual. The stools are sometimes hard and dry. Usually, however, they are loose. They are more acid than normal, have an acid odor and irritate the buttocks. The undigested starch in the stools may be mistaken for mucus. It can be easily distinguished from mucus by the addition of some preparation of iodine. This stains starch dark blue and does not effect mucus. Even if it is not visible macroscopically, it can be found microscopically in all cases in which there is disturbance of the digestion from it. Many iodophilic bacteria are almost always found with it. These organisms are often present, moreover, even when there is no starch. When found, they always suggest that a disturbance of the digestion from starch is likely to develop.

The prognosis of disturbances of digestion caused by an excess of starch in milk mixtures is good. Recovery is almost always prompt when the cause is removed. The treatment consists in cutting the starch entirely out of the food. It can ordinarily be put back in reasonable amounts after a short time. Trouble will seldom recur, if the percentage of starch is not over 0.75%, which is the optimum amount to use when the object is to prevent the formation of large casein curds by its colloidal action.

Fortunately very few babies are fed in this country on foods composed entirely of starch or of starch and sugar. Such foods are more likely to cause marked disturbances of nutrition than indigestion. When they cause the symptoms of both, the symptoms of malnutrition are always more marked than those of indigestion. The disturbance of nutrition which the exclusively starchy foods cause is due partly to their insufficient caloric value but more to their deficiency in protein and salts. Babies fed exclusively on these foods may seem to thrive for a time, in that they gain in weight, have a fair color, and seem lively and well. Even at this time, however, there is usually an exaggerated muscular tonicity, which is one of the early manifestations of disturbance of nutrition. After a few weeks, however, they begin to lose in weight and color and their muscles become flabby. If the exclusively starchy diet is continued they gradually take on all the characteristics of the starved atrophic infant. Some of them become markedly edematous. Many of

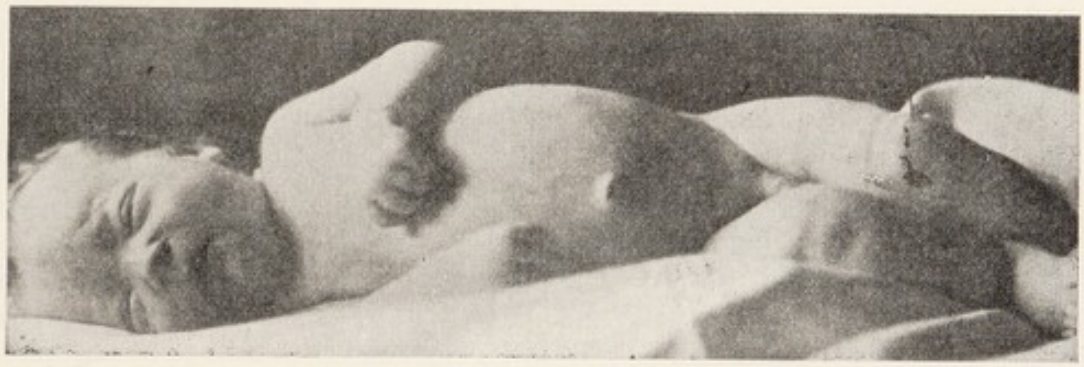

FIG. 68.—Edema from indigestion.

them die of incurrent infections before reaching the atrophic stage, as the resistance to infection is almost always markedly lowered in disturbances of nutrition resulting from an exclusively starchy diet.

The prognosis in chronic disturbances of the digestion due to an exclusively starchy diet is always a grave one, partly because of the marked disturbance of the nutrition and partly because of the lowering of the resistance to infection. Many weeks and sometimes months are required to overcome the disturbance of the nutrition. The prognosis in acute disturbances of the digestion from starch is good. Recovery is almost always prompt with proper treatment.

The treatment in the acute cases is immediate stopping of the starchy food. Human milk should be given, if possible. It may be necessary to dilute it with water at first. If human milk is not obtainable, a modified milk, in which the percentages of all the food elements are low and in which the relations of the fat, milk sugar and protein to each other are similar to those in human milk, can usually be given at once. These first mixtures should contain from 1% to 2% of fat, from 4% to 5% of milk sugar and 0.75% to 1.25% of protein. Whey and whey mixtures are often useful in the beginning. The treatment in the chronic cases is along the same lines. It is more difficult, however, to find a food which fits the digestive capacity in these than in the acute cases, because the functions of the digestion and metabolism of fat and protein have usually been weakened by disuse and by the impairment of the nutrition. It is always advisable in these cases to give human milk, if it can be obtained.

The disturbance of the nutrition when the purely starchy foods are partially dextrinized is as great as when they are not, because their

caloric value is no greater and they are just as deficient in protein and salts as before they were dextrinized. The symptoms of disturbance of the digestion from starch are, however, diminished, although those from an excess of malt sugar may take their place. When the maltose-dextrins preparations or other sugars are added to the starchy foods their caloric value is increased, and on this account the disturbance of nutrition from an insufficient caloric intake is less. That due to the deficiency of protein and salts is, however, the same. The symptoms of disturbance of the digestion caused by these sugary and starchy foods are a combination of those due to an excess of starch and those due to an excess of sugar. The symptoms caused by the excess of sugar usually predominate. They vary somewhat according to the kind of sugar present in the food.

Indigestion from an Excess of Protein

Human Milk.—Indigestion from an excess of protein in human milk is much more common than from an excess of fat or sugar. The percentage of protein is more likely to be excessive during the early part of lactation, before the equilibrium of the breast milk has been established and the mother has resumed her normal life, than it is later. An excess of protein may be due to anxiety or nervousness on the part of the mother or to either fatigue or lack of exercise, all of which increase the protein content of the milk. It is impossible to know in advance what percentage of protein will be too much for an individual infant. Some babies are upset, if the protein is more than 1.50%, while others can take 2.50% or even 3.00% without being disturbed in any way.

Flatulence and colic, with consequent restlessness, crying and sleeplessness are common symptoms. In many instances they are very marked and very troublesome. Vomiting, on the other hand, is a comparatively uncommon symptom. There is, however, almost invariably an increase in the number of stools. These are either loose or watery. They are usually brownish yellow instead of golden in color, but are sometimes green. They often contain mucus and small, soft, fat curds. The fat curds may be due to an accompanying fat indigestion, but are more often the result of increased peristalsis and consequent interference with absorption. The reaction of the stools is alkaline or feebly acid. The odor is not characteristic. It may be acid or a little foul. The stools do not ordinarily irritate the buttocks. The temperature is usually not elevated, but may be in acute cases when there has been a sudden and marked increase in the percentage of protein. In such cases it may be considerably elevated. The nutrition is not as much effected as would be expected from the severity of the symptoms. There is ordinarily not much loss of weight. Many babies continue to gain, although slowly, while occasionally a baby will gain rapidly in spite of much colic and many loose stools.

Occasionally, when there has been a sudden and marked rise in the percentage of protein, babies may die within a few days. It is not certain, however, that the cause of death in such instances is really an increase in the amount of protein. It may perhaps be due to some unknown chemical change in the milk or to the presence of toxic substances of some sort. As a rule, disturbances of the digestion from an excess of protein in human milk are not dangerous, and recovery is prompt when the cause of the increase is removed. When there is an excess of fat and sugar as well as of protein in human milk, the indigestion

is really an indigestion from an excessively rich milk rather than from an excess of protein. The treatment is described under that condition. When the percentages of fat and sugar are within normal limits, while the percentage of protein is alone excessive, it is very difficult to diminish the percentage of protein without affecting the other elements also. Diminishing the relative proportion of protein in the diet should, however, be tried. If the excess of protein is the result of the mother's inactivity, it can be diminished by making her take more exercise. Exercise in the open air is preferable to that indoors. So also is exercise which is enjoyable to that which is disagreeable. Care must be taken, however, that the mother does not take too much exercise and become fatigued, because fatigue increases the amount of protein in the milk. If the excess of protein in the milk is due to fatigue or overwork, it can be diminished by resting the mother and keeping her more quiet. When the high percentage of protein is due to worry or anxiety, the removal of the cause of the worry or anxiety will at once result in a diminution in the percentage of protein. When it is due to nervousness, inherent to the mother, it is very difficult and often impossible to arrange her surroundings so as to overcome her natural temperament.

The percentage of protein can also be diminished somewhat by cutting down the mother's food, by increasing the length of the intervals between the nursings and by giving the baby water at the time of the nursing. These measures all diminish the percentages of fat and sugar at the same time, however, and thus may weaken the milk so much that its caloric value is insufficient.

Artificial Food.—Indigestion is almost never due to an excess of protein in an artificial food, unless that food is cow's milk or some modification of cow's milk. When there is a disturbance of the digestion as the result of an excess of protein in cow's milk, the excess is almost invariably of casein not of whey protein. Indigestion from an excess of casein is far more common now than it was a few years ago, because of the present fashion of feeding whole milk and whole milk dilutions.

Whey Protein.—When babies that are being fed on whey or mixtures containing a high percentage of whey protein have a disturbance of the digestion, this is due, in the vast majority of instances, to the milk sugar and salts in the whey rather than to the whey protein. The symptoms are, therefore, those of an excess of milk sugar and of salts. It is possible, however, that in rare instances the whey protein may cause a disturbance of the digestion. The chief symptom of such a disturbance of the digestion is an increased number of loose, watery stools. These may be otherwise normal, but are sometimes brownish and alkaline and have a musty odor. There also may be flatulence and colic. Vomiting is unusual.

Indigestion from an excess of whey protein is usually not severe and yields promptly to proper treatment. This consists in stopping the whey or in diminishing the percentage of whey protein and giving the necessary percentage of protein in the form of casein.

Casein.—Indigestion from an excess of casein in the food may be either acute or chronic. It is far more often chronic than acute. When acute, there is almost always some elevation of the temperature, which is sometimes high. When high it is possible, however, that the rise may be due to some other cause, such as an excess of salts. In such cases there is an increase in the number of stools, which are loose or watery. They are brownish in color, alkaline in reaction and have a musty odor.

At times they contain an excess of mucus, but almost never curds. In some instances there are marked signs of intoxication. In other instances the symptoms of acute indigestion from an excess of casein are less marked. There is simply the vomiting of large curds, some flatulence and colic and the passage of large, hard curds in the stools. In such cases there is usually little or no elevation of the temperature.

In chronic disturbances of the digestion from an excess of casein there is often vomiting. The vomitus often contains very large curds. These curds may be fairly soft or tough and leathery. The vomitus ordinarily has but little odor. It may smell slightly acid, but is never strongly acid. Flatulence and colic are often marked and very troublesome. The most common abnormality in the stools is the presence of large, hard curds. The number of stools may or may not be increased. In many instances the stools are normal in character, except for the presence of curds. If there are no other evidences of disturbance of the digestion, the presence of large, hard curds in the stools is not of great importance. Another common evidence of disturbance of the digestion from an excess of casein in the food is constipation. This is often very marked. The stools are hard and dry, usually alkaline. Microscopically, of course, they show nothing abnormal. There is usually no fever in chronic disturbances of the digestion from casein. There is, however, not infrequently disturbance of the nutrition, shown by failure to gain in or loss of weight. It is possible, however, that a part of the disturbance of the nutrition may be due to an insufficient caloric value of the food, because when an excess of casein is given there is usually an insufficient amount of fat and often of carbohydrates in the mixtures.

The prognosis of acute disturbances of digestion in which there are numerous, loose, brown, alkaline, musty stools is always grave. It is not serious in the acute cases in which the symptoms are vomiting and the passage of large curds in the stools. It is good in the chronic disturbances.

When the stools are watery, brown and musty, the protein must be cut entirely out of the diet for a time and some form of carbohydrate given in its place. Any of the cereal waters, to which milk sugar or one of the maltose-dextrins mixtures may be added, is suitable. Protein in some form must be added to the diet again as soon as possible, however, in order to prevent disturbance of the nutrition from the loss of body protein as the result of the lack of protein in the food. The same method of treatment should be adopted in the milder acute cases with the vomiting of curds and the passage of large curds in the stools. It is almost always possible in such cases to return to a mixture containing casein in the course of forty-eight hours.

When the indigestion is chronic, the amount of casein in the food must be cut down or measures taken to prevent the formation of large casein curds and thus to make the digestion of the casein easier. The simplest method is to diminish the percentage of casein in the milk mixture. Care must be taken, however, not to diminish it so much that the protein needs of the baby are not covered. If it is not considered desirable to diminish the amount of protein in the mixture, a part of it may be given in the form of whey protein. If it does not seem advisable to diminish the percentage of casein, any of the methods which interfere with the formation of large casein curds may be tried. These methods are given in detail in the chapter on the General Principles of Artificial Feeding. It is often very hard to decide which method

to use in a given case. A careful study of the conditions in the case and a thorough comprehension of the way in which the formation of casein curds is prevented in each method will usually show, however, which one is most suitable under the circumstances.

Indigestion from an Excess of Salts[1]

There is no doubt that the salts play a most important part in the metabolism of the other food elements and that the metabolic processes cannot progress normally unless the proper salts are present in the proper proportions. There is unquestionably a disturbance of the metabolism of the salts in all disturbances of nutrition in infancy. It is very difficult to determine, however, whether the disturbance of the nutrition in any given case is due primarily to a disturbance of the salt metabolism from an insufficiency or improper combination of the salts in the food or whether the disturbance of the salt metabolism is secondary to an insufficiency, excess, or improper combination of one or more of the other food elements and to the disturbance of the digestion caused by them.

There is no doubt, moreover, that the salts play an important part in every digestive disturbance in infancy. It is probable, but not certain, that the salts may of themselves cause disturbance of the digestion independently of the other food elements. Very little is known as to the symptoms which an insufficiency or an excess of the salts as a whole in the food may cause. If the salts are cut out of a food, which is otherwise unchanged, the weight falls. When they are put back again, the weight rises. This variation in the weight is probably largely, but not entirely, due to variations in the retention of water. The sodium salts favor the retention of water. The salts of calcium diminish its retention to a moderate extent. It is also known that the withdrawal of salts from the food results in a lowering of the body temperature. An excess of calcium in the food also lowers the temperature. If a large amount of sodium chloride is given to a baby suffering from a disturbance of the digestion, there is usually a rise in the temperature. If there is no disturbance of the digestion, there is ordinarily no elevation of the temperature. Variations in weight and temperature are, however, common to all disturbances of the digestion and do not, therefore, justify the diagnosis of indigestion as the result of some abnormality in the salts of the food. There being no symptoms peculiar to abnormalities in the salts of the foods, it is, therefore, impossible at present to make a diagnosis of indigestion from an excess or from an improper combination of the salts in the food. The condition may be suspected, but that is all.

It being impossible to make a positive diagnosis of indigestion from an excess or an improper combination of the salts in the food, it is evidently impossible to make a definite prognosis or to lay down any rules for treatment.

The Medicinal Treatment of Disturbances of the Digestion in Infancy.—The treatment of disturbances of digestion in infancy consists primarily in regulation of the diet. All other methods of treatment are relatively unimportant. They have their place, however, and cannot be dispensed with. They are especially useful for the relief of symptoms.

It is advisable to stop food entirely for from twelve to forty-eight hours in all acute disturbances of digestion, whatever their cause. There

[1] This section is copied from "Diseases of Nutrition and Infant Feeding" by Morse and Talbot, published by the Macmillan Co.

is no danger in stopping the food, if the baby is given as much water as it would take of food. Babies can get along very well for a time without food, but they cannot get on without water. No fear need be entertained of the development of acid intoxication. If they object to plain water, they will usually take it willingly, if it is sweetened with saccharin. There is no objection to giving it in the form of very weak tea sweetened with saccharin, if the baby is a German or likes it better that way. The length of time during which food is withheld depends on the severity of the symptoms in the given case. Great care must be taken, however, in stopping the food of babies suffering from chronic disturbances of nutrition, even when there is an acute exacerbation of the symptoms. Such babies can not bear acute starvation on top of chronic inanition. The complete withdrawal of food is very likely to kill them. It is much wiser, therefore, under these conditions, to weaken or change the food than to stop it entirely.

The bowels should be thoroughly cleaned out in every acute disturbance of the digestion, whatever its cause, unless this disturbance is very slight. The most useful drug is castor oil, because it is effective and acts quickly, and, as Abt has shown, causes less irritation of the bowels than calomel and strong salines. The dose should be from one to three teaspoonfuls, according to the age of the baby and the effect desired. It is important to give enough. More than enough does no harm, because it is carried out quickly as the result of the catharsis produced by it. Most babies not only do not object to the taste of castor oil, but like it. It should, therefore, be given plain and no effort made to disguise its taste. The stools produced by castor oil always contain mucus. Too much importance must not be attached, therefore, to the presence of mucus in the stools after a dose of castor oil. It must also never be forgotten that, if castor oil is given repeatedly, the stools will continue to contain mucus. Castor oil is often retained, even when food is vomited. It should, therefore, be tried, even if the baby is vomiting. If it is vomited twice, calomel may then be tried. It is best given in doses of $\frac{1}{10}$ of a grain combined with one grain of bicarbonate of soda, every half hour, until one grain has been taken. It is advisable, but not necessary, to give one or two teaspoonfuls of milk of magnesia three or four hours after the last dose of calomel. It should not be forgotten that calomel often gives the stools a peculiar green color, which may be mistaken for that resulting from disturbances of the digestion in which the stools are highly acid.

If less vigorous catharsis is desired than that usually produced by castor oil or calomel, milk of magnesia or one of the preparations of senna may be used instead. Milk of magnesia is preferable to senna. The dose is from one to three teaspoonfuls, according to the age of the baby and the effect desired. It may be given in the food, plain, or diluted with water.

When the disturbance of the digestion is a chronic one, it is, as a rule, inadvisable to begin treatment with catharsis. Catharsis is a weakening procedure and when a baby is in a debilitated and feeble condition as the result of a long disturbance of the nutrition it is liable to do serious harm. It may, in fact, take away the baby's last chance of recovery.

If the disturbance of the digestion is acute, severe and associated with considerable elevation of the temperature, it is also advisable to wash out the colon with salt solution or at least to give an enema of suds to clean out the bowel from below.

If babies with acute disturbances of digestion continue to vomit after all food has been stopped, they may be given plain water or water sweetened with a little saccharin. If the vomiting is severe, they should be given a teaspoonful every twenty to thirty minutes of water to which bicarbonate of soda has been added in the proportion of one level teaspoonful to eight ounces. If the vomiting is severe or persistent, the stomach should be washed out once or twice daily with a solution of bicarbonate of soda of the strength of one rounded teaspoonful to a pint of water.

Lavage is not a difficult procedure in infancy. It is usually easy to introduce a catheter, even in a young baby. A soft rubber catheter, #16, American scale, or one a little smaller, should be used. The catheter should be attached by a short piece of glass tubing to a rubber tube attached to a funnel. A medicine dropper should never be used as the piece of glass tubing, because, as a chain is no stronger than its weakest link, the caliber of a tube is no larger than its smallest part. The baby should be well wrapped up and it and the nurse protected by a rubber apron or sheet. It should be held upright in the nurse's lap with its back to her, facing forward and bending a little forward. The mouth can be held open by the forefinger of the left hand around which a towel may be wrapped. The tube should be introduced through the mouth, not through the nose. The catheter, which is held in the right hand, is pushed to the back of the throat and downward. It passes most easily when the baby gags. The distance from the gums or incisor teeth to the cardia during infancy is between seven and eight inches. The catheter should not, therefore, be introduced further than this. It is very difficult to pass a catheter anywhere except into the esophagus. It can be passed through the larynx, however, because I have done it myself. Water should never be poured in, therefore, until the baby has cried clearly or food has come up through the tube. It is easier to start the siphonage, if the tube is introduced full of water. The washing should be continued until the water returns clear. It is useless to keep on after this happens.

It is almost never necessary or advisable to give an emetic to infants suffering from disturbances of the digestion. A teaspoonful of the wine of ipecac is the safest and best emetic, if one is necessary. It is better to give a teaspoonful than a smaller dose, because this is certain to be vomited at once and thus causes less irritation than a smaller dose which is not vomited.

The best treatment for the flatulence and colic which so often accompanies disturbances of the digestion is the removal of the cause. The only way to remove the cause is, of course, by regulation of the diet. While the cause is being removed, the symptoms must, however, be relieved, if possible. It is advisable to try the simplest remedies first. These are hot applications to the abdomen and hot water by the mouth. If these measures are not effective, a quarter or a half of a soda mint tablet dissolved in an ounce of hot water may be tried, or from two to five drops of the essence of peppermint in the same amount of hot water. Five or ten drops of Wyeth's elixir of catnip and fennel in one or two tablespoonfuls of hot water sometimes helps. An enema of warm water will almost always stop the colic, if other measures have failed. It is never advisable to give any form of alcohol for the treatment of flatulence and colic in infancy. It is never advisable and very seldom necessary to give paregoric or any other form of opium for flatulence and colic in infancy. If given, they should be used only occasionally, never regularly. Flatulence

and colic are often due to the swallowing of air during the act of nursing, whether from the breast or bottle. The swallowed air tends to collect in the fundus after the stomach is partially full and the air cannot reach and be discharged from the cardiac orifice, the mechanical condition being essentially the same as in the trap of the ordinary toilet. If the baby is picked up from time to time during the nursing, held upright and its back patted, the air can escape and flatulence and colic will be prevented.

There is little or no place for the so-called "digestants" in the disturbances of digestion in infancy. They act only as placebos. There is almost never a deficiency of pepsin or hydrochloric acid in the gastric secretion and rennin is always present. The pancreatic ferments cannot pass through the stomach without being destroyed. The only place for the digestive ferments in the treatment of these conditions is, therefore, in predigesting the foods before they are taken by the infant. The addition of lactic and other acids to the milk, which is in vogue at present, does good chiefly by preventing the formation of large, casein curds.

There is little or no absorption of fat through the skin, certainly not enough to have any appreciable effect on the nutrition. Cod liver oil is no more useful than other oils; it merely makes the baby smell worse. The only way in which inunctions of cod liver or other oils in chronic disturbances of nutrition can be of use is through the stimulation of the peripheral circulation and muscular tone incident to the rubbing.

Indigestion with Fermentation

By this term is meant a condition in which, as the result of the abnormal growth and activity of microörganisms in the intestinal contents, there is fermentation in the intestines. The term fermentation is used here in its broad sense and includes all the changes which take place in the various food elements—fats, carbohydrates and proteins—as the result of the action of microörganisms. These organisms may be the normal inhabitants of the intestinal tract or may not form part of the normal intestinal flora. It is very hard to draw distinct lines between simple indigestion and indigestion with fermentation on the one hand and between indigestion with fermentation and infectious diarrhea on the other. It is assumed that in simple indigestion there is no fermentation. This assumption, however, is not strictly true, because there is undoubtedly some fermentation going on normally and more in simple indigestion. In simple indigestion bacteria play no part in the etiology, and fermentation as the result of bacterial activity plays but a small part in either the pathology or symptomatology of the condition. In indigestion with fermentation, however, fermentation is the chief factor. It makes no difference whether the abnormal bacterial activity develops secondarily, as the result of disturbances of the normal processes of digestion, or primarily, as the result of the introduction of an excessive number of bacteria, whether or not they are members of the normal intestinal bacterial flora, into the intestines. The symptoms are likewise the same when the abnormal bacterial activity is the result of a change in the normal relations of the bacteria to each other from a badly balanced diet. Whatever the cause, the symptoms are due almost entirely to the presence of the abnormal products of bacterial activity. It is also assumed that in indigestion with fermentation there are no pathologic lesions in the intestinal wall, while in infectious diarrhea there are severe, characteristic intestinal lesions. The distinction between these conditions is, however,

probably not quite so definite, as there are undoubtedly minor changes in the intestinal wall in severe cases of indigestion with fermentation.

Etiology.—It is very difficult to make any positive statements, not only as to what organism or organisms are causing the trouble in a given case, but also as to what organisms may in general cause excessive fermentative changes. There is some evidence to show that butyric acid bacilli, the B. acidophilus and the B. putrificus may cause abnormal fermentation in the intestinal contents. So also may the colon bacillus under certain conditions. The normal lactic-acid forming organisms of the intestinal flora may also, if they are unduly active, cause abnormal fermentative changes. The B. perfringens has been shown more conclusively than any other to be a cause of fermentative diarrhea. It is very difficult to draw any positive conclusions from the microscopic examination of the stools as to what bacteria may be at fault. Furthermore, it is not safe to draw positive conclusions as to what bacteria are most active in the intestinal contents from those found in the feces. Far more definite and practical conclusions can be drawn as to the type of organisms causing the trouble from an examination of the stools and especially from their reaction. The important point is not just what special kind of bacteria is the cause, but whether this organism thrives on carbohydrate or protein media.

Pathology.—The pathologic changes in the intestines in indigestion with fermentation are comparatively slight. In most instances there is probably nothing more than a little injection of the mucous membrane with an increase in the secretion of mucus and perhaps slight desquamation of the superficial epithelium. Even in the severe cases the process does not get beyond that of a mild catarrhal inflammation.

In the severe cases of indigestion with fermentation there are more or less degenerative changes in the parenchymatous organs, especially in the liver and kidneys. These changes are not infrequently fatty. True inflammation of the kidneys is, however, uncommon. Secondary infections of other organs, especially of the middle ears and lungs, as the result of general weakening of the resistance, are not uncommon in the severe cases.

Symptomatology.—Indigestion with fermentation is much more often acute than chronic. When it is acute, there is almost always some elevation of the temperature, not infrequently as high as 104° F. or 105° F. The temperature seldom continues very high for more than three or four days, although it may be several days longer before it reaches normal. It is presumable that the fever is due chiefly to toxic absorption. It is possible, however, that it may be due in part to dehydration. It is usually not elevated in chronic cases.

The appetite is usually impaired. Vomiting is not a common symptom. There is nothing characteristic about the vomitus, if there is vomiting. There is likely to be considerable flatulence and colic. The abdomen is usually, but not always distended. Loss of weight is almost always rapid in the acute cases.

The most important symptom in all cases, whether acute or chronic, is diarrhea. This is more marked, of course, in the acute cases. The stools are loose and watery. Every other characteristic depends upon which of the food elements is being attacked by the microörganisms which are the the cause of the trouble in the individual case. In the vast majority of instances the fermentation is due to organisms which produce fermentative changes in carbohydrates and to a less extent in fats. The stools are, therefore, usually green in color, strongly acid in odor and reaction and

irritating to the skin. They are likely to contain a considerable amount of mucus as the result of the irritation of the intestinal mucosa by the acid intestinal contents. They are often frothy and not infrequently contain many small, soft, fat curds. When the disease is caused by the abnormal activity of organisms which thrive upon proteins, the stools are more often yellow or yellowish brown than green. They are alkaline in reaction and have a foul odor. If very alkaline they irritate the skin of the buttocks in the same way as acid stools. They seldom contain curds and, as a rule, not much mucus. In some instances the stools are dark brown, alkaline and have a peculiar musty odor.

There is almost always a moderate polynuclear leucocytosis. It is usually not above 20,000, but in very severe cases may be higher. In the very severe cases there may be no leucocytosis, however, because the system is overwhelmed by the toxemia.

The urine is usually diminished as the result of the loss of fluid through the bowels and the diminution in the intake. In severe cases it not infrequently shows the evidences of acute degeneration of the kidneys. Acute inflammation of the kidneys is very unusual. The urine rarely contains sugar, unless there is marked toxemia or very large amounts of sugar are being ingested.

In the very severe and fatal cases certain symptoms are likely, just as in infectious diarrhea, to develop. These symptoms are uncontrollable vomiting, marked prostration, hyperpyrexia and symptoms of irritation of the nervous system. These symptoms are probably due in part to toxic absorption from the intestines. They are also probably due in part to dehydration and in part, perhaps, to a secondary acid intoxication resulting from functional inability of the kidneys from dehydration.

Diagnosis.—Indigestion with fermentation may be confused with simple indigestion and infectious diarrhea. It is often very difficult to distinguish between simple indigestion and indigestion with fermentation, because there is a certain amount of fermentation in the intestinal contents as the result of bacterial activity in all but the mildest cases of simple indigestion. The border line between them is, therefore, very indefinite and must often be arbitrarily drawn. Furthermore, the symptoms of simple indigestion of the various food elements are very similar to those of fermentation of these same elements as the result of abnormal bacterial action, the difference between them being largely in degree. This makes it still more difficult to draw the line. It has to be drawn on the relative severity of the symptoms in general and especially on the degree of the evidences of fermentation. When these predominate the picture, the diagnosis of indigestion with fermentation is justified. In general, moreover, all the symptoms are more marked in indigestion with fermentation than in simple indigestion.

Only severe cases of indigestion with fermentation are likely to be confused with infectious diarrhea. The temperature may be high in both. There may be marked evidences of toxic absorption in both and considerable amounts of mucus in the stools in both. The most important single symptom in the diagnosis is probably the temperature curve, the elevation of temperature in severe cases of indigestion with fermentation being as a rule high and of short duration, while in infectious diarrhea, although not usually very high, it is constant and continuous. The stools show, as a rule, more evidences of fermentation in indigestion with fermentation than in infectious diarrhea and never contain blood, as they

do in infectious diarrhea. In a certain number of cases, however, it is impossible to make a positive diagnosis without a bacteriologic examination of the stools or by an agglutination test.

Prognosis.—The cases in which the evidences of carbohydrate fermentation predominate are usually milder than the other types and yield fairly readily to rational treatment. Those cases in which the stools are watery and dark brown, with a musty odor, are always serious. A high temperature is not of itself of especially bad prognostic import. Neither are the presence of considerable amounts of mucus in the stools or of albumin or other evidences of degeneration of the kidneys in the urine. A low leucocyte count is more serious in a severe case than a high count. Repeated vomiting, marked prostration, and evidences of irritation of the nervous system are of serious import. Recovery is the rule, however, in acute cases, even when the symptoms are very marked, if they survive the first three or four days. Chronic cases almost always recover, but are likely to drag on for a long time.

Treatment.—The intestinal tract should be at once thoroughly cleaned out in all acute cases of indigestion with fermentation. Castor oil is the best drug. Not less than two teaspoonfuls should be given. It should be given plain. If it is vomited, another dose should be given. If the second dose is vomited, calomel should then be tried in doses of one tenth of a grain combined with one grain of bicarbonate of soda. A dose should be given every half hour until one and one half grains have been given. This should be followed in two or three hours by two or three teaspoonfuls of the milk of magnesia.

All food should be stopped for from twelve to twenty-four hours. It is not safe to continue the period of starvation longer than twenty-four hours when the organisms which are causing the trouble are of the proteolytic type, because the intestinal secretions are protein in nature and, therefore, provide a suitable culture medium for proteolytic bacteria. There is no objection to a longer period of starvation when the microörganisms are of the types which thrive on fats and carbohydrates, if, for any reason, it is indicated. It is necessary, however, to give water freely during this period, because, although babies bear temporary starvation well, they cannot get on without water. At least as much water should be given as the baby would ordinarily take of liquid in the form of food in the given time. The water may be given either warm or cool. It may be sweetened with saccharin, if desired. It may be given in the form of weak tea, either with or without saccharin. If the baby will not take it in any other way, it should be given through a tube. If it is vomited and the condition is a serious one, the water should be given subcutaneously, intraperitoneally or even intravenously.

The primary object in the treatment of indigestion with fermentation is to destroy or inhibit the activity of the microörganisms which are the cause of the disease. It is, of course, useless to attempt to destroy these bacteria by giving drugs by the mouth. It is impossible to give large enough doses to have any effect on the pathogenic bacteria without poisoning the baby. Furthermore, if the drugs did have any action, it would be exerted upon the antagonistic as well as upon the pathologic bacteria and would, therefore, be just as likely to do harm as good. It is possible that the salts of bismuth may diminish the severity of the symptoms to a small extent. They do not, however, have any curative action. If they are used, they should be given in doses of from ten to twenty grains every two hours. It is better to use the subcarbonate or

the milk of bismuth than the subnitrate, because of the danger of nitrite poisoning when the subnitrate is used in large doses. It is useless to attempt to get rid of the pathogenic bacteria by irrigation of the bowels, because the fluid used in irrigation does not reach higher than the ileocecal valve, while the seat of the trouble is in the small intestine.

It is possible, however, to inhibit the activity and diminish the number of the pathogenic organisms in some instances by the administration by the mouth of antagonistic bacteria. It is doubtful if it is ever possible to destroy them entirely in this way. Tissier has shown that the B. bifidus has an antagonistic action on the B. perfringens and there is considerable evidence to show that the lactic-acid forming organisms are antagonistic to the organisms of the gas bacillus group. There seems little doubt, moreover, that organisms of the lactic acid group have an inhibitory action on proteolytic bacteria and proteolytic on the lactic acid group. The B. acidophilus also has an antagonistic action on proteolytic bacteria. Too much should not be expected, however, from the use of bacteria for this purpose. When there are evidences, however, of abnormal activity of proteolytic organisms, it is advisable to give the B. acidophilus or some organism of the lactic acid group. It is much better to give these organisms in some form of milk in which they have been cultivated than to give them in broth cultures or in tablets. The tablets are likely to be inert and relatively small numbers of the organisms are given in broth cultures, while immense numbers are given in food in which they have been grown. Furthermore, the lactic acid which is formed by them in the milk probably also has a favorable effect. It must not be forgotten that when buttermilk and ripened milk mixtures are pasteurized or boiled the organisms which they contain are killed and, therefore, can do no good.

The best way to diminish the number of organisms causing indigestion with fermentation and to inhibit their activity is to change the character of the food. A change in the character of the food results in a change in the character of the intestinal contents, that is, in the medium in which the pathogenic organisms are growing. If these are of the types which thrive on a carbohydrate medium, the percentage of the carbohydrates should be diminished and that of the protein increased. The percentage of fat should also be diminished, because, when there is abnormal fermentation of the carbohydrates, there is very likely to be a secondary fermentation of the fat. When the organisms are of the butyric acid forming type, the percentage of fat should be much diminished, that of the carbohydrates moderately diminished and that of the protein increased. When the organisms are proteolytic, the percentage of protein should be diminished and that of the carbohydrates increased. The general principles to be followed as to the choice of carbohydrates in the treatment of indigestion with fermentation are the same as in the treatment of simple indigestion. It is evident that it is often possible to combine changes in the food with the administration of antagonistic bacteria.

Clinically, when the stools are loose, green, acid and irritating, the percentages of fat and carbohydrates in the food should be reduced and that of the protein raised. It is in cases of this type that "albumin milk," the dried milk preparations and the preparations of casein often give satisfactory results, even when given without any very intelligent understanding of why they are indicated. Beef juice, broths and albumin water may also be given because of their relatively high protein content. They have but little nutritive value, however. It must always

be remembered, moreover, that when a foreign protein is given under these conditions there is always a possibility that it may pass through the intestinal wall unchanged and sensitize the baby. This is especially likely to happen with egg albumin. Unless the fermentation is due to lactic-acid forming organisms, buttermilk and ripened modified milk mixtures containing low percentages of fat and carbohydrates and a high percentage of protein give better results than similar modifications unripened. If the fermentation is due to lactic acid organisms, the simple modifications give, of course, much more satisfactory results.

When the stools are brownish, alkaline and foul, the percentage of protein should be much reduced and that of the carbohydrates much increased. That of the fat should be kept low. Protein foods, such as beef juice, broth and albumin water, should not be given. In the beginning it is often advisable to restrict the food to starches and sugars, that is, to the cereal waters with added sugar. Ripened modified milk mixtures containing a low percentage of fat and protein and high percentages of carbohydrates usually give good results. So also does breast milk.

The treatment of the severe symptoms which develop in some cases is discussed in the treatment of infectious diarrhea and of acid intoxication secondary to dehydration.

INDIGESTION IN CHILDHOOD

Indigestion in childhood may be either acute or chronic. Acute indigestion, while not at all uncommon, is seldom a serious condition and is of relatively little importance. Chronic indigestion is probably just as common as acute indigestion. In most instances it is not a serious condition. In others, however, it is very serious and sometimes may be fatal.

Acute Indigestion

Etiology.—Acute indigestion in childhood is most often due to some gross indiscretion in diet. This indiscretion may be simple overeating. It is more often, however, due to eating one or several varieties of indigestible food. The most common cause is an excess of sweets. Acute indigestion, however, may be the result of eating when overtired, excited or hurried. It may also be due to a chill or overheating. All of these things temporarily inhibit the digestive powers.

Symptomatology.—It is hardly necessary to describe the symptomatology of acute indigestion. Everyone is familiar with it. Two main types are, however, recognizable. One in which the chief symptom is nausea and vomiting; the other in which the chief symptom is diarrhea. Both may, of course, be present at the same time. There is nothing characteristic about the vomitus, which at first consists simply of the food which is the cause of the trouble. If the vomiting continues, the vomitus then consists of the gastric secretions and mucus. It is not infrequently mixed with bile. Occasionally, when the vomiting has been very severe, the vomitus may be blood streaked or brownish. There is also nothing characteristic about the stools. They usually contain undigested food and often mucus. They may be either acid or alkaline, but are more often acid. Headache is a not uncommon symptom. Abdominal discomfort and pain are also not uncommon. There may be no fever or the temperature may be moderately or much elevated. In

some instances, especially in young children, the attack is ushered in by a convulsion. Sometimes there are repeated convulsions. In most instances the symptoms usually cease or diminish rapidly when the stomach and bowels are emptied. In other instances, however, they persist much longer.

Diagnosis.—When the onset is acute with vomiting and fever, it is much like that of the acute febrile diseases. When it begins with diarrhea the diagnosis is usually at once evident. The acute diseases with which acute indigestion is most likely to be confused at the onset are tonsilitis, meningococcus meningitis, infantile paralysis, scarlet fever and pneumonia. It is hardly necessary to go into a detailed differential diagnosis between acute indigestion and these diseases. In acute indigestion there is usually a history of a definite indiscretion in diet, of overfatigue or overexcitement, or of chilling or overheating. Such a history does not, however, prove that the trouble is simply indigestion, because these things may be simply coincidental with the onset of one of the other acute diseases. If there is something more the trouble than acute indigestion, a careful physical examination will usually show some evidence of some other disease. In tonsilitis and scarlet fever, for example, the throat will be reddened and the tonsils enlarged, while in scarlet fever the tongue will show the characteristic changes. In pneumonia the rate of the respiration is increased out of proportion to that of the pulse, while it is not in indigestion. In meningitis there are almost always some physical signs of irritation of the central nervous system. In infantile paralysis there is likely to be hyperesthesia and slight stiffness of the back. The diagnosis of indigestion is, therefore, made in part on the history of some cause for indigestion, but more by exclusion of other diseases.

Acute indigestion is often mistaken for acid intoxication or, rather, acute indigestion is carelessly or thoughtlessly called acid intoxication. The differential diagnosis between acute indigestion and acid intoxication is taken up in the discussion of acid intoxication.

The diagnosis between acute indigestion and other diseases is usually soon evident, because improvement is usually rapid in acute indigestion, while other symptoms develop and the disease progresses in the other diseases.

Treatment.—In the type in which vomiting is a marked symptom, Nature fortunately empties the stomach without help. If there is any question as to whether the stomach has been thoroughly emptied or not, an emetic should be given. One of the best is the wine of ipecac. One or two teaspoonfuls should be given in order to empty the stomach quickly and thoroughly. There is no danger of giving an overdose, because it is vomited before it has time to cause much irritation. A small dose is far more likely to irritate the stomach than a large one. Another method is to give a glass of warm water in which a teaspoonful of bicarbonate of soda has been dissolved. This may be repeated several times. I have never found it necessary to give mustard and water or other powerful emetics. It is seldom necessary to wash out the stomach of children. It is not difficult to do, however, if it seems advisable.

If there is diarrhea, a cathartic should be given at once. Castor oil is the best, because it is the most active and the least irritating to the intestines. The dose is one or two tablespoonfuls. Castor oil does not taste badly. There is no reason why it should not be given plain. The reason that children object to it is usually because it has been suggested

to them by their elders that it is a nasty drug and that they ought to dislike it. It is easier to take when it is cold. It is also advisable to give a little orange juice or lemon juice after it. It is useless to try to disguise its taste by mixing it with other things. If children have not been properly brought up and trained to do what they are told, they should be held, their nose pinched and the castor oil given to them. If castor oil is vomited, as it sometimes is, one of the preparations of senna may be used in its place. Calomel may also be used. The dose for a child is one quarter of a grain combined with one grain of the bicarbonate of soda, given every half hour until one and one half or two grains have been given. It should be followed in two or three hours with a saline.

If there is no diarrhea, it is usually advisable to give a cathartic, or, at any rate, a laxative in order to get whatever undigested food may have passed through the stomach into the intestines out of them. In such cases milder laxatives, such as milk of magnesia, in doses of one or two teaspoonfuls, or one of the other salines, may be used. It is also advisable in acute indigestion, if the bowels have not moved well, to give a large soap-suds enema in order to wash out the lower bowel.

It is advisable in all cases of acute indigestion to stop food temporarily. The length of time depends, of course, upon the severity of the symptoms and especially upon the amount of vomiting. It is a good plan in every case to stop it long enough so that the child realizes that overeating and indiscretions in diet mean hunger later. If there is persistent nausea and vomiting, a teaspoonful dose of a solution of one level teaspoonful of bicarbonate of soda in a glass of water every ten or fifteen minutes is often helpful. Water should be given frequently. It is not advisable to give orange juice. It is still less advisable to give grape juice or ginger ale, things which a child ought not to have when it is well, much less when it is sick. The best food to give at first is usually clear broth and dry toast, which corresponds to the tea and toast which the old ladies take when they have indigestion. In more serious cases whey is often useful as the first food. In other instances diluted skimmed milk may be given first. Lime water or citrate of soda may be added to it, or any of the things done which tend to prevent the formation of large, casein curds. The next things which may be given are junket, gruels, milk toast and the simple cereals. An egg may be given later, meat, vegetables and fruit still later. The rapidity with which the diet can be increased depends, of course, very largely on the severity of the symptoms in the individual case.

In all cases of acute indigestion the child should be put to bed and kept quiet. If there is much vomiting or headache, the room should be darkened and kept cool. An ice bag to the head often relieves the headache. If this is not sufficient, the bromides may be given either by mouth or rectum, according to the circumstances. If the temperature is high, it should be treated as in other conditions. The treatment for convulsions and other marked nervous symptoms is described in the treatment of convulsions.

Chronic Indigestion

Etiology.—Chronic indigestion in childhood is almost never a primary condition, but is due to a disturbance of the equilibrium between the digestive powers and the work to be done in digestion. The equilibrium may be disturbed either by a decrease in the powers of digestion or by an increase in the work to be done in digestion. The decrease in the powers

of digestion, when there is no disease of the digestive tract, may be due to the action of diseases other than those of the digestive tract or to overfatigue, either physical or mental. The increase in the work to be done in digestion may be due to too much digestible food, to indigestible food or to improper methods of eating.

Chronic disease of the tonsils or of the accessory sinuses is a very frequent cause of indigestion, probably more often than any other disease. An unsuspected pyelitis is also not an infrequent cause. Inflammation about the teeth and gums is a less common cause than in adult life. Overfatigue is an even more common cause of chronic disturbances of digestion in childhood than are diseases outside of the digestive tract. The whole difficulty in many instances is due to overplay, overstudy, overexcitement or too late hours. It hardly seems necessary to take up all the possible causes of physical and mental overfatigue in childhood.

Improper food is a very common cause of chronic indigestion. The most common error in the diet is an excess of sweets. Unfortunately, the average layman and many physicians have no conception of what constitutes a reasonable diet either for a child or for themselves. Many children "get by" without having indigestion. Many others, unfortunately, do not. A good many children overeat. Some of the reasons that they overeat are that they eat hurriedly, do not chew their food properly and wash it down with various liquids before it is properly masticated. All of these bad habits, of themselves, are also causes of indigestion. Other causes of indigestion are eating when tired, nervous or excited. Still other causes are irregularity in eating and eating between meals. Eating between meals is especially harmful when the food taken consists of candy, ice cream, soft drinks, cookies and cake, all of which are not only indigestible, but tend to spoil the appetite for proper food at the proper time. Indigestion in childhood is much less often due to an excess of a single food element than it is in infancy, except in the case of sugar. Even when it is due to sugar, however, the symptoms are almost never as characteristic as in infancy. Indigestion seldom develops simply from an excess of fat or starch in the food. Nevertheless, in severe cases there is often a very marked intolerance for starch or fat. It is to these severe cases with marked intolerance for starch, fat or both that the name of "coeliac disease" is applied. I object to the use of this term in this way, because those who use it lump all severe chronic cases of indigestion together under this head and fail to distinguish between the cases in which there is a fat intolerance and those in which there is an intolerance for starch. The English, who first used this term, described a condition, far more common at that time in this country as well as in England than at present, which was due to an intolerance for fat. Now, almost all of these cases show a marked intolerance for starch. Fermentation plays a rather less important part in chronic disturbances of the digestion in childhood than in infancy. In almost all of the serious cases, however, there is fermentation in the intestinal contents as the result of abnormal bacterial activity. This is almost always marked in the severe cases. It may take place in any of the food elements, fat, carbohydrates or protein. It is most often in the carbohydrates, next in the fats and least often in the proteins.

Symptomatology.—All types of chronic indigestion in childhood have certain general symptoms in common, such as failure to gain properly in or loss of weight and other manifestations of disturbed nutrition. Among these are dryness of the skin and hair, cold extremities, pallor, irritability,

peevishness and disturbed sleep. Children with chronic indigestion not infrequently sweat profusely at night. They often do not get on well at school. They are easily tired and not able to keep up with their playmates. Other symptoms, which vary according to the type of indigestion, are diarrhea, constipation and the alternation of constipation and diarrhea. The abdomen may be normal in size, distended or sunken. There may or may not be vomiting. Fever of varying degrees may or may not be present. In the mild cases the stools are not characteristic. In the severe cases, however, the character of the stools varies with the type of indigestion.

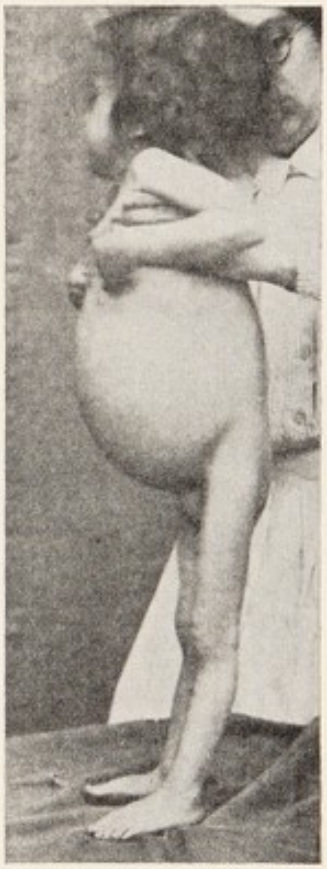

FIG. 69.—Distention of abdomen in chronic indigestion.

Diagnosis.—The diagnosis of chronic indigestion ought not be made until all other diseases have been excluded, disturbance of the digestion not infrequently being one of the symptoms of various diseases. Severe chronic indigestion with emaciation and distension of the abdomen is not infrequently mistaken for tuberculous peritonitis. The physical signs are sometimes very confusing, because in chronic indigestion, when there is much enlargement of the abdomen and dilatation of the intestines, if there is a large amount of fluid feces in the intestines, there may be shifting dulness and, in very rare instances, a suggestion of a fluid wave. In indigestion there are never any masses to be felt and there is never any free fluid in the peritoneal cavity. The history of the onset and the character of the stools is also rather different in the two conditions. A negative tuberculin test rules out tuberculous peritonitis, but a positive one does not, of course, prove that the difficulty is tuberculous peritonitis.

Prognosis.—The prognosis depends, of course, upon the etiology and the severity of the symptoms in the individual case. When the indigestion is due to some disease outside of the digestive tract, which can be cured, or to some error or errors in the daily routine, which can be corrected, the outlook for a rapid recovery is very good. It is also good when the cause is improper food or improper methods of eating, which can be corrected. Recovery is the rule, even in the more severe cases, in which an intolerance for one or more of the individual food elements has been established. Occasionally, however, in spite of treatment a child dies. In any case, improvement is always slow and almost certain to be interrupted by relapses. Many months and sometimes years are required before the lost tolerance can be reëstablished. Improvement is about equally slow in the cases in which there is a marked intolerance for starch and in those in which there is a marked intolerance for fat.

Treatment.—The first and most important element in the treatment of chronic indigestion in childhood is the discovery and removal of the cause. The first thing to be done is to find out exactly what the child eats, when it eats it, and how it eats it. The next is to find out exactly how its time is occupied, what its school hours are, how many extras it has, how much it plays, when it goes to bed, whether it takes a rest during the day or not. Other points to be found out are whether it is overtired

physically, whether it is overworked at school or has too many extras, whether it is overexcited, and whether it is worried by its work at school or by its home surroundings. There are also many other details of its life which must be gone into. The next thing to do is to make a careful general physical examination, including the throat, the accessory sinuses, the teeth and the urine, to determine whether there are any diseased organs which can account for the disturbance of the digestion.

The treatment, of course, consists in the removal of all those causes which have been found which may weaken the digestive powers. Diseased tonsils, adenoids and teeth should be removed. Diseased sinuses should be treated. Treatment should be instituted for pyelitis or any other diseases which have been found. The daily life must be carefully regulated so that the child is no longer overfatigued physically, mentally or nervously. Proper methods of eating must be enforced. Children must be made to eat slowly, chew properly and never allowed to wash down their food. They should be made to rest for a time before eating and not allowed to eat when tired, excited or nervous. Eating between meals should be stopped. A rational diet for the age should be adopted. In the vast majority of instances this is all the treatment that is necessary.

In the severe cases, however, in which an intolerance for one or more of the food elements has been established, the only way in which it is possible to regulate the diet satisfactorily is by the findings of examinations of the stools. Something can be told from the condition of the bowels, the odor of the breath, the condition of the tongue, the presence or absence of gas, nausea or vomiting, and from the history in general as to the type of indigestion in these cases. An accurate diagnosis, however, can only be made from an examination of the stools. The macroscopic examination is often sufficient to show what the trouble is. It should be verified, however, by a microscopic examination. The methods for the examination of the stools have been explained elsewhere. So also have been the characteristics of the stools in different types of indigestion. The characteristics of the stools in the different types of indigestion are much the same in indigestion in childhood as in infancy. When there is an intolerance for fat, however, the stools in childhood are more often loose, frothy, gray and very acid than dry, hard and gray or white. They are more likely to contain mucus than in infancy. The fat is seldom in the form of neutral fat, but most often in the form of fatty acids or soap, the largest proportion usually being in the form of fatty acids. In indigestion due to an intolerance for starch the stools are almost always very large and loose. They are usually a light yellow-brown, but are sometimes green and sometimes gray, especially when there is a secondary intolerance for fat. The odor is acid, sometimes being that of acetic or lactic acid and sometimes that of butyric acid. In the worst cases it is very strong and very peculiar, resembling that of a pigpen more than anything else. The reaction is always strongly acid. Mucus is not uncommon. They show microscopically large amounts of starch, partly broken down and partly not, and very many iodophilic bacteria.

In protein indigestion, which is very uncommon, the stools are loose, brownish and alkaline in reaction. The odor is foul or musty. They usually do not contain mucus.

Little additional information can be obtained from the bacteriologic examination of the stools in chronic disturbances of digestion in childhood. The intestinal flora is, of course, either fermentative or putrefactive,

that is, one which forms acids or alkalies as it grows in the intestinal contents The type of the intestinal flora can always be determined from the reaction of the stools. In the acid stool of carbohydrate indigestion, however, the presence of large numbers of organisms of the gas bacillus group is of some importance in indicating the form of treatment.

There is no place for the so-called "digestants" in the treatment of chronic indigestion in childhood. There is probably never an insufficiency of either hydrochloric acid or pepsin and, as pancreatin is destroyed in the stomach, it cannot possibly be of any use when given by the mouth. Treatment, outside of the general methods which have already been mentioned, consists, therefore, in regulation of the diet to fit the digestive capacity of the individual child. The element or elements of which it cannot take care must be cut down to the point where it can take care of them. This point can be best determined by the examination of the stools. The deficiency in the calories brought about by the cutting down of one or more of the food elements must be made up by increasing the amount of the others. The amount of the element that is causing the trouble must be increased as fast as the increasing tolerance will allow. It is not enough, in severe cases of indigestion due to intolerance of one or more of the food elements, to give general directions as to the diet. The diet must be laid out explicitly and the number of grams of the offending food element allowable daily must be distinctly stated. The number of calories which the child needs must also be stated and a list showing the caloric value and the contents in grams of the various food elements in the foods allowed must be given to the parents and its use explained. In my experience, almost all parents are intelligent enough to use these tables. I have had no difficulty in getting their interest and coöperation.

There is not a great deal of difficulty in laying out a diet to contain little or no fat. The exclusion of butter, cream, bacon and other easily recognizable oils and fats is usually sufficient. In severe cases, however, it is not enough to skim the milk, it must be made fat-free by centrifugalization. The yolks of eggs and everything made with yolks of eggs must be excluded because of the fat which they contain. In the severest cases, even the small amount of fat which is present in crackers is enough to cause disturbance and to show in the stools. So far as I know, there is nothing which can be substituted for fat in the diet.

It is a simple matter to eliminate sugar from the diet, except for the milk sugar which is in milk, although it is often quite hard to make people realize that it is necessary to stop the use of sugar in cooking. Fortunately, saccharin can be used in place of sugar in the preparation of many foods, although with certain foods saccharin gives a bitter instead of a sweet taste.

It is also a simple matter to cut starch, either wholly or partially, out of the diet. It is very difficult, however, to keep a child contented on a diet which contains none or but little starch, because a large portion of a child's diet is normally made up of starchy foods. It is advisable, whenever starch is entirely or almost entirely eliminated from the diet, to give a certain amount of some form of sugar daily in order to prevent the development of acid intoxication. One of the best forms to use is corn syrup. From one to four ounces of this may be used daily either in the milk or with other foods. The caloric value of one ounce of corn syrup is 136. There are a number of substitutes for the starchy foods which are taken fairly well by most children. Most of these are made

from the various preparations of casein I have had more experience with diaprotein flour than with the others. Incidentally, a measure of diaprotein flour contains 112 calories. Cottage cheese may also be used as a substitute for some of the starchy foods. The cheese made from a pint of skimmed milk contains fifteen grams of protein, which is equal to a little more than sixty calories. A pint of skimmed milk makes three rounded tablespoonfuls of cheese.

When there is marked protein indigestion, all animal proteins, except those in milk, should be eliminated from the diet. Vegetable proteins are usually well borne.

It is always a good rule in beginning the treatment of any severe type of chronic indigestion in childhood to either entirely or almost entirely cut out the food element for which there is an intolerance and then to gradually increase it. Much more rapid progress is made in this way than by attempting to cut it down slowly. It must never be forgotten that in the very severe cases of intolerance for either fat or starch a very slight increase in the element above the child's intolerance may cause very severe symptoms and delay recovery for weeks or months.

There is undoubtedly bacterial fermentation going on in all cases of chronic indigestion in childhood. This is quite marked in most of the severe cases, evidences of fermentation being common and sometimes severe. In such cases it is, of course, impossible to know what proportion of the symptoms is due to bacterial fermentation and what proportion to disturbance of the chemical processes of digestion. It is also impossible to know whether the primary difficulty was due to bacterial fermentation or to disturbance of the chemical processes of digestion. Fortunately, it makes but little difference. Whether primary or not, however, abnormal bacterial activity must be stopped. It is impossible permanently to change the intestinal bacterial flora by giving bacteria by the mouth, although the flora may be temporarily somewhat modified, if the bacteria are given continuously. The intestinal flora can be changed by changing the composition of the food, that is, the flora may be changed from the acidophilic to the putrefactive by changing the composition of the food and vice versa. Cutting down the proportion of the carbohydrates and increasing that of the protein in the food changes the flora from fermentative to putrefactive and cutting down the proportion of protein and raising that of the carbohydrates changes it from putrefactive to fermentative. This can be proved by bacteriologic examination of the stools, but is shown equally well by the change in the reaction of the stools, the stools being acid when the bacterial flora is mainly fermentative and alkaline when it is mainly putrefactive. Organisms growing on fat have relatively little to do with fermentation in the intestinal tract, but the products of their activity increase the acidity of the stools. Treatment by regulation of the diet in order to meet the digestive capacity of the individual child meets the indications for treatment for changing the intestinal flora. Such regulation of the diet thus not only aids the weakened digestive powers, but also changes the bacterial flora.

When the organisms of the gas bacillus group are the cause of the fermentation in the intestinal contents, something may also be done to limit their activity by the administration of organisms that produce lactic acid. The best type for this purpose is probably the B. bulgaricus, which is best given in buttermilk ripened by it. The lactic-acid forming organisms are also sometimes of benefit in the treatment of putrefactive

conditions. It must be remembered, however, that these organisms cannot change the bacterial flora permanently. This can only be done by so changing the diet as to change the character of the culture medium in the intestines. In certain cases of starch intolerance it has seemed to me that the B. acidophilus was helpful. It is practically useless, however, to give it in the form of cultures. It should be used to ripen milk and the milk then given to the child. These organisms of course, do not change the bacterial flora permanently, any more than any other organisms.

There is no place for drugs, except for the temporary relief of symptoms, in the treatment of chronic indigestion in childhood. The tincture of nux vomica, in doses of one drop for each year of age, three times daily, before meals, sometimes seems to improve the appetite. All the thick, sweet preparations of malt are contraindicated in disturbances of the digestion of starch and sugar. They are unnecessary in disturbances of the digestion of fat and protein. Cod liver oil and other medicinal fats are contraindicated in disturbances of the digestion of fat. They are unnecessary in other disturbances, butter, cream and eggs being better borne and much more palatable for the child. The treatment of flatulence and colic and discomfort is along the same lines in childhood as in infancy. Finally, cure can be brought about only by regulation of the life and diet. In severe cases the most minute attention to every detail is absolutely essential. In these cases recovery is a matter of many months and often of years, while relapses are frequent. Recovery is, however, almost always possible, provided the treatment is careful enough and is kept up for a sufficiently long time.

INFECTIOUS DIARRHEA

(DYSENTERY)

Etiology.—Infectious diarrhea was formerly very common in Boston and vicinity in the summer. As the result of improvement in the local milk supply and of the campaign of education as to the care of babies it is now a rarity. Its greater frequency in hot weather is due in part to the lowering of the general resistance to infection produced by heat and in part to the fact that the heat favors the development outside of the body of the microörganisms which are the cause of the disease. In children the onset is often preceded by gross indiscretions in diet, notably the eating of green apples and green corn. Such indiscretions are, of course, not the real cause of the disease, but merely favor infection by the etiologic organism. It is fair to assume, however, that infection would not have taken place, if there had been no indiscretion in diet. In the vast majority of cases some form of the dysentery bacillus is the etiologic agent. In a smaller number of instances the infection may be by the gas bacillus and similar organisms or by a variety of other organisms, the most important of which are streptococci, the colon bacillus and the B. pyocyaneus. The symptoms produced by these different types of organisms are practically identical. It is usually impossible to determine from them which type of organism is causing the disease.

Many investigators believe that the gas bacillus and the other organisms never cause infectious diarrhea. The work of Kendall and his associates at the Boston Floating Hospital some years ago seems sufficient, however, to justify the belief that these organisms can cause the same set of symptoms as the dysentery bacilli and that epidemics in

certain years may be due to other organisms than dysentery bacilli. The results of treatment based on a varied etiology also justify the belief that not all cases are caused by dysentery bacilli.

Pathology.—The pathologic lesions are usually limited to the large intestine and the last two or three feet of the small intestine. They are almost always most marked in the large intestine. The lesions are very varied. There may be only a catarrhal inflammation. In other cases there are in addition superficial ulcerations. In others there is hyperplasia of the solitary follicles and Peyer's patches. In many instances ulceration takes the place of the hyperplasia of these structures. In still others there is a pseudomembrane formed, which may involve considerable areas. The severity of the symptoms does not always coincide with the severity of the intestinal lesions. In general, however, the symptoms are most marked in the cases in which the lesions are most serious.

There is almost invariably a hyperplasia of the mesenteric lymph nodes. This almost never, however, goes on to suppuration. There are always more or less marked degenerative changes in the parenchymatous organs, especially in the liver and kidneys. Fatty degeneration of the liver is not uncommon. True inflammation of the kidneys is unusual. Secondary infections of other organs, especially of the middle ears and lungs, by other organisms, because of the general weakening of the resistance, are not infrequent.

Symptomatology.—The onset of infectious diarrhea is usually acute. It may be preceded for a few days by symptoms of indigestion, but ordinarily there are no premonitory symptoms. The first symptom in most cases is diarrhea. The first stools are composed of fecal matter. Mucus and blood soon appear, however, and after a few hours or one or two days the stools are composed almost entirely of mucus and blood. Pus is seldom visible macroscopically until several days after the onset and in many instances it is never present. It can, however, almost always be found with the microscope. It is of no prognostic importance, however, when it is present only microscopically. Membrane is present in the severest cases. The mucus is often stained green or brown. The odor of the stools when they are made up chiefly of mucus and blood is very slight, but sometimes resembles that of wet hay. When the stools contain much pus or membrane as the result of deep ulcerative or gangrenous lesions in the intestine, the odor is putrefactive or gangrenous. The reaction of the stools is usually somewhat alkaline. The number of stools is always large, twelve, twenty-four or even more, in twenty-four hours. The stools are usually small, being often merely a stain of blood and mucus. In a general way, the larger the number of stools the smaller is each stool.

Pain in the abdomen and tenesmus are early, marked and severe symptoms. Tenesmus is especially troublesome and annoying and often keeps the patient restless or disturbed and prevents it from getting the proper amount of sleep. Prolapse of the rectum is not at all infrequent, as the result of the straining. Vomiting is not a common symptom and is seldom troublesome. The appetite is usually much impaired. Not infrequently there is a great distaste for food of any kind.

The abdomen is sometimes distended, but in the vast majority of instances is much sunken. It is not at all unusual to see the outline of the backbone through the abdominal walls after a few days. There is almost never any spasm of the abdominal muscles. There is sometimes tenderness over the course of the colon. There is usually no enlargement

of either the liver or spleen. Slight enlargement of the spleen is, however, not very uncommon. In some instances the liver becomes very large and this enlargement may develop very rapidly. The liver will sometimes enlarge enough in three or four days to reach well below the navel and to the anterior superior spine. The enlargement in these cases is due to fatty change. Loss of weight is always rapid and often extreme.

The temperature is always elevated in infectious diarrhea. It usually runs between 100° F. and 102° F., but may be several degrees higher. It is more likely to be high in the beginning than later. It is usually fairly constant, without marked intermissions or remissions, even in cases in which the stools show evidences of ulceration and suppuration. It lasts throughout the active stage of the disease.

The symptoms are, however, not always so characteristic. The number of stools may be but little increased. Mucus and blood may be scanty or even absent and there may be no tenesmus. The symptoms may be, in fact, precisely like those of severe, simple indigestion or of indigestion with fermentation. In such instances, the continued temperature is the most suggestive symptom. The real condition can only be recognized in such cases by the agglutination test or by a bacteriologic examination of the stools.

The blood almost always shows a moderate polynuclear leucocytosis, usually somewhere in the neighborhood of 20,000. It may, however, be much higher and in the severest cases, in which the toxemia is extreme and the system is unable to react, there may be no leucocytosis or even a leukopenia.

The urine is almost invariably diminished as the result of the loss of fluid through the bowels and the diminution in the intake. It not infrequently shows the evidences of acute degeneration of the kidneys. Acute nephritis is very unusual. The urine rarely contains sugar, unless the toxemia is extreme or very large amounts of sugar are being ingested.

In the most severe and fatal cases certain symptoms such as uncontrollable vomiting, marked prostration and hyperpyrexia, are likely to develop. In other instances there are marked symptoms of irritation of the nervous system. These are probably due in part to toxic absorption and in part to dehydration.

It is impossible to determine from the symptoms what form of organism is the cause of the disease in the individual case. There is nothing about the stools which will aid in the differentiation, except in rare instances the peculiar green color caused by the B. pyocyaneus. If the green color is produced by this organism, it will disappear when nitric acid is added to the stool. If it is due to bile, the characteristic colors of Gmelin's test will appear when nitric acid is added. The microscopic examination of the stools is of little assistance in differentiating the various types, unless the streptococcus is the cause, in which case it is usually present in large numbers and easily recognized. The presence or absence of the gas bacillus can be determined in from eighteen to twenty-four hours or even less. Dysentery bacilli can be found by proper cultural methods in a large proportion of cases in which they are the cause of the lesions. Repeated negative stool cultures do not, however, rule out dysentery bacilli as the cause of the trouble. Two or three days are required for an examination of the stool. The agglutination tests are not reliable until after the sixth day. To be reliable all of the different types of the organisms must be used. Blood cultures are of little use, because dysentery bacilli seldom enter the blood

stream. Baker (Journal of Immunology 1917, II, p. 453) has developed, however, an intracutaneous test for the dysentery bacillus which gives positive results in from six to eighteen hours. The tests for the gas bacillus are easy and are given below. It must be remembered, however, in interpreting the results of these tests, that the presence of a few gas bacilli does not necessarily prove that they are the cause of the disease. There is unfortunately no method for determining the presence or absence of dysentery bacilli that does not require special media and a fairly well equipped laboratory.[1]

Diagnosis.—The diagnosis between severe indigestion with fermentation and mild infectious diarrhea is sometimes quite difficult, because in severe cases of indigestion with fermentation there may sometimes be a few streaks of blood in the stools while in mild cases of infectious diarrhea there may be no blood in the stools. Indigestion with fermentation is, however, less often mistaken for infectious diarrhea than mild infectious diarrhea for indigestion with fermentation. Fever, abdominal discomfort, loss of appetite, wasting and symptoms of toxic absorption are common to both conditions, differing only in degree. They may indeed be more marked in indigestion with fermentation than in mild cases of infectious diarrhea. The most important single symptom in the diagnosis is probably the temperature cure. The elevation of the temperature in indigestion with fermentation being ordinarily either very slight or high and of short duration, while in infectious diarrhea, although usually not very high, it is constant and continuous. In many instances a positive diagnosis can only be made by a bacteriologic examination of the stools or by the agglutination test.

Infectious diarrhea may be confused with intussusception. It is usually, however, not difficult to distinguish between these two diseases. Intussusception begins acutely with pain in the abdomen and evidences of shock, while the onset of infectious diarrhea is less acute, pain is usually not present at the onset and there are no symptoms of shock. Mucus and blood usually appear sooner in the stools in infectious diarrhea than

[1] Tests for the gas bacillus: A small portion of the stool is added to a test tube of milk. The infected tube is then gradually brought to the boiling point of water in a water bath and kept there for three minutes. In this way all the bacteria not in the spore state are killed and the development of whatever spores may be present into vegetative cells is unrestrained by the presence of non-spore-forming organisms. The tube is then incubated at body temperature for from eighteen to twenty-four hours. When the gas bacillus is present the casein is largely dissolved (usually at least 80%), the residual casein is somewhat pinkish in color and filled with holes. The odor of the culture is much like that of rancid butter as the result of the formation of butyric acid by the gas bacillus. Gram stained preparations made from the milk show rather thick, short, Gram-positive bacilli, with slightly rounded ends. The fermentation is more easily observed if the milk, after being boiled, is put in a sterile fermentation tube. "Pseudoreactions" may occur in which there is some liquefaction of the casein, but the shotted appearance of the residual casein is absent and there is no odor of butyric acid (Kendall & Smith, Boston Medical and Surgical Journal, 1910, CLXIII, p. 578). Another method described by Sylvester and Hibben (Archives of Pediatrics, 1915, XXXII, p. 457) is as follows: Fill a fermentation tube and large test tube with concentrated nitric acid. Pour off the acid after three minutes and rinse with hot tap water until neutral to litmus. With a glass spatula, also soaked in acid and washed until neutral, place about 1 c.cm. of dextrimaltose and 1 c.cm. of stool in one third of a test tube of water. Boil vigorously one half minute and pour into fermentation tube, tilting back and forth to eliminate bubbles. Stopper tube with flamed cotton and place in incubator at 37° C. for twenty-four hours. Then inspect tube for gas and note amount.

If no gas is formed or the bubble is no larger than a pin head the result is negative. If there is less than one half inch of gas the result is questionable. If there is one half inch or more of gas the result is positive.

in intussusception. The stools contain no fecal matter in intussusception after the fecal matter in the intestine below the intussusception has been passed. The stools in infectious diarrhea continue to show fecal remains. Fever is common to both diseases, but is usually higher in infectious diarrhea than in intussusception. The abdomen is almost always sunken in infectious diarrhea, but likely to be distended in intussusception. There is never any muscular spasm in infectious diarrhea, usually some in intussusception. There may be abdominal tenderness in both conditions. It is seldom marked in either, however, and is not of importance in the differential diagnosis. There is never a tumor in the abdomen or rectum in infectious diarrhea, while there often is one in intussusception. The absence of a tumor does not, however, rule out intussusception. Both conditions are usually, but not always, accompanied by a leucocytosis.

Infectious diarrhea due to dysentery bacilli or other organisms may be confused with amoebic dysentery. I have never seen a case of amoebic dysentery in a child which originated in New England. The differential diagnosis in this part of the country is, therefore, unimportant.

Prognosis.—Infectious diarrhea is a serious disease in both infancy and childhood. It is more serious in infancy. The prognosis should always be a guarded one. It is impossible to know in the beginning what the result is to be. Death may occur in three or four days, but most often takes place during the second week of the disease. It may be delayed, however, for several weeks. Improvement usually begins, in the cases which recover, at the end of the first or during the second week, but may be delayed for several weeks. Recovery is almost always slow and likely to be interrupted by relapses. In some instances the disease runs into a chronic form, which may last for weeks, months or even years. Most of these cases eventually die, but some recover.

Treatment.—The first thing to be done in infectious diarrhea is to thoroughly clean out the intestinal tract. It must not be forgotten, however, that cleaning out the intestinal tract does not remove the organisms which are in the intestinal wall, and, therefore, does not reach the seat of the pathologic process. The best drug is castor oil. It works quickly, thoroughly and causes less irritation of the intestines than any other cathartic. The dose should not be less than two teaspoonfuls for a baby and two tablespoonfuls for a child. It should be given plain. Castor oil should be tried first, even if there is vomiting, because it is often retained when food and water are vomited. If it is vomited, calomel may be given in its place. The usual dose for a baby is one tenth of a grain, combined with one grain of bicarbonate of soda, every half hour, until one or one and a half grains have been given. The dose for a child is one quarter of a grain given every half hour until two or two and one half grains have been given. It is wise to follow the calomel in two or three hours with milk of magnesia or some other saline. The lower bowel should also be irrigated at once with physiologic salt solution (approximately 1 teaspoonful of salt to a pint of water).

All food should be stopped for from twelve to twenty-four hours. It is not advisable, as a rule, to withold food longer than this, unless there is vomiting. Water should be given freely during this period, however, because, although babies and young children bear temporary starvation well, they cannot get along without water. At least as much water should be given as would normally be taken in the form of food in the given time. The water may be given either warm or cool. It may be

sweetened with saccharin, if desired. There is no objection to giving it in the form of weak tea sweetened with saccharin, if it is taken better in this way. It should be given through a tube, if the baby will not take it otherwise.

The most important element in the treatment of infectious diarrhea is the diet. The character of the diet depends on the variety of microörganisms which is causing the disease. These microörganisms can be divided, as far as the determination of the diet to be used is concerned, into two groups.

a. The various forms of the dysentery bacillus and the other organisms, except the gas bacillus, which cause the disease.

b. The gas bacillus and allied organisms.

The other organisms, although of many different varieties, are grouped with the dysentery bacilli because, as regards their growth and the production of toxic substances from protein and carbohydrate media, they behave in the same way. It is so seldom that infectious diarrhea is due to the gas bacillus and allied organisms that it is safe to assume in beginning treatment that they are not the cause of the illness.

The dysentery bacillus, the colon bacillus and streptococci belong to the class of facultative bacteria. This class of organisms can thrive upon either carbohydrate or protein medium. They produce harmless products from carbohydrates and toxic substances from protein. They act upon and use up the carbohydrate material before they attack the protein, when both are present in the medium in which they are growing. The products of the breaking down of the carbohydrate material have, moreover, when produced in sufficient amounts, an inhibitory action on the development of dysentery bacilli and, to a less extent, of streptococci. It is evident, therefore, that when infectious diarrhea is caused by bacteria of this type the food should be largely carbohydrate in character. In this way the organisms are prevented from forming toxic substances and their growth is, to a certain extent, inhibited. A carbohydrate diet, however, does not prevent injury from the endotoxins of the dysentery bacillus, except in so far that it may inhibit their growth. The prolonged withdrawal of food is also contraindicated because the intestinal contents are then made up entirely of the intestinal secretions which are protein in character. Some form of carbohydrate should, therefore, be given after a few hours. Sugar is preferable to starch, because it is much more easily utilized by bacteria. Lactose is preferable to the maltose-dextrins preparations, because it is more slowly broken down during the processes of digestion and is present in the intestinal contents longer and farther down. It is probable, moreover, that a larger proportion of lactic acid is formed from milk sugar than from the other sugars, and lactic acid has an inhibitory action on the development of the dysentery bacillus. The lactose should be given in the form of a 5% or 7% solution in water. It is better to give it frequently in small amounts at short intervals than in larger amounts at longer intervals, because in this way a continuous supply of lactose is brought to the intestines. At least as much of the lactose solution should be given as the baby would take of food under normal conditions. Half as much more is advisable. There is little or no danger of producing sugar indigestion or glycosuria, if no more than this is given. The milk sugar may be given from the first in a cereal water containing from 0.75% to 1% of starch. In any case it is advisable to give the cereal waters after from twenty-four to forty-eight hours. The starch provides a little

more nourishment and, being still more slowly broken up and absorbed, favors still further the continuance of a carbohydrate medium in the intestine. If children object to the sugar solution and the cereal waters, they may be given cereals with sugar, bread, toast and crackers, There is no objection to giving clear soup or bouillon with the bread and crackers, if they like them better that way, as the protein in clear soup and bouillon is so small that it is negligible.

It is advisable to add some protein to the food as soon as possible in order to prevent excessive protein waste of the organism. Care must be taken, however, not to give so much as to neutralize the action of the carbohydrates. The attempt should be made to give enough to cover the protein needs. The best form in which to add the protein is as skimmed milk, beginning with enough to give 0.50% of protein and increasing the amount 0.50% at a time until the protein needs are covered. In order to prevent the formation of casein curds the mixture should be boiled, or, if desired, lactic acid may be added to it. No fat should be given until convalescence is well established.

Irrigations of the colon with solutions of lactose or dextrose, while theoretically indicated, are of little practical value. It is also of little use to give intravenous injections of dextrose with the idea of inhibiting the growth of dysentery bacilli which may have entered the circulation, because they almost never do. Intravenous injections of dextrose may, however, be of value in keeping up the nutrition and preventing the development of acid intoxication.

If there is no improvement or an increase in the symptoms on a carbohydrate diet, if the examination of the stools shows the evidences of a gas bacillus infection or if it is known that the given epidemic is caused by the gas bacillus, the diet should be different. The gas bacillus and allied organisms grow rapidly in the intestinal tract when there is an excess of utilizable carbohydrate in the bowels and at the same time an insufficient number of those organisms which form lactic acid from carbohydrates to produce enough lactic acid to inhibit their growth, the gas bacillus being sensitive to lactic acid. The indications, therefore, are to cut down the carbohydrates in the diet and to introduce acid-producing bacteria into the intestines. These indications are best met by using unheated buttermilk or, better, mixtures containing no fat, 3% or 4% of milk sugar and from 1.50% to 2.50% of protein, ripened with lactic acid forming organisms. It is not possible to cut out the sugar entirely, because, if this is done, the lactic acid forming organisms will have nothing on which to grow. The lactic acid already present in the food exerts an inhibitory action upon the bacilli, but, like lactic acid given by the mouth, does not have a continuous action, being rapidly broken down and absorbed. The lactic acid forming organisms in the food, however, by keeping up their production of lactic acid, have a continuous action. Pasteurized buttermilk, in which the lactic acid forming organisms are destroyed, is less valuable than raw buttermilk because there is no continuous production of lactic acid. Albumin milk is often useful in this type of infectious diarrhea. Skimmed milk mixtures to which no sugar has been added are also suitable, although less useful than mixtures ripened with lactic acid forming organisms. It is advisable to boil them in order to prevent the formation of large, casein curds.

Irrigation of the bowels once or twice in twenty-four hours is a useful procedure. The object of the irrigation is simply to cleanse the colon. It is impossible to use antiseptic solutions strong enough to have any

appreciable action upon the intestinal wall, even if this was desirable, or strong enough to have any effect upon the pathogenic bacteria without running serious risk of poisoning the baby. The irrigating solution should therefore, be mild and nonirritating, like physiologic salt solution or a 1 % solution of boracic acid. The irrigation should be given with a soft rubber catheter, #25 French, passed as high as posssible into the bowel with the patient lying on the back and the hips elevated. There is considerable doubt, however, whether irrigation with a catheter is any more satisfactory than with an ordinary nozzle. The fluid is then allowed to run in from a bag hung not more than two feet above the level of the patient. It should be allowed to run in until the abdomen is slightly distended, then allowed to run out, and so on, until the wash water returns clear. The object of the irrigation being to cleanse the colon, enough liquid should be used to do this, whether it is much or little. Irrigation should seldom be done more than twice in twenty-four hours. If it depresses or disturbs the patient, it should be given up at once, as under these circumstances it does more harm than good.

In subacute or chronic cases, in which blood and pus persist in the stools after the temperature has dropped and the evidences of toxemia have disappeared, injections of nitrate of silver are sometimes useful and seem to hasten the healing of the bowel. They seldom do good in the acute stage. The colon should first be irrigated with sterile water in order to cleanse it. Salt solution should not be used because the sodium chloride forms with the silver nitrate an insoluble silver salt which is precipitated and the action of the silver solution is consequently diminished. After the bowel has been washed out, from six to sixteen ounces, according to the age of the patient, of a 2% or 3% solution of nitrate of silver should be allowed to run into the colon and the tube then withdrawn. No attempt should be made to have the fluid either retained or expelled. These injections seldom cause any marked discomfort. If they do, the silver solution may be washed out with salt solution, or an opium suppository given. The injections should be repeated every day or every other day. If there is no evident improvement after three or four injections, it is useless to keep on with them. The first stools passed after an injection usually contain more blood and considerable dirty gray material, consisting of slough from the ulcers, intestinal secretions and pus, discolored by the silver nitrate. In favorable cases, however, there is marked improvement in the character of the stools within twenty-four hours.

The so-called intestinal antiseptics are of little or no value in the treatment of infectious diarrhea. It is impossible to give them in large enough doses to have any effect on the pathogenic bacteria in the intestines without poisoning the baby. If they did have any action, it would be exerted on the antagonistic as well as on the pathogenic bacteria. The salts of bismuth are of little value during the acute stage. During the chronic stage they sometimes seem to diminish the peristalsis. If used, they should be given in doses of from ten to twenty grains every two hours. It is safer to use the subcarbonate or the milk of bismuth than the subnitrate, because of the possibility of nitrite poisoning when the subnitrate is used.

The results of the treatment of infectious diarrhea in early life with sera are unsatisfactory. There being many strains of dysentery bacilli, the serum is, of course, useless unless it was obtained by the use of the organism present in the individual case. The chances are that it was not.

Even when polyvalent sera are used they may or may not be antagonistic to the strain in the given case.

Pain and tenesmus are often very troublesome symptoms. Injections of starch solution, of the strength of one drachm of starch to one ounce of water, to which are added from three to five drops of laudanum may be tried. They are usually not retained long enough, however, to do any good. It is usually wiser, therefore, to give the opium by mouth, if it is necessary to use it. It must always be remembered, when giving opium, that its action is to diminish peristalsis and that, if the peristalsis is diminished enough to interfere with the free emptying of the bowels, serious harm will be done. Only enough should be given, therefore, to control the tenesmus and to prevent the frequent stools due to excessive peristalsis. The safest form of opium to use is paregoric. It may be given in doses of from five to twenty drops to babies and in doses of from twenty drops to one teaspoonful to children. Dover's powder, in doses of from one eighth to one grain, according to age, may also be used. It is better to give small doses at short intervals than larger doses at longer intervals. The use of hot stupes or compresses to the abdomen will, however, often relieve the pain and tenesmus and render the use of opium unnecessary.

It is often difficult to induce the patient to take a sufficient amount of water or, if it does take it or it is given through a tube, it is vomited. In such cases physiologic salt solution should be given subcutaneously to make up the deficit. From four to six ounces may be given at a time and repeated as often as necessary. It is useless to give a second injection before the first one is absorbed. Salt solution may also be given through the bowel by seepage. Considerable amounts can sometimes be introduced in this way, even when many stools are being passed. Salt solution may also be given intraperitoneally or intravenously. It is very seldom, however, that it is necessary to use these methods.

Stimulants may be necessary as in other acute diseases. There are no special rules to be followed in infectious diarrhea. Stimulation is discussed in the treatment of pneumonia.

Babies or children that are seriously ill with either indigestion with fermentation or infectious diarrhea are quite likely to develop certain serious symptoms. One of these symptoms almost invariably develops towards the end in fatal cases. These symptoms are excessive vomiting, hyperpyrexia, symptoms of irritation of the central nervous system, prostration and collapse. Part of these symptoms are presumably due to toxemia and part to dehydration, which, in turn, may or may not be the cause of functional inability of the kidneys and a resulting acid intoxication. It is very difficult to know in a given case what is the cause of these symptoms. Furthermore, the intoxication may be due theoretically at any rate, to the absorption of bacterial endotoxins, extracellular toxins, the products of bacterial fermentation in the intestinal contents and to purely chemical disturbances of metabolism.

When any of these symptoms appear, if there is any doubt as to whether the bowels have been thoroughly emptied or not, it is advisable to repeat the initial catharsis and irrigation. It is also advisable, if the condition of the nutrition warrants it, to withhold food for about twelve hours or, at any rate, if the cause of the infectious diarrhea is not the gas bacillus, to go back to carbohydrate solutions. In all of these cases, unless sufficient liquid is being taken and retained by the mouth, it is advisable to give salt solution subcutaneously or by seepage. This is

especially important, if there is reason to believe that the symptoms are due largely to dehydration.

There is not much to be done for excessive vomiting beyond the general measures already given, except to withhold food entirely and wash out the stomach with a solution of bicarbonate of soda of the strength of one level teaspoonful to the pint of water. Small amounts of this same solution of bicarbonate of soda may be given at frequent intervals. The vomitus not infrequently contains brownish or reddish flakes or streaks as the result of capillary hemorrhages into the stomach. This sign is of serious, but not necessarily of fatal, import.

The hyperpyrexia is best treated by the use of cold externally. It is very seldom advisable to give the coal tar products to children to reduce the temperature. The methods for the use of cold externally are described in the treatment of typhoid fever. An ice bag may also be applied to the head. It must not be forgotten, however, that a baby's skull is very thin and that the effect of the cold is, in consequence, greater than in the adult. Great care must be exercised, therefore, in the use of an ice cap in infancy, especially when the fontanelle is open. Lowering the temperature of the liquid used in irrigating also aids in reducing the fever. It may be reduced in desperate cases to as low as 90° F.

The symptoms of irritation of the nervous system vary markedly in different cases. The patients are sometimes stupid, comatose or relaxed. In other instances they show the typical picture of coma vigil. Marked restlessness is very common. Twitching is not unusual and convulsions are not very uncommon. In many instances there are marked signs of meningeal irritation. The head may be retracted, the pupils unequal and the knee-jerks exaggerated. The picture may be, indeed, almost exactly that of meningitis, so that a diagnosis can only be positively made by lumbar puncture. The results of lumbar puncture are, however, sometimes misleading, because the cerebrospinal fluid in this condition sometimes shows a slightly positive globulin test and a moderate excess of mononuclear cells. The pathologic condition is presumably one of meningeal irritation or serous meningitis. If the fontanelle is full, a lumbar puncture should be done and often gives relief. An ice bag to the head often helps. Bromide of soda, in doses of from five to ten grains by mouth for babies and from ten to twenty grains for children, may be tried for restlessness and excitement. It may be combined with from one to four grains of chloral hydrate, according to the age of the patient. It is ordinarily useless to give these drugs by enema in these conditions, as they are almost never retained. If the bromide and chloral do not control the symptoms, morphin may be given by mouth or subcutaneously in doses of from $\frac{1}{100}$ of a grain to $\frac{1}{32}$ of a grain, always beginning with a small dose and increasing, if this is ineffectual.

Prostration and collapse should be treated as they are when they occur in other diseases. It is important to remember, however, that all forms of treatment weaken and exhaust the patient. Irrigations must be omitted and the patient disturbed as little as possible. It must be kept warm and protected in every way. There is likely to be some vasomotor paralysis and lowering of the blood pressure. Alcohol is, therefore, contraindicated. Epinephrin, in doses of from two to ten minims of the 1–1000 solution, given subcutaneously, is sometimes of considerable value. It is useless when given by the mouth. Strychnia is in general the most useful of the stimulants, while caffein and camphor are the best quick stimulants. Strychnia may be given in doses of from

1/1000 to 1/200 of a grain to a baby and from 1/200 to 1/60 of a grain to a child. The dose of the citrate of caffein by mouth for a baby is from one eighth to one half of a grain and of caffein-sodium benzoate or salicylate, subcutaneously, about the same. The dose for a child varies from one half to one grain. Camphor may be given subcutaneously in oil in doses of from one to four grains, according to the age of the patient. It must never be forgotten that the action of caffein and camphor is fleeting and that they should not be used continuously, but only in emergencies.

CHOLERA INFANTUM

Only those who have not seen the symptom-complex usually known as "cholera infantum," can have any doubt as to its existence. It has all the ear-marks of an acute, specific, infectious disease. No specific microörganism has, however, been found for it. It may be, indeed, that it is not caused by any form or forms of microörganisms, but is simply a peculiar manifestation of some unusual type of intoxication or disturbance of metabolism. It is a rare condition. I imagine that many of the younger pediatricians have never seen a case. It almost never occurs in children and never occurs except in hot weather.

Pathology.—There are practically no pathologic changes. The tissues are all drained of their liquid. There are no lesions of the intestines beyond moderate hyperemia of the mucous membrane or desquamation of the superficial epithelium. Sometimes there are evidences of cerebral hyperemia or of edema, but these are usually lacking. The kidneys show evidences of degeneration, but no changes sufficient to account for the symptoms. The other parenchymatous organs also show degenerative changes. There are no data with which I am acquainted to show whether there is an acid intoxication or not.

Symptomatology.—Clinically, the symptoms are best explained by the action of some toxic substance upon the heart and nervous system, the vasomotor nerves of the intestines being especially affected, and by the draining of fluid from the various organs. The onset of the disease is usually preceded by symptoms of indigestion, but it may develop without any premonitory signs. The development of the symptoms, when they once appear, is extremely rapid, so rapid, indeed, that a baby may be moribund in five or six hours. The first symptoms are usually restlessness or prostration, with abdominal discomfort and a rising temperature. Vomiting begins in a few hours and is accompanied or quickly followed by profuse diarrhea. The first vomitus and stools are made up of whatever happens to be in the stomach and bowels at the time of the onset. After that the vomitus and stools are composed almost entirely of serum. The vomitus is often blood stained. The stools are large, watery, almost colorless and without odor. The reaction is usually acid in the beginning, but quickly becomes neutral and then alkaline. Microscopically they show large numbers of epithelial cells, a few leucocytes and very many bacteria. There is sometimes considerable tenesmus, but, in most instances, the sphincters are relaxed and the liquid simply runs out of the bowel every few minutes. There is no tenderness in the abdomen and no spasm of the abdominal muscles. The abdomen is usually sunken. The tongue is dry and red. Emaciation is very rapid, because of the loss of fluid from the tissues. The face is pinched, the eyes sunken, the skin dry and the fontanelle depressed. Thirst is very marked. The secretion of urine is much diminished. It is concentrated and highly acid. It

usually contains albumin and sometimes casts and blood. Presumably because of the accumulation of blood in the abdominal organs as the result of the vasomotor paralysis of the abdominal vessels, there is interference with the peripheral circulation. The extremities become cold, the skin pale, and sometimes cyanotic. The surface temperature is usually, but not always, low. The rectal temperature is high, ranging from 103° F. to 104° F. In fatal cases it may reach as high as 108° F. The pulse is rapid from the first and soon becomes feeble and irregular. The respiration is usually rapid and irregular, but is at times slow or sighing. It may be of the Cheyne-Stokes or Biot types. I have never seen the rapid, deep respiration of acid intoxication. In the beginning the baby is usually restless and whimpers constantly. After a time it becomes either listless and stuporous or symptoms of cerebral irritation develop. The head is retracted. There may be local paralyses. The extremities are rigid, and twitching and convulsions develop.

Prognosis.—The prognosis is very grave. Recovery is most unusual. Death usually occurs during the first forty-eight hours after the onset. The disease is, however, apparently self-limited and, if the baby survives for two or three days, it usually recovers. Recovery is usually surprisingly rapid after it begins. Sometimes, however, the acute symptoms may be replaced by those of various types of indigestion. Sclerema sometimes develops under these conditions. Recovery may take place even then, but death usually finally takes place from inanition.

Treatment.—It is evident that in such a rapid and fatal disease treatment must be immediate and vigorous. It is probable that there is a vasomotor paralysis of the gastrointestinal vessels. Hence, food and drugs introduced into the alimentary canal cannot possibly be absorbed. They can do no good and perhaps may do harm.

It is useless to give purgatives in this condition as Nature is doing all the purging, and more, that is necessary. It may perhaps be of advantage, however, to wash out the stomach and irrigate the colon in the beginning.

It is most important to restore fluid to the tissues which are being so seriously drained. It is idle to suppose that fluids given by the mouth will be retained. They are almost invariably vomited at once. Cold sterile water in small amounts may be tried, however. It is also useless to attempt to supply fluid to the tissues by enemas or by seepage. It is immediately expelled. The best method of introducing fluid into the system is by the injection of physiologic salt solution subcutaneously. It should be given in doses of from four to eight ounces at a time, the dose being repeated as soon as the fluid is absorbed. It may be given intraperitoneally or intravenously. There would seem to be no special advantage in giving glucose solution, because the symptoms are presumably not due to starvation or acid intoxication, but simply to dehydration. The injection of salt solution not only supplies fluid to the tissues, but probably helps to restore the surface circulation and perhaps assists in eliminating toxic substances from the blood.

Irrigations of cold water tend to restore the surface circulation and also to lower the temperature. The best methods for restoring the surface circulation are rubbing, mustard baths and the warm pack. These procedures, however, are not those best fitted for the reduction of temperature. For this purpose cold externally must be used. For the treatment of individual patients it is often necessary to determine whether it is the internal congestion or the high temperature which is doing the more harm and then to treat the more serious condition.

Stimulation is always necessary. As drugs given by the mouth are not absorbed, it must be given subcutaneously. The usual stimulants, strychnia, caffein and camphor, are the best. Strychnia may be given in doses of from $\frac{1}{1000}$ to $\frac{1}{200}$ of a grain, subcutaneously. The dose of caffein-sodium benzoate or salicylate is from one eighth to one half of a grain and that of camphor, one or two grains. The camphor should be given in oil. It must not be forgotten that the action of caffein and camphor is fleeting. If a continuous action is desired, therefore, they must be given at least as often as every hour. Atropin is supposed to be especially useful in these cases. I am not certain whether it is or not. It should always be tried, however. It should be given in doses of from $\frac{1}{500}$ to $\frac{1}{800}$ of a grain, repeated every two or three hours as necessary. Morphin is indicated when the diarrhea and vomiting are extreme or when the nervous manifestations are very marked. Doses of $\frac{1}{100}$ of a grain are usually sufficient. They should, of course, be given subcutaneously. Care must be exercised, however, not to give too much or to keep it up too long.

If improvement begins, stimulants and water may be given by mouth, and soon after this, small amounts of food. The best food to give in the beginning in these cases is diluted human milk, in the proportion of one part of milk to two or three parts of water. No more than an ounce should be given at a feeding. If this is tolerated, the strength and the amount at a feeding should be increased as rapidly as possible. There are no very definite indications as to what combination of the food elements should be most suitable when human milk cannot be obtained. The only thing that is certain is that the food must be a very dilute one. Whey is probably as likely to agree as anything. If this is tolerated, a dilute whey mixture containing 0.25% of fat, 5% of milk sugar, 0.75% of whey protein and 0.25% of casein may be given next. If this agrees, the percentages of fat and casein should be gradually increased. A 1% solution of starch, with milk sugar added up to 5%, is also a rational mixture in beginning feeding. If this is used first, it may be gradually strengthened by the addition of skimmed milk, which should be boiled in order to prevent the formation of large, casein curds. If this mixture is tolerated the percentages of milk sugar and protein should be gradually increased and fat cautiously added.

CHRONIC COLITIS

This condition, often called ulcerative or membranous colitis, is very rare in both infancy and childhood. It becomes somewhat more frequent, however, with increasing age. Severe chronic indigestion with fermentation is often mistaken for it. In rare instances it follows acute infectious diarrhea due to the dysentery bacillus. The chronic condition which persists is, however, almost never caused by the same organism. The infection of the colon may be by a variety of organisms. Why infection occurs is not known. Mechanical injury may perhaps play a part, while superficial lesions secondary to severe indigestion with fermentation may be the portal of entrance. Whatever the cause, the pathologic conditions and symptoms are essentially the same. They do not differ materially from those in adult life.

Symptomatology.—It is hardly necessary to describe the symptoms of chronic colitis in early life. They are those of a severe indigestion with fermentation, the type of stool depending upon the type of indigestion. In addition the stools contain a large amount of mucous and sometimes

blood, pus and membrane. In some instances the mucus is very abundant and appears as long strings or casts. Vomiting is unusual. The abdomen may or may not be enlarged. It may or may not be tender. Pain and tenesmus may or may not be present. Loss of weight occurs in all cases and may be extreme. Various disturbances of nutrition accompany the loss of weight. The temperature may be mormal, subnormal, or irregularly elevated. There may or may not be a polynuclear leucocytosis.

Diagnosis.—The diagnosis from indigestion with fermentation depends largely on the finding of blood, pus and membrane in the stools. The other condition for which it is occasionally mistaken is tuberculous ulceration of the intestines. In this condition there are almost always evidences of tuberculosis elsewhere. The evidences of indigestion in the stools are less marked and they contain, as a rule, less mucus. Blood and pus may be present in the stools in both conditions. The tuberculin test is positive in tuberculous ulceration of the intestines and tubercle bacilli are present in the stools.

Prognosis.—Chronic colitis in early life is always a serious condition. If recovery occurs, it is only after months and years, even in mild cases. Death is not uncommon in the severe.

Treatment.—Treatment consists in regulation of the diet on the same principles as in indigestion with fermentation. Bacteria may be given by the mouth for the same indications as in indigestion with fermentation. Drugs by the mouth are useless. Irrigation of the colon with various solutions should be used, as in the treatment of chronic colitis in adults. The results are likely to be equally unsatisfactory. In severe, long-drawn-out cases, in which all sorts of treatments have been tried without relief and in which a fatal outcome seems inevitable, an artificial anus should be made in the cecal region. In this way only can rest for the colon be secured and an opportunity for healing given. It is necessary to keep the artificial anus open for several years. Recovery sometimes occurs as the result of operation in cases, which, before it was done, seemed absolutely hopeless.

PROCTITIS

The term proctitis is usually applied only to those inflammatory processes in the rectum which are limited to the rectum, although, of course, the rectum is almost always involved whenever there is inflammation of the colon. The pathologic process may vary from a simple catarrhal to a membranous inflammation, and may go on to ulceration. Membranous inflammation and ulceration of the rectum are so uncommon in early life that it hardly seems necessary to describe them. Catarrhal proctitis is not infrequently due to pin-worms. The inflammatory process is, however, then seldom severe. It is often caused by the frequent use of irritating enemas or of suppositories, occasionally by the careless use of syringes or thermometers. In some instances it is due to the extension of an inflammatory process upward from the outside through the anus or to direct infection with the gonococcus or some other organism. Catarrhal proctitis is an occasional complication of measles and scarlet fever.

Symptomatology.—The symptomatology is what would be expected, namely, a feeling of heat and burning in the rectum, tenesmus, and pain on defecation. If the inflammation is severe, there is also fever, which in babies may be high. The symptomatology in babies, however, is

very much less definite. They are unable to tell how they feel and simply show that they are uncomfortable and in pain by crying or whining constantly and having frequent movements of the bowels. They are evidently sick and not infrequently have a high temperature.

The stools are either coated or mixed with mucus. If there is much inflammation, a large amount of mucus is secreted, which is not infrequently passed without feces. It sometimes takes the form of a cast. The mucus is not infrequently mixed with bright blood. Sometimes considerable blood is passed. Prolapse of the rectum is not very uncommon in infancy. A secondary inflammation of the skin about the anus is also not uncommon. Eversion of the mucous membrane often shows the catarrhal condition. It can be easily seen with the aid of a speculum. The course of catarrhal proctitis is, fortunately, usually short. It may persist, however, in a subacute form for many weeks or even months.

Treatment.—When catarrhal proctitis is due to pin-worms, the treatment consists primarily in that for the pin-worms. When they are under control, the proctitis quickly recovers. It is important to keep the bowels from becoming constipated. Care must be taken, however, not to bring on a diarrhea. Simple injections of salt solution or of any of the mild alkaline washes are usually all that are necessary to cure the condition. Injections of two ounces of starch solution, of the strength of one drachm of starch to one ounce of water, will sometimes relieve the discomfort. If there is much tenesmus, they are usually expelled, however, before they do any good. If there is marked tenesmus and discomfort, from two to five drops of laudanum may be added to the starch solution. I have had very little success with either opium or cocaine suppositories. Suppositories of one twenty-fourth of a grain of opium may be tried in babies of one year of age and of one eighth of a grain for children of four years. In long continued cases, the injection of three or four ounces of a 2% nitrate of silver solution at intervals of several days may be tried.

APPENDICITIS

Acute appendicitis is said to be uncommon in infancy, to increase steadily in frequency up to from five to seven years and from that time on to be as frequent as in adult life. I am inclined to believe that appendicitis is not as uncommon in infancy and early childhood as is usually believed and that its apparent rarity is because, on account of the indefiniteness of the symptoms and the difficulty in their recognition, it is usually overlooked unless perforation occurs and general peritonitis develops.

Etiology and Pathology.—The etiology of and the pathological changes in acute appendicitis are essentially the same at all ages. Some authors are inclined to attribute considerable importance to pin-worms in the etiology of appendicitis in childhood. It is undoubtedly true that children having pin-worms also have appendicitis, that in certain cases of appendicitis pin-worms have been found in the inflamed appendix and that occasionally pin-worms have been found in the peritoneal exudation after perforation. None of these things, however, proves that the pin-worms were the cause of the inflammation of the appendix. There is no reason why pin-worms in the colon should not get into the appendix, but finding them there does not show that they were the cause of the inflammation. It is almost certain that pin-worms are unable to cause anything more than a very superficial inflammation.

It is possible, however, that such superficial lesions may open the doors for the invasion of pathologic organisms.

The symptoms and physical signs of appendicitis in childhood are modified somewhat by the position of the cecum, which in infancy and early childhood lies wholly or in part between horizontal lines drawn parallel with the crest of the ilium and the anterior superior spine. It gradually works downward, so that in later childhood it is in the adult position. The opening from the cecum into the appendix is funnel-shaped and is relatively larger in early life than later. Drainage from the appendix into the cecum is, therefore, better. The appendix is also relatively longer and larger in early than in adult life. It is also more likely to be peculiarly situated.

The pathologic process is likely to progress more rapidly in early than in adult life. It is also more likely to extend to the peritoneum. When it does, however, it is more likely than in the adult to cause a localized inflammation instead of a general peritonitis.

Symptomatology.—The symptoms in acute appendicitis are essentially the same in infancy and early childhood as in late childhood and adult life. The difference in the symptomatology lies in the difficulty in recognizing these symptoms. The onset is usually thought to be more acute in early than in late childhood. The chances are, however, that it is not and that the apparent acuteness of the onset is because children either do not notice or do not say anything about slight or moderate discomfort. It is probable that pain is one of the earliest symptoms, if not the earliest symptom, as in adults, and it probably may be either dull or colicky in children, as in adults. Babies, while they are able to show that they have pain, cannot indicate in any way where it is. Young children are often unable to tell in what part of the body pain is and, even if they know that it is in the abdomen, they are unable to locate it. Even older children, when asked where the pain is, always put their hand in the middle of the abdomen and, if asked to point to the location of the pain, put the finger on the navel. Vomiting is also a very constant symptom. It may or may not precede pain. There is nothing characteristic about the vomiting. Constipation is more common than diarrhea, but either condition may be present. There may or may not be moderate distention of the abdomen. Other less constant symptoms are unwillingness to move, apparently on account of the pain which motion causes, flexion of the right thigh on the abdomen, pain on defecation and frequent micturition. The latter symptom is probably due to irritation of the bladder, when the appendix is situated behind it. The temperature is usually not much elevated. It may be nearly normal. If it is over 103° F., the difficulty is probably not appendicitis.

There is almost always a polynuclear neutrophilic leucocytosis. This is usually moderate, but may be slight and sometimes is marked. In my experience, it makes very little difference either in prognosis or treatment how high the white count is. If the child is not in a serious condition and there is no leucocytosis, the chances are much against appendicitis. If the child is in a serious condition, the absence of leucocytosis does not count against appendicitis.

The course of appendicitis is much the same in early life as later, although changes are likely to occur more rapidly. The inflammatory process may quiet down and go on to recovery. Localized inflammation of the peritoneum with or without abscess formation may occur. General peritonitis may develop either with or without perforation. Perforation

and peritonitis may come on insidiously or very marked symptoms may develop in the course of a few hours. When the onset of the symptoms of peritonitis is insidious, the patient is more likely to be an infant than a child and, when it is acute, a child.

Physical Signs.—The principal signs of appendicitis are localized tenderness and muscular spasm. It is absolutely necessary to gain the child's confidence in order to make a satisfactory examination of the abdomen. It must be approached quietly and handled gently. Nothing can be learned, if it is frightened. Its attention must be distracted, if possible, from the abdomen. Even under the best conditions it is often very difficult to determine localized tenderness in a child. It is as difficult for them to localize tenderness as it is to localize pain. With the best intentions, they are entirely unable to help in this way. It is often easier to determine whether there is localized tenderness or not by watching the child's face than by accepting what it says. It is usually not as hard to find out whether there is localized spasm or not, as it is not necessary to depend for this on the child's answers. Great care must be taken not to confuse general voluntary abdominal resistance with localized spasm. Even if the child cries, it is usually possible to determine whether there is spasm or not when the abdomen loosens up between cries. Localized dulness is a relatively unimportant symptom, because this is not present early. A tumor is also not present early in the disease. When there is any question as to the diagnosis, a rectal examination should always be made. The parts are so small that much more can be learned from a rectal examination in babies and young children than later. It is easier to recognize localized tenderness by rectal than by abdominal examination. A tumor can often be felt by the rectum when it cannot be felt from the outside and in certain instances, when the appendix is retrocecal, tenderness and tumor can be made out when nothing can be determined from the outside.

Diagnosis.—Appendicitis is most often mistaken in early life for some form of indigestion associated with colic or abdominal pain. The diagnosis is often very difficult. In general there is more likely to be some evident cause for indigestion than for appendicitis. The pain in indigestion, while often temporarily more acute, is seldom so persistent as in appendicitis. In indigestion there is not the same desire to avoid movement or to keep the thigh flexed as in appendicitis. There is nothing about the vomiting, the condition of the bowels, the presence or absence of distention or the temperature which helps in any way, except that, if there is no rise in temperature, the chances are in favor of indigestion. The leucocyte count is of little help, if it is slightly or moderately elevated. A normal count is in favor of indigestion; a great increase in favor of appendicitis. The diagnosis depends almost entirely on whether there are localized tenderness and spasm or not and on the findings by rectal examination.

Appendicitis may be confused with acute intestinal obstruction and intussusception. Acute intestinal obstruction, except from intussusception, is extremely rare in both infancy and childhood. The chances are, therefore, always in favor of appendicitis. The symptomatology of intussusception is so characteristic that no confusion should occur. Localized tenderness and rigidity are uncommon in intestinal obstruction and always present in appendicitis. Abdominal distention is more common in obstruction and the vomiting is often fecal, while it never is in appendicitis. The symptoms of toxemia are much more marked in intestinal obstruction than in appendicitis.

Appendicitis is very often confused with lobar pneumonia, because children with pneumonia are very likely to refer pain in the chest to the abdomen and to have distention of the abdomen either as the result of toxemia or of diminished motion of the diaphragm. The differential diagnosis is often extremely difficult. The onset in pneumonia is usually more acute. The temperature is almost always higher. A temperature of 103° F. or over is strongly in favor of pneumonia. The rate of the pulse and the respiration rise together in appendicitis. In pneumonia, while both rise, the increase in the rate of the respiration is out of proportion to that in the rate of the pulse. This is probably the most important single point in the diagnosis between these two conditions. In rare instances, however, the rate of the respiration may be increased disproportionately in appendicitis as the result of distention of the abdomen or of beginning peritonitis, while occasionally the rate of the respiration is not much increased in pneumonia. In pneumonia there may be general abdominal tenderness and spasm of the abdominal wall. It is sometimes more marked on the right side. It is never sharply localized, however, as in appendicitis. Rectal examination is negative in pneumonia, usually positive in appendicitis. Less important points are the presence of cough in pneumonia and its absence in appendicitis, a degree of leucocytosis higher on the average in pneumonia than in appendicitis, and a greater use of the abdominal muscles in respiration in pneumonia than in appendicitis. A diminution of the movement of the chest and of the respiratory sound on one side is strongly in favor of pneumonia. A positive diagnosis of pneumonia can usually be made, if necessary, with the Roentgen ray. It must not be forgotten, moreover, that a child may have both pneumonia and appendicitis at the same time.

Acute inflammation of the retroperitoneal, and sometimes of the mesenteric lymph nodes, is not infrequently mistaken for appendicitis. Appendicitis is almost never mistaken for inflammation of these nodes. The inflammation of the nodes may be either tuberculous or non-tuberculous. In acute cases it is usually non-tuberculous. In sub-acute or chronic cases it is usually tuberculous. The differential diagnosis is always difficult and often impossible. Inflammation of the lymph nodes is not as likely to be associated with vomiting and disturbance of the bowels. If it is tuberculous, there is usually no leucocytosis. The physical signs are, however, the same in both conditions. If there is any doubt as to the diagnosis, it is wiser to operate, because no harm is done, if the trouble is inflammation of the nodes, while great harm may be done, if operation is not done and the trouble is appendicitis. It is probable that the abdominal pain which is often present in throat infections is due to a coincident inflammation of these lymph nodes. Appendicitis is, however, a not very unusual complication of inflammation of the throat.

The symptoms of acute pyelitis at times resemble very closely those of appendicitis. If the appendix is situated behind the bladder, there is often frequent micturition, which suggests strongly infection of the urinary tract. In pyelitis the tenderness is usually over the kidneys, but sometimes is over the ureters in the same situation as that in appendicitis. In appendicitis there is almost never any tenderness or muscular spasm over the kidneys. In pyelitis the urine, of course, contains pus and usually bacteria. In appendicitis the urine does not contain pus and bacteria. Occasionally, however, there may be a complicating pyelitis with appendicitis. Rectal examination is usually of great help in the diagnosis.

A psoas abscess may cause pain, tenderness and muscular rigidity in the right lower quadrant with flexion of the thigh. The onset of the symptoms is, however, slower than in appendicitis. There is usually no vomiting or disturbance of the bowels. Careful examination, moreover, shows that there is disease of the spine or of the hip. If this disease is tuberculous, the leucocyte count is usually not elevated. This condition should not be mistaken for appendicitis, if reasonable care if used in the physical examination.

Recurrent attacks of appendicitis are sometimes confused with attacks of recurrent vomiting. They ought not be, however, because in recurrent vomiting there is no abdominal pain, tenderness or spasm and the abdomen is sunken, not distended. Rectal examination is, moreover, negative. The temperature is usually but little elevated in recurrent vomiting and there is not likely to be a marked leucocytosis. The only points in common are the vomiting and constipation. The vomiting is the one marked symptom in recurrent vomiting. It is a relatively slight one in appendicitis.

Prognosis and Treatment.—In the first place, if there is any reason to suspect that an infant or a child may have appendicitis, it should not be given a cathartic. There is nothing which can be done which will do more harm in appendicitis than giving a cathartic to move the bowels. It is of advantage, however, to empty the colon from below with enemas. All food should be stopped at once. Water may be given, however, freely. When the diagnosis of appendicitis is made, an operation should be immediately performed. It is true that in many instances the inflammation will quiet down and the operation can be done later. On the other hand, however, it is impossible to tell whether the inflammation will quiet down or not. If it does not, the chances of a successful operation a little later are much diminished. It is wiser, therefore, to operate in every instance just as soon as the diagnosis is made. It is even more necessary to operate early on a child than on an adult because of the greater tendency of appendicitis in early life to involve the peritoneum. If operation is done early, recovery takes place in the vast majority of instances. If it is delayed until the inflammation has spread to the peritoneum, the chances of success are much smaller. If a general peritonitis has developed, the chances of recovery are very small. They are better, however, in the child than in the adult.

Subacute and Chronic Appendicitis

The symptomatology of intermittent subacute appendicitis and of very mild chronic appendicitis associated with adhesions is very indefinite. An inflammatory condition of the appendix of this sort should always be thought of as a possibility when children have repeated attacks of indigestion without apparent cause, especially if these attacks are associated with slight pain and tenderness in the lower right quadrant. It should also always be thought of when children have a mild continuous disturbance of the digestion with loss of appetite, occasional vomiting, a tendency to constipation and do not thrive as they should. When they are properly fed and no cause for the trouble can be found in their general routine, slight tenderness or resistance in the right lower quadrant are very suggestive.

Diagnosis.—The diagnosis of subacute and chronic appendicitis is always very difficult. Examination with the fluoroscope or the taking of Roentgenograms after an opaque meal often makes a diagnosis pos-

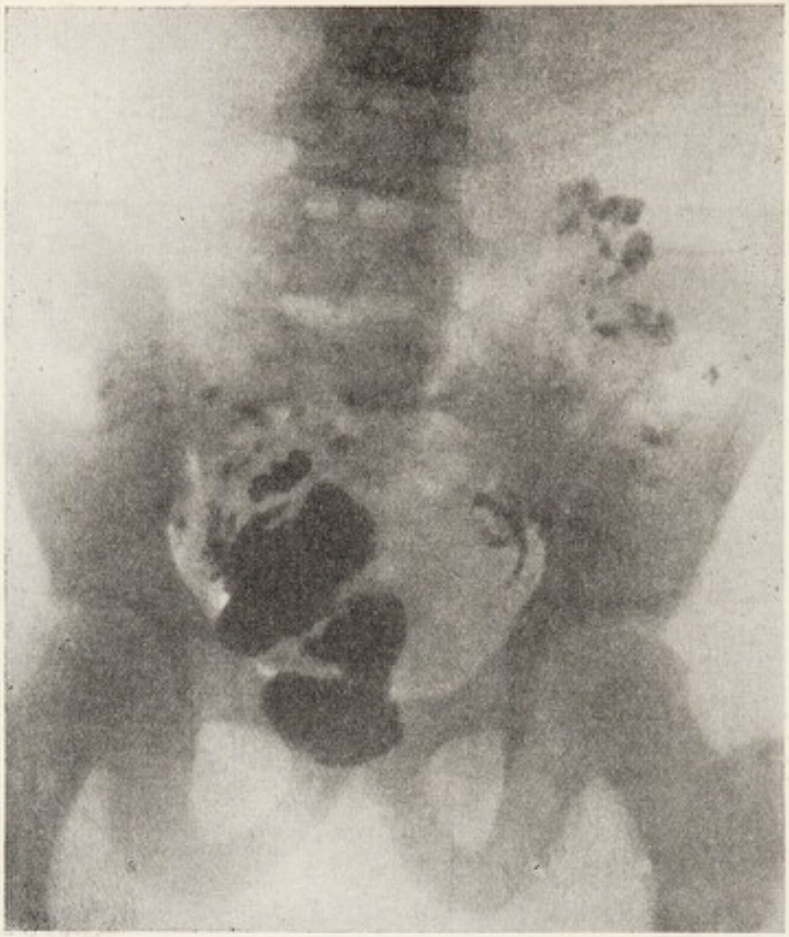

FIG. 70.—Chronic appendicitis. Note that portion of opaque meal remains in twisted appendix after rest of meal has passed on.

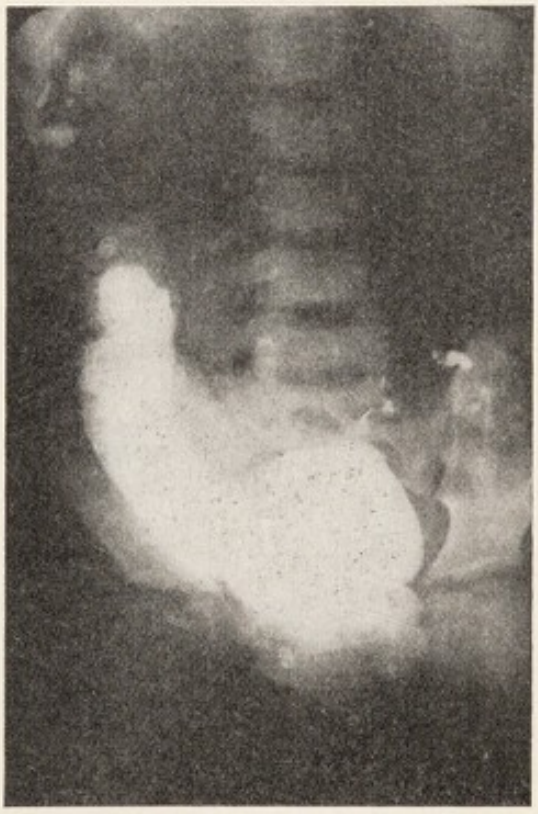

FIG. 71.—Chronic appendicitis. Taken twenty-four hours after an opaque meal. Note that a portion of the meal remains in the appendix and that it is irregularly distributed in it.

sible. Normally the opaque meal enters the appendix. It is suggestive of trouble, if it does not. Peristaltic emptying of the appendix is also normal, but stasis in the appendix is pathologic. When a part of the opaque meal remains in the appendix after the rest of the meal has passed on, the diagnosis of trouble in or about the appendix is justified. If that part of the opaque meal which remains in the appendix is irregularly distributed, a positive diagnosis is practically certain. Failure of any of the opaque meal to remain in the appendix does not, however, exclude chronic appendicitis.

Chronic inflammation in or about the appendix is, I believe, far more common than is generally thought. I have, in a number of instances, seen evidences of chronic inflammation in and about the appendix in children who had not done well and had had frequent disturbances of the digestion at an operation for an acute attack and seen all the troubles of the past disappear after the appendix was removed. I have seen also a considerable number of other cases in which the diagnosis of chronic appendicitis, which was made on the findings with the Roentgen ray was confirmed at operation and in which the symptoms were relieved by an operation.

Prognosis and Treatment.—Operation is indicated when the findings with the Roentgen ray show trouble about the appendix. It is justified when the symptoms suggest such trouble and no other cause can be found, even if the findings with the Roentgen ray are negative. When trouble is found, complete relief of the symptoms results in most instances and considerable in all.

RECURRENT VOMITING

This condition, which is also called cyclic vomiting and periodic vomiting, is in all probability not a definite disease, but simply a clinical entity or symptom complex. Nothing is known as to its pathology and very little as to its etiology. It usually does not begin until after infancy and seldom persists after twelve or, at the most, sixteen years of age. It is not infrequently replaced by migraine in later life.

Etiology.—It is equally common in boys and girls. It is perhaps rather more common in delicate children than in the strong and vigorous, but occurs in them also. It is, undoubtedly, more common in children of the nervous type and in the children of neurotic parents. What part the nervous system plays in its etiology, however, is entirely obscure. It occurs somewhat more frequently in children whose digestion is not perfect than in those with a good digestion. Nevertheless, the onset is almost never preceded by an indiscretion in diet. There is far more likely to be a history of unusual fatigue or excitement of some sort. The attacks not infrequently come on in connection with some minor illness or complicate the onset of some more serious illness.

What evidence there is points to some disturbance of the metabolism as the cause of the vomiting. It is probable that the disturbance of the metabolism is not always the same. There is much difference of opinion as to the relation between this condition and acid intoxication with the acetone bodies. These bodies are always present in the urine after the vomiting has persisted for from twenty-four to forty-eight hours. Their presence is, however, almost certainly due, in the vast majority of instances, to the starvation secondary to the vomiting, and does not prove that the vomiting is caused by them. Occasionally, however, these

bodies are present in the urine and in the breath at the beginning of an attack, or even for a number of hours before it, which suggests a closer connection between them. It is very possible, however, that the vomiting may not be due to the acetone bodies, but that both symptoms have a common cause. In rare instances cases which begin like recurrent vomiting and for a time follow the characteristic course, develop hyperpnea and show the chemical changes of acid intoxication. It is very difficult to know whether the symptoms in these cases are all due to acid intoxication or whether the acid intoxication is a secondary manifestation. However this may be, it is certainly incorrect to consider recurrent vomiting and acid intoxication as one and the same thing, as is done by so many physicians.

Symptomatology.—There is no regularity about the appearance of the attacks of recurrent vomiting. They may occur at intervals of a few weeks or of many months, the frequency varying from time to time in the same child. The terms periodic and cyclic vomiting are, therefore, not justified by the course of the disease. The attacks gradually diminish in frequency and severity and finally cease some time in the early teens.

The onset of the vomiting may be preceded for twenty-four hours or even for two or three days by slight evidences of indigestion, such as coated tongue, loss of appetite, irritability and constipation. In most instances, however, the onset is very acute, the first symptom being vomiting. The vomiting continues at longer or shorter intervals, the intervals sometimes being an hour or more, sometimes only a few minutes. I have known one child to vomit seventy-two times in twenty-four hours by actual count. The vomitus consists, at first, of the food taken at the last meal. After this it is made up of mucus and the gastric secretions. Sometimes it is mixed with bile. Not infrequently, when the vomiting is severe, it contains streaks of blood, and at times may be coffee-colored. The vomiting usually persists for two or three days, not often over five days and seldom over a week. In typical cases it stops as quickly as it began and in a few hours a child that was unable to retain anything, even water, can take reasonable foods and retain them without difficulty. In other instances the cessation of the vomiting is more gradual and it is some time before feeding can be resumed. The bowels are almost invariably obstinately constipated. Occasionally, however, there is diarrhea. There is no abdominal pain or tenderness, although the abdomen may be sore from retching. The abdomen is invariably sunken. The thirst is terrible. Except for constant demands for water, the patients are usually quiet and apathetic. The breath is usually foul, but sometimes is aromatic, or has the odor of the acetone bodies. The tongue is dry and the throat often red. Loss of weight and strength is very rapid. It is almost unbelievable how emaciated a child with this condition may become in two or three days. Fortunately, the weight is recovered almost as quickly during convalescence as it was lost. The temperature is usually slightly elevated, ranging between 100° F. and 101° F. It may, however, go as high as 102° F. or 103° F. Not infrequently it is normal. If the vomiting has persisted for several days, it is often subnormal. The pulse naturally becomes frequent and feeble as the result of weakness and the respiration rapid and superficial. The urine is much diminished in amount. There is nothing characteristic about the blood picture.

When hyperpnea develops the picture becomes that of severe acid intoxication, which, of course, it is.

Diagnosis.—The diagnosis of recurrent vomiting is easy when children have had previous attacks. It is often difficult, however, in the first attack. At the very beginning it is likely to be mistaken for an attack of acute indigestion. The more common mistake, however, is to mistake an attack of acute indigestion for recurrent vomiting. The onset may be the same in both conditions. When the vomiting is due to indigestion, however, there is almost always a history of some indiscretion in diet. The vomiting, as a rule, does not persist long after the stomach has been emptied, while it does persist in recurrent vomiting after the stomach has been emptied, whether food is taken or not. Recurrent vomiting may be confused with appendicitis or some form of intestinal obstruction. In these conditions, however, there is almost always pain or tenderness in the abdomen and the abdomen does not become sunken as in recurrent vomiting. It is either normal or distended. The temperature is usually higher in these conditions and there is a more marked leucocytosis. Recurrent vomiting ought not to be confused with intussusception, because intussusception almost always takes place in infancy, the onset is acute with pain and shock, the vomiting is not as persistent, and the stools contain blood. There may also be a palpable tumor in intussusception, which is never present in recurrent vomiting. Vomiting is sometimes a marked symptom in nephritis. In such cases, however, the child has usually been sick for some time and there is almost always edema. An examination of the urine will, of course, settle the diagnosis at once. Recurrent vomiting is at times mistaken for tuberculous meningitis. It ought not to be, however, because the onset of vomiting is not as acute in tuberculous meningitis as in recurrent vomiting. Children with recurrent vomiting may be quiet and apathetic, but are always clear mentally when aroused. They show none of the physical signs of meningeal irritation which are almost invariably present in tuberculous meningitis. A lumbar puncture will settle the diagnosis at once because the cerebrospinal fluid is normal in recurrent vomiting, but abnormal in tuberculous meningitis. The characteristic symptoms of other forms of meningitis develop so quickly and are usually so marked that these conditions are rarely confused with recurrent vomiting. Perhaps the most common error in diagnosis is to mistake recurrent vomiting for acid intoxication. The tendency of many physicians in this part of the country is to apply the term, acid intoxication, to every condition in a child in which there is vomiting. The connection between acid intoxication and recurrent vomiting is considered in the etiology. A diagnosis of acid intoxication is not justified unless the typical symptom, hyperpnea, is present or the characteristic changes of acid intoxication are present in the blood and in the expired air.

Prognosis.—The prognosis in these cases is almost invariably good. Death is very unusual, although, in many instances, it does not seem as if the child could possibly recover. It is not improbable that when death does take place the condition was really an acid intoxication from the beginning or a secondary acid intoxication developed. Death may occur, however, from exhaustion without the development of acid intoxication. Recurrence of the attacks is to be expected. Something can be done to diminish their frequency, however, by regulation of the life and diet. They eventually cease, but, as already stated, are not infrequently replaced later by attacks of migraine.

Treatment.—When children that have had attacks of recurrent vomiting show any of the premonitory symptoms of an attack, such as

loss of appetite, foul breath, constipation or irritability, all food should be stopped at once and the bowels thoroughly emptied both from above and below. They should be put to bed and given water frequently.

If there are no premonitory symptoms and the first sign of trouble is vomiting, they should be put to bed and kept as quiet as possible. All food should be stopped. The bowels should be opened from below by enemas and a laxative given by the mouth. Unfortunately laxatives are very likely to be vomited. One of the most satisfactory methods is to give repeated doses of from one half to one teaspoonful of the milk of magnesia every hour until the bowels have moved. Another drug which is not always vomited is calomel. It should be given in doses of one tenth of a grain, combined with one grain of bicarbonate of soda, every half hour until from one to three grains, according to the age of the child, have been given. Other laxatives and cathartics are almost invariably vomited. It is wiser not to attempt to give anything, even water, by the mouth. The children are so thirsty, however, that it is usually necessary to give them cracked ice to suck or small amounts of water from time to time, even though it is vomited. I have never seen any advantage in ginger ale or the carbonated waters over plain water, although they may be tried, if desired. The tendency of many physicians is to treat these children as if they had acid intoxication and to give them orange juice and sugar solution at once. These are not likely to be retained, however, and, like everything given by the mouth, are vomited at once. The old-fashioned methods of treatment with bromide and chloral by the rectum and morphin subcutaneously are more likely to be helpful than the treatment for acid intoxication. Ten grains of bromide may be given by the rectum to a child of four years and twenty grains to a child of eight years. If bromide alone is not sufficient, one grain of chloral may be combined with each ten grains of bromide. Morphin subcutaneously should not be used except in the most serious cases. It is always advisable to use extreme care in giving morphin to children. A small dose ought always to be tried first. If this is not sufficient, it can then be increased. The beginning dose for a child of four years subcutaneously is one forty-eighth of a grain and for a child of eight years one thirty-second of a grain.

If the child is becoming dehydrated, it should be given physiologic salt solution by enema. If this is not retained, it should be given by the drip method. Children of four years seldom retain an enema larger than six or eight ounces, and usually cannot absorb more than ten drops in a minute, when the drip method is used. If these methods are ineffective, salt solution should be given subcutaneously. There is no objection to using a 5% solution of glucose instead of salt solution by the rectum, as it supplies water as well as the salt solution and may possibly do something to prevent the development of secondary acid intoxication and to keep up the nutrition. If acid intoxication develops, the treatment is that of acid intoxication due to the acetone bodies.

When the vomiting has ceased for a number of hours, food may be given by the mouth. Whey is more likely to be retained than anything else. Cereal gruels and dilutions of skimmed milk with added sugar or some of the maltose-dextrins preparations may also be tried. In general, it is possible to increase the diet quite rapidly and have the child back on its usual diet in a few days.

It is difficult to know what to do to prevent a recurrence of the attacks. In general, children seem less likely to have recurrences

when they are kept on a diet low in fat and without sugar. Such a diet should, therefore, always be tried. Children subject to these attacks should be guarded most carefully against overfatigue and excitement of all kinds. They should have daily rests, go to bed early and have their daily routine so regulated that they are not subjected to nervous strain. It has not seemed to me that the administration of the alkalies or of the salicylates has any effect in preventing a recurrence of the attacks.

CONSTIPATION

Constipation is not a disease. It is a condition in which the number of stools is less or the consistency of the stools is greater than is normal for the individual at the given time.

Constipation in the New-born

Constipation in the new-born must not be confused with those conditions in which there is an absence of stools as the result of congenital malformations of the intestine, such as imperforate rectum and atresia of the intestine, which mechanically prevent the passage of feces. It is ordinarily due at this time to an insufficient intake of food as the result of delay in the secretion of breast milk. In other instances, in which the supply of breast milk or of artificial food is sufficient, the difficulty seems to be sluggishness of the intestinal peristalsis, apparently as the result of lack of use, or an innate feebleness of the intestinal muscles.

Constipation in Infancy

Etiology.—There are many causes of constipation in infancy, several of which are not infrequently active at the same time. These causes can be divided into several classes which are so different that they must be considered separately.

General Causes.—These causes, which may perhaps be better classed as unclassified, are quite different. They should always be thought of and investigated first. Among them is heredity. The large number of instances in which constipation occurs in both parents and infants makes it very probable that heredity does play some part in the etiology of constipation in infancy. The part which it plays is, however, probably much smaller than is usually supposed, the true explanation of constipation in parents and infants usually being simply coincidence.

Thyroid insufficiency should always be thought of as a possible explanation of constipation in infancy. It is probable, however, that, except in those cases in which there are other definite signs of thyroid insufficiency, as in cretinism, it is seldom the etiologic factor. Insufficiency of the secretions of the intestinal glands and liver is theoretically also a cause of constipation. It is impossible to recognize such an insufficiency, however, and when the constipation is attributed to it, it is probably, in most instances, due to some other cause.

Another cause of obstinate constipation in infancy is opium. This is sometimes given by the mothers, but more often by nurses or nursery maids without their knowledge, to keep the baby quiet and prevent it from crying. It is most often given in the form of "soothing syrup" or paregoric. It should always be considered as a possible cause, whenever babies that are constipated are unusually quiet and sleep unusually well. A careful investigation should be made in all such cases, even

though the parents deny that any opium is being given. Small pupils are, of course, strong confirmatory evidence in favor of this etiology.

Mechanical.—The large intestine is relatively longer in comparison to the small intestine in infancy than in later life and its mesentery proportionately longer. The sigmoid flexure makes up a relatively larger proportion of the colon than later in life and its mesentery is also proportionately longer. These anatomical conditions render possible the production of bends and kinks in the colon which, while they do not obstruct the lumen of the gut entirely, interfere with the passage of the intestinal contents and thus mechanically cause constipation. In other instances a Jackson's membrane or some other malformation in the neighborhood of the cecum or bands resulting from inflammatory conditions in the past may also hinder the passage of the intestinal contents. These conditions can only be determined by examination with the Roentgen ray after opaque meals or enemas. Abdominal tumors, most often in the pelvis, may also, in rare instances, be the cause of constipation. In still rarer instances, a stone in the bladder may be large enough to partially obstruct the rectum.

Spasmodic.—Spasmodic constipation is usually due to the pain which defecation causes. The pain is sometimes, but very rarely, due to hemorrhoids. It is more often due to fissure of the anus and still more often to the passage of large, hard stools. The pain which these conditions cause makes the baby put off having a movement as long as possible. It may also bring on a spasmodic contraction of the anal sphincter. This form is often the result or a complication of other forms of constipation. It may persist for a long time after the cause has been removed, because the baby continues to be afraid to have a movement on account of the pain which it used to cause. Occasionally there is a tonic spasm of the sphincter not secondary to pain on defecation. The etiology of the spasm in these cases is obscure.

Dietetic.—Abnormalities in either the quantity or the quality of the food are among the most common causes of constipation in infancy. In the breast-fed the most common abnormalities in the milk are an insufficient amount of milk, a dilute milk and a mik containing a small amount of fat. The cause of the constipation is, of course, the same in all, that is, the solids of the milk are so completely absorbed that there is not sufficient residue left to form the normal amount of feces. A high percentage of fat in breast milk may be, in rare instances, the cause of constipation, in the same way as an excessive amount of fat in an artificial food.

Constipation in the artificially-fed may also be due to an insufficient amount of food or to too weak a food, the explanation of the constipation being the same as in the breast-fed. An artificial food which is low in fat, although it contains enough sugar and proteins to supply the caloric needs, may cause constipation. The explanation is that the carbohydrates and proteins are largely absorbed and, therefore, make but little fecal matter, while there is not enough fat, which makes up a large proportion of the infant's stool, to form a sufficient residue. A far more common cause of constipation in the artificially-fed is an excess of fat in the food. The fat combines with the alkaline earths to form the so-called "soap stools." These vary in color from light yellow to gray and are often large and hard. In other instances they are dry and crumbly, resembling the stools of a dog which has been eating bones. An excess of starch in the food may also sometimes cause constipation. When the

constipation is due to this cause, the stools are large, brownish, dry and brittle.

The pasteurization of milk probably has very little influence in the production of constipation. Boiling the milk, however, undoubtedly does predispose to a slight extent to constipation. It is a far less common cause of constipation, however, than is usually believed. In most instances it has no appreciable effect. An excessive amount of milk is a common cause of constipation during the second year. At this time babies should seldom take over a quart and never more than forty ounces of milk in twenty-four hours. An insufficient amount of cereals and of fruit at this age is also sometimes a cause of constipation.

Atonic.—Muscular weakness is another very common cause of constipation in infancy. The intestinal muscles are always involved, while the abdominal muscles are not infrequently affected at the same time. One of the most common causes of weakness of the intestinal muscles is prolonged indigestion, especially if it is associated with fermentation. Rickets is very frequently associated with or a cause of atonic constipation. Malnutrition, anemia, and wasting diseases are other causes of weakness of the intestinal and abdominal muscles. Lack of exercise is another cause of muscular weakness, especially of the abdominal muscles. Many babies are kept so quiet and so bundled up that they not only do not develop, but do not keep up the tone of their muscles.

Constipation, perhaps not exactly in this class, often develops because babies have not been trained to have a movement of the bowels at a regular time and have not been taught to use their muscles in defecation. It must be remembered in this connection, however, that the constant use of suppositories or other local methods of procuring a movement at a regular time may make the baby dependent upon them and thus lead to constipation. In some young babies the presence of feces in the rectum normally causes but little desire to empty the bowel. In such instances the constipation is due simply to lack of voluntary effort on their part. In such cases there are no general symptoms of constipation and the bowels move at once, if the rectum is stimulated in some way, as by the introduction of a suppository. The continued use of laxative drugs may also bring on a condition which is very similar to that in atonic constipation, the intestines having lost their power to respond to normal stimuli.

Symptomatology.—The symptoms of constipation in infancy are often obscured by those due to the causative condition. It is often very difficult to separate the two. In a general way constipated babies are likely to be irritable and sleepless and to show evidence of general discomfort. Their tongues are coated. Their breath is foul and they suffer from flatulence and colic. None of these symptoms, however, are in any way pathognomonic. In the spasmodic form defecation is almost always associated with pain. A very important point to be remembered is that, when the constipation is due to trouble low down in the bowel, there are almost never any general symptoms associated with it. Furthermore, it must always be remembered that constipation of this sort is a much less serious condition than constipation due to trouble higher up in the bowel or to disturbance of the general condition.

Treatment.—The first step in the treatment of constipation in infancy is to discover the cause of the constipation. This entails a very careful and detailed study of the diet and of the baby's habits, as well as an examination of the stools. It involves also a careful physical examination. In most instances it is advisable also to make a rectal examination. In

this way only can spasm of the sphincter muscles be discovered. If no sufficient cause is found, an examination should be made with the Roentgen ray after an opaque meal or enema. In this way only can abnormalities of the intestine be discovered. A lack of general symptoms suggests that the cause of the constipation lies in the rectum. If the introduction of a suppository is immediately followed by the passage of a normal stool the rectum is certainly at fault. The general symptoms of disturbance are most marked when the difficulty is due to disturbance of the digestion in the small intestine.

After the cause is discovered, the treatment must be directed to its removal. Certain of the general causes, such as heredity, cannot, of course, be relieved. The administration of thyroid extract will relieve constipation due to insufficiency of the thyroid. If the constipation is due to opium, it will soon cease when the opium is stopped. When it is due to the continued use of laxative drugs, they also should be stopped as far as possible.

The growth of the parts with time is the only thing that will remedy such conditions as the long infantile colon and sigmoid flexure. The other mechanical conditions can only be remedied by operative interference. In most instances, however, the remedy is worse than the disease, and it is inadvisable to operate unless the constipation is very severe.

The treatment of spasmodic constipation consists, of course, in the removal of the cause of the spasm, that is in stopping the pain on defecation. The treatment of fissure of the anus and of hemorrhoids is discussed elsewhere. The stools can be prevented from being large and hard by proper regulation of the diet and the temporary administration of laxatives. Tonic spasm of the anal sphincter is usually easily relieved by stretching the sphincter.

The treatment of constipation due to errors in the diet is self-evident. When the dietetic errors are corrected, the constipation will cease. These errors can be determined in many instances, however, only by a most careful study of the diet and of the stools.

The chief element in the treatment of the atonic form of constipation is the relief of the causative condition, whatever it is. Another important element is the training of the baby to have a movement at a regular time and to use its abdominal muscles. Massage of the abdomen with the oiled hand twice a day for from five to ten minutes is often of considerable help. It is of less value in the other types. The abdomen should be rubbed at first superficially and then kneaded deeply, beginning in the right lower quadrant, working upward, across, and then downward to the left lower quadrant.

While the cause is being removed it is often necessary to relieve the symptom, constipation, in some way. Foods which stimulate intestinal peristalsis are of value, especially in the atonic form. Orange juice or prune juice, in doses of from one to four tablespoonfuls daily, may be given after the baby is a few months old, and baked apples, apple sauce and prune pulp added toward the end of the first year. In some instances it is safe to give a few teaspoonfuls of strained peas, string beans, spinach, asparagus tips or cooked celery daily during the last half of the second year. Great care must be taken, however, not to disturb the digestion by giving an excessive amount of fruit and vegetables and thus to increase the constipation or to set up a diarrhea. It is seldom advisable to give bran in any form to babies for constipation. It is quite likely to disturb the digestion and do far more harm than good. It should always be

remembered that when the constipation is due to mechanical interference with the passage of the intestinal contents, increasing the bulk of the food increases the trouble instead of relieving it.

If the stools are hard and dry, water, best given between the feedings, is often helpful. Agar-agar, in doses of from one to three teaspoonfuls daily, mixed or cooked with the cereals or given in broth will often keep the feces moist and also increase their bulk. It is, however, usually quite difficult to get babies to take agar-agar. Mineral oil, in doses of from one half to one teaspoonful, once, twice or even three times daily, also prevents the stools from becoming hard and dry. The agar-agar may be combined with the mineral oil.

In young babies the addition of the cereal waters or jellies to the food sometimes helps constipation. Oatmeal is supposed to be more laxative than the other cereals. In some instances, however, it is constipating, while some of the other starches are laxative. The substitution of one of the maltose-dextrins mixtures for milk or cane sugar also sometimes relieves constipation in little babies. The larger the proportion of maltose, the greater is the laxative action of these sugars. Those preparations which contain the potassium salts are apparently somewhat more laxative than those containing the sodium salts.

If these measures are unsuccessful, it is necessary to move the bowels by the administration of drugs by the mouth or by stimulation of the intestines from below. When the cause of the constipation is in the rectum, stimulation from below is evidently indicated. When the seat of the trouble is higher up or is a more general one, it is often very difficult to decide which method to adopt. A good general rule is not to use the same method continuously, as there is less danger of establishing a bad habit, if the methods are varied. There is more danger of making a baby dependent on stimulation of the rectum to produce a movement than of making it dependent on drugs. The danger of causing constipation by the use of suppositories to teach babies to have regular habits has already been mentioned. Suppositories are apparently more dangerous in this connection than enemas. The simplest and least irritating type of suppository is a roll of paper dipped in sweet oil. Gluten suppositories are less irritating than glycerine suppositories. The soap stick stands midway between them. The best and simplest form of enema is that composed of soap and water. No more should be given than is necessary to produce the desired result. From two to four ounces is usually enough, but more may be given if necessary. It is best given with a soft rubber ear syringe, but a fountain syringe may be used if desired. If the stools are hard and dry, an enema of from one half ounce to one ounce of sweet oil, given to be retained and followed later by a suds enema, if necessary, is very useful. The oil should be given with a glass or hard rubber syringe. It is not advisable to use glycerine enemas or anything stronger in the treatment of constipation in infants.

The simplest laxative for a baby is milk of magnesia. One teaspoonful a day is usually sufficient, but more may be given if necessary. It is best to give it all at one dose, preferably in the last feeding at night. Most babies are not disturbed by it, but it occasionally causes considerable pain and discomfort. Babies very seldom become dependent upon it. If milk of magnesia causes discomfort, phosphate of soda, in doses of from ten to sixty grains, may be tried in its place. This is also best given in the milk. Olive oil is useful in some instances. It must never be forgotten, however, that olive oil is a fat and that, when constipation is

due to fat, olive oil increases instead of relieving it. Olive oil may also, like any other fat, disturb the digestion.

During the later part of the second year phenolphthalein, in doses of from one to three grains, may be use instead of magnesia or phosphate of soda. Cascara sagrada, in doses of from one half to one grain or of from five to thirty drops of one of the liquid preparations, may be used. So also may be some of the preparations of senna. These, however, should not be used continuously, as they are likely to cause secondary constipation. Purgative drugs, such as castor oil and calomel, should never be used continuously in the treatment of constipation. They are too powerful and have a secondary constipating action. They are, however, most useful when it is necessary, for some reason, to thoroughly empty the bowels.

Constipation in Childhood

Etiology.—The etiology of constipation in childhood is not materially different from that in infancy, although the importance of the various causes is quite different. Heredity may perhaps play a slightly more

Fig. 72.—Long colon in boy of seven years. Taken after opaque enema. Chronic constipation and recurrent attacks of intestinal stasis.

Fig. 73.—Chronic constipation at ten years. Taken twenty-four hours after an opaque meal. Note kink at splenic flexure.

important part at this time than earlier. Insufficiency of the thyroid secretion is probably of even less importance than earlier. Children are not, of course, given opium to quiet them, but occasionally they do get enough in cough mixtures, which are taken for a long time, to cause constipation.

Mechanical constipation from a relatively long colon or sigmoid flexure becomes, of course, progressively less common as the child grows older, and ceases normally at about six years, when the relations between the large and small intestine are essentially the same as in the adult. On the other hand, constipation from slight congenital malformations and from intestinal adhesions is likely to become rather more marked.

Dilatation of the colon may also develop at this time. Pelvic tumors and vesical calculi are also slightly less uncommon causes.

Spasmodic constipation is less common, as children have fissures of the anus much less often than infants and seldom have hemorrhoids. It may, as in infancy, however, be caused by large, hard stools. Tonic spasm of the anal sphincter seldom plays a part after early childhood.

An insufficient amount of food is seldom a cause of constipation in childhood, although it may be occasionally. Insufficient amounts of the leafy and coarser vegetables, of fruit and of water are, however, not infrequent causes of constipation in childhood. An excess of fat in the food is sometimes a cause of constipation in early childhood as in infancy. Occasionally an excessive amount of food which has a large residue may be the cause of constipation. In most cases, however, in which errors in diet are the cause of constipation in childhood, it is because they have brought on indigestion. This may cause a secondary insufficiency of the intestinal and abdominal muscles. Such an insufficiency from indigestion is uncommon, however, except in early childhood. Weakness of the intestinal and abdominal muscles is more often in childhood the result of marked general disturbance of the nutrition from some other disease. Lack of exercise is occasionally a cause of constipation, most often in girls in late childhood. This is a less common cause, however, than it used to be, because girls in general lead a far more active life than they did in the past.

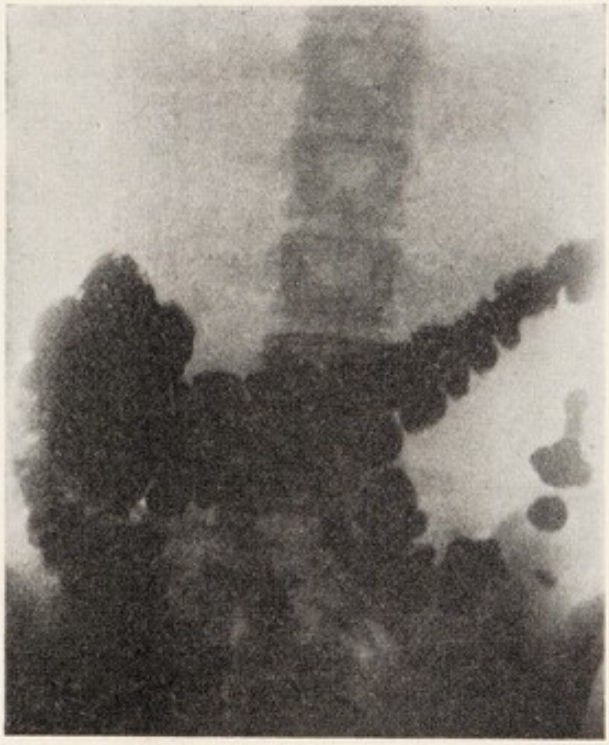

FIG. 74.—Chronic constipation from peritoneal adhesions. Age, fifteen years. Taken twenty-four hours after opaque meal. Note low position of cecum and bend in ascending colon.

Improper habits are a far more common cause. Children are not trained, as they should be, to go to the toilet immediately after breakfast and are not made to realize the importance of regular habits. Constipation is also far more often due to the abuse of laxative drugs in childhood than in infancy.

Symptomatology.—In many instances there are no recognizable symptoms of constipation in childhood. Not infrequently, however, constipated children have coated tongues and foul breaths. They are likely to be troubled by gas and occasionally by pain. They do not thrive as well as the normal child. When constipation is associated with, or is the result of, indigestion, as it often is, it is almost impossible to determine what proportion of the symptoms is due to constipation and what to indigestion. This is also the case when constipation is secondary to other diseases.

Treatment.—The first step in the treatment of constipation in childhood, as in infancy, is to discover the cause of the constipation. This entails a very careful and detailed study of the diet and of the child's

habits, as well as a thorough physical examination. The examination of the stools is helpful, but not as much so as in infancy. If no sufficient cause can be found, an examination should be made with the Roentgen ray after an opaque meal or enema, as in this way only can abnormalities of the intestine be discovered.

After the cause is discovered the treatment must be directed to its removal. Certain of the general causes, such as heredity, cannot, of course, be relieved. Constipation secondary to other diseases will, of course, cease when the other diseases are cured. When constipation is due to an insufficient amount of food, it can be cured by increasing the amount of food. When it is due to an insufficient intake of green and coarse vegetables or fruit, it can be cured by increasing the amount of these foods in the diet. It is important to remember, however, that it is possible to cause constipation or to exaggerate a previously existing constipation by giving too many vegetables and too much fruit and thus setting up indigestion. In my experience it is inadvisable to try to overcome constipation by giving vegetables more than once, or at the most twice a day, or fruit more than twice a day. It can also be cured by increasing the amount of water ingested, if a lack of water is the cause. When constipation is the result of indigestion, the treatment is, of course, to so regulate the diet as to cure the indigestion. When this is cured, the constipation will cease. The treatment of constipation due to lack of exercise is also self-evident. In such cases special attention should be given to strengthening the abdominal muscles. An abdominal belt is sometimes helpful. When constipation is due to improper and irregular habits of defecation, the treatment consists in the establishment of regular habits. Children should be made to go to the toilet immediately after breakfast. The breakfast hour should be so fixed that the child has time enough to go to the toilet without feeling hurried or worried about getting to school. It should be made to try for ten minutes, if the bowels have not moved before. It is inadvisable for it to try longer. It should not be given anything to read or to play with at that time. If it is, the toilet simply takes the place of a chair. If the bowels have not moved after breakfast, another attempt should be made after dinner and, if necessary, another after supper. The child should be made to understand that a regular movement of the bowels is just as important as the meal which precedes it. Regular massage of the abdomen and exercises to strengthen the abdominal muscles are helpful, especially in young children.

While the cause is being removed it is often necessary to relieve the symptom, constipation, in some way. Bran has a certain value in that it stimulates intestinal peristalsis. It is less likely to do harm in childhood than in infancy. It must not be forgotten, however, that bran and other bulky foods may increase the constipation in those cases in which there is already an excessive amount of intestinal contents, in that they increase the bulk of the stools. Bran may also disturb the digestion. One of the best ways to give it is in the form of Pettijohn's breakfast food. It may be cooked with other cereals or made into the form of bran crackers or bran muffins. It must also be remembered that, when constipation is due to mechanical interference with the passage of the intestinal contents, increasing the bulk of the food increases the trouble instead of relieving it.

If the stools are hard and dry, water, best given between meals is often helpful. Agar-agar is also often useful because it keeps the feces

moist and also increases their bulk. Children take agar-agar better than infants do. Mineral oil also prevents the stools from becoming hard and dry. It may be given in doses of from one teaspoonful to one tablespoonful once, twice or three times daily. Agar-agar may be combined with the mineral oil.

If these simple measures are not sufficient, the bowels must be moved in some way from either above or below. In general it is wiser to move them by giving drugs by the mouth than by the regular use of suppositories or enemas. In early childhood milk of magnesia and phosphate of soda are the best laxatives, if they are efficient. If they are not, the most useful drugs are phenolphthalein, cascara sagrada and senna. The doses must, of course, vary with the age of the child and the degree of the constipation in the individual case. In older children aloin is often useful. The combination of aloin and podophyllin is also sometimes useful. It should not be forgotten that aloin acts chiefly on the lower bowel and podophyllin chiefly on the upper bowel. Another useful drug in children, which has been almost forgotten in recent years, is rhubarb. It is irrational to combine these laxative drugs with strychnia and belladonna, because the time at which these drugs act is so different that they cannot reënforce each other's action as is so commonly believed. It is needless to say that the continuous use of all of these drugs is likely to cause secondary constipation, that they should be used as little as possible and given up as soon as may be. They should be looked upon simply as temporary expedients to be used while the cause of the constipation is being removed.

INCONTINENCE OF FECES

Incontinence of feces is normal in infancy. The time at which control of the anal sphincter is acquired depends very largely on how much attention is paid to training the baby. It is also influenced by the general condition of the baby, control being acquired later in babies that are feeble muscularly or that have been ill.

Incontinence of feces is a symptom of many diseases of the spinal cord in which the control of the lower centers in the cord from above is cut off, the reflex arc remaining intact. Incontinence of feces may also occur in the course of any serious illness, whether acute or chronic, either from loss of cerebral control or as the result of extreme weakness. It is said to be not uncommon in severe chorea. I have, however, seldom seen it in this disease. It may also develop after operations for atresia of the anus or rectum.

Incontinence of feces may occur, however, in children when the cause is not so obvious. Spina bifida occulta should always be thought of when there is persistent incontinence of feces, especially if it is associated with incontinence of urine. Incontinence of feces is occasionally due to reflex irritation from renal or vesical calculi. It is sometimes the result of overstretching of the rectum with feces, so that the sphincters cannot act. In such cases the liquid feces from above pass through a canal in a hardened mass of feces which distends the rectum and almost fills the pelvis. Incontinence of feces from this cause is often associated with incontinence of urine. The treatment of incontinence of feces from this cause consists primarily in emptying the rectum mechanically. Enemas are often not sufficient and the mass of feces has to be dug out. Not infrequently recovery is immediate after the rectum is emptied. Further treatment consists in the prevention of a recurrence of the impaction.

Incontinence of feces is more often due to a general lowering of the nervous and muscular tone as the result of overfatigue, either physical or nervous, or of a general disturbance of the nutrition. Under these conditions there may be increased excitability of the centers in the lumbar cord to reflex or other stimuli or weakness of the sphincter muscles. The treatment of this type of incontinence of feces consists in regulation of the child's life to pevent overfatigue and general measures to build up the nutrition. It should be taken out of school, given a large amount of rest and properly fed. Change of environment is often helpful. Numerous drugs are recommended in the treatment of this condition. It is doubtful if any of them do much good. Strychnia in fairly large doses is probably more likely to help than the others. Local treatment with electricity may be tried in order to stimulate the sphincter muscles. I am inclined to believe, however, that recovery occurs just as quickly without it. All the cases of this sort which I have seen have responded promptly to common-sense regulation of their life without drugs or local treatment.

INTESTINAL PARASITES

In Boston and its vicinity intestinal parasites are uncommon in childhood and when present seldom cause any symptoms. I have seen many children that were thought to have tapeworms and round-worms, but have almost never found any worms in these children. Those that had worms did not show any symptoms, the worms being discovered by accident in the stools. I am aware, however, that the experience of physicians in other sections of the country is different from mine and that in certain parts of the country worms are more common and do cause definite symptoms. Pin-worms however, often cause definite and characteristic symptoms. I have never seen a case of hook-worm infection which originated in this vicinity or of infection with the bothriocephalus latus.

TAPEWORMS

The cases which I have seen have been about equally divided between the beef and pork tapeworms. I have seen but one case of infection with the dwarf tape-worm (tenia nana), although this worm is said to be common in New York. It makes no difference to the child which type of worm it has. Infection occurs as in adults. I have never seen a case which could be attributed to the use of beef juice.

I have never seen a case in which the presence of a tapeworm was suspected before segments of the worm were found in the stools. After segments were found, the parents sometimes thought of symptoms which might have been due to the worm. The general condition of the children has always been good. They have never had a large appetite. Eosinophilia has been usually, but not always, present.

Treatment.—Infection with the beef and pork tapeworms can be prevented by never eating either beef or pork which has not been thoroughly cooked. The cysticercus stage of the dwarf tapeworm being unknown, it is impossible to know how to avoid infection with it.

It is usually possible to get rid of a tapeworm at the first trial, if sufficient care is taken in carrying out the details of the treatment. If it is not, the treatment often has to be repeated several times.

It is very important to have the intestines emptied of everything but the worm before the anthelmintic is administered. The diet should be

limited for two days to foods which have but little residue, such as clear soups, whey, white of egg and orange juice, and the amount should be limited to that just sufficient to satisfy the pangs of hunger. A very little toast or cereal may be given, if necessary. Enough of some cathartic, preferably epsom salts or castor oil, should be given to procure several large loose movements of the bowels each day. A cup of hot beef tea or clear broth should be given on waking the morning of the third day. This should be followed in one half hour by the anthelmintic. The best anthelmintic is Tanret's preparation of the tannate of pelletierine. One bottle of this preparation contains about five grains of the drug. The dose for a child of four years is one third of a bottle. For a child of eight years about two thirds of a bottle and for a child of twelve years or over a whole bottle. If this drug is not obtainable, ten minims of the oleoresin of aspidium may be given in its place. I have occasionally found pumpkin seeds very useful when neither pelletierine nor aspidium were effective. One half hour later a cathartic should be given. I have found epsom salts and compound licorice power the most satisfactory. An ounce of epsom salts should be given to a child of four and proportionately larger doses to older children. One half this dose should be given every subsequent hour until the worm is passed. It must not be forgotten that, if the salts do not move the bowels, it is possible they may cause considerable depression. The anthelmintic and the cathartic are less likely to be vomited, if the child is kept up and walking about than if it lies quietly in bed. When the worm begins to come, the child should sit on a vessel filled with warm water, because the worm is less likely to break off and is more certain to be passed intact, if it finds itself in comfortable surroundings. It should never be pulled, as it is certain to break, if any force is used. If part of the worm remains in the bowel while the rest is out, it can often be dislodged by a large enema of warm water.

Everything which is passed should be saved and carefully examined in order to determine whether or not the head has been passed, the treatment being of no avail unless this is obtained. It is important to remember in this connection that the head is dark colored and not much larger than the head of a pin and that the upper part of the neck is very thin. Unless this is borne in mind and explained to the attendants, the head is very likely to be overlooked and thrown away, as the laity usually think that the head looks like that of a snake or an eel. Needless to say the child is usually a good deal of a wreck after the treatment is over.

ROUND-WORMS

(ASCARIS LUMBRICOIDES)

The round-worm looks like an ordinary earth-worm. It varies in length from five to ten inches. The female is larger than the male. It is straight while the male is curved. The round-worm is cylindrical in shape and tapers toward the ends. It is yellowish white or reddish yellow in color. It is hard to mistake round-worms for anything else. They live in the small intestine. There are usually several worms, always more than one, and sometimes many. The round-worm lays many eggs which pass out through the anus. The embryos develop outside the human body and enter it through the mouth in drinking water.

Symptomatology.—I have known a round-worm to crawl out of a child's mouth. I have never known them to do many of the strange

things that they are supposed to do, although one may have occasionally poked its head into the common duct or into the appendix without my knowing it.

Just as in the case of tapeworms, I have never seen a child that presented the symptoms supposed to be characteristic of round-worms, such as picking the nose, gritting the teeth, and restlessness at night, that had any worms. Loss of appetite and other symptoms of indigestion, which are so often attributed to worms, are almost always due to improper food or improper habits of eating. Sleeplessness is due to indigestion and over-fatigue; picking the nose to crusts, irritation in the nares or bad habits. When worms are found, parents, on looking back, always find symptoms which can be attributed to them. In one instance, attacks of convulsions, supposed to be epileptic, ceased after several round-worms were passed. I have never seen any other nervous manifestation which could be attributed to round-worms. The blood usually, but not always, shows an eosinophilia.

Diagnosis.—The diagnosis can only be made by finding the worms or their eggs in the stools. A vermifuge often has to be given, however, to satisfy the grandparents and the neighbors that the child does not have worms. They are usually convinced that it has not, when none are found in the stools after a vermifuge has been given. True's Elixir is the favorite in this part of the country. A better method, however, is to examine the stools for ova, which are usually numerous. The ova measure between 0.05 and 0.07 millimeters in length and 0.04 and 0.05 millimeters in breadth. They are elliptical in shape, with a thick transparent shell. This has an external coating of albumin which sometimes forms protuberances. The albuminous coating is stained yellow by the feces. The eggs of the pin-worm are oval and measure 0.05 by 0.02 millimeters. They are thin shelled.

Treatment.—Infection with round-worms can usually be avoided by the exercise of proper care in the selection of drinking water. The treatment of round-worms is along the same lines as that of tapeworms, but much less drastic. The anthelmintic of choice is santonin. A dose of epsom salts or some other saline, sufficient to thoroughly clean out the bowels, should be given. Salines are better than castor oil, because castor oil favors the absorption of santonin. When the bowels are well emptied, the child should be kept on broth with a little toast for twenty-four hours and given three doses of from one quarter to one grain each of calomel and santonin at four hour intervals according to age. This should be followed by one or more doses of epsom salts. This course of treatment will in all probability result in the passage of all the worms in the intestines. It is advisable, however, to examine the stools for ova after three or four weeks. If any are found, this treatment should be repeated. I have never seen any serious symptoms, such as nervous excitement or delirium, result from the use of santonin, although it has occasionally caused vomiting and, in a few instances, yellow vision.

PIN-WORMS

(OXYURIS VERMICULARIS)

Pin-worms look like pieces of white linen thread. The female varies in length from one quarter to one half inch. The male is considerably smaller and its tail is curled up. They are usually extremely numerous. Their habitat is in the large intestine, but they are often found in the

lower part of the small intestine and sometimes even higher. The worms and ova are passed in large numbers in the feces. The worms also not infrequently crawl out through the anus at other times. The ova are likely to be left in the fold of the nates and on the buttocks. The children get them on their fingers and then put them in their mouth. The ova hatch out as they go down the digestive tract, mature and produce more ova, which are in turn put into the mouth, a real vicious circle being established. The ova may also be carried by flies to food and water and be taken into the mouth with them, thus infecting the same or other children.

Symptomatology.—The most prominent symptom is usually itching about the anus. This is more common at night, because the worms are more likely to crawl out at this time when the child is quiet and warm. The itching causes scratching, which is the cause of various secondary skin lesions. The itching also makes children restless and sleepless at night. The worms not infrequently crawl forward into the vulva of little girls, causing vulvitis and vaginitis. They also occasionally cause balanitis in boys. They occasionally, by reflex irritation, cause frequent micturition and sometimes enuresis. In rare instances they get into the rectum and cause catarrhal proctitis. There are no general symptoms of pin-worms, except those which are due to the loss of sleep and the annoyance caused by the itching. These may, however, be fairly marked. They do not cause picking of the nose or disturbance of the digestion. If these symptoms are present, they are coincidences, not results. Pin-worms do not usually cause eosinophilia.

Diagnosis.—Pin-worms should be suspected, if children are restless after going to bed or if they itch about the anus. The worms can usually be discovered by looking at the buttocks and around the anus after the child has been in bed for an hour or so. If none are seen then, they usually can be seen by everting the mucous membrane of the anus. If none are seen then, they can usually be obtained with an enema. It is very important not to mistake pieces of fruit or vegetable fiber for pin-worms. Parents do this constantly. It is easy to distinguish them, however, if it is remembered that pin-worms, when passed, are alive and that vegetable and fruit fibers are not. Pin-worms are, moreover, smooth and pointed at the ends, while vegetable and fruit fibers are almost never smooth and are likely to have irregular or shredded ends. If no pin-worms are found on examination or by enema after several trials, it is safe to conclude that there are none.

Prognosis.—It is always difficult, but always possible, to get rid of pin-worms. Treatment should be kept up until no worms are seen and then for at least six weeks longer. If it is stopped as soon as the pin-worms disappear, they are almost certain to return, because they have not been entirely eradicated. The treatment has to be kept up until all the worms have come down from above and been passed out.

Treatment.—The life history of the parasite shows the lines along which treatment must be directed. The eggs enter through the mouth and are hatched in the small intestine. The worms reach their full development in the large intestine and lay their eggs in the rectum. The children get the eggs on their fingers, put their fingers in their mouths and the circle is completed. Every precaution must be taken, therefore, to insure strict cleanliness and to prevent reinfection. It is advisable also to bathe the anus and buttocks with a 1-10,000 solution of corrosive sublimate after each defecation. Children must be taught not to put

their hands to the anus, buttocks or genitals, not to put their fingers in their mouths and to wash their hands before they eat.

The next thing to do is to dislodge the worms from the upper bowel, wash them down and, if possible, out. This is best done with epsom salts or some other saline. Salines are better than castor oil, because castor oil favors the absorption of santonin. When the bowels are well emptied the diet should be limited to broth and toast for twenty-four hours and the child given three doses of from one quarter to one grain each of santonin and calomel, according to its age, during the day. This should be followed by another dose of epsom salts or some other saline. In case the pin-worms persist, this course of treatment should be repeated about once in four weeks.

It is evident that drugs given by the mouth can do but little good after the upper bowel has been thoroughly cleared of the worms, since those that remain are in the large bowel and rectum and must be reached from below. The most satisfactory method of treatment from below is to give an enema of from two to six ounces of sweet oil with a hard rubber or glass syringe every night. This should be followed in ten or fifteen minutes by an enema of soap suds as large as the child can take comfortably. The worms are caught in the oil and are washed out by the suds. These enemata must be kept up until no worms are obtained and then for some weeks longer. If they are given up at once, the worms will almost certainly appear again in a few weeks. I have found this method preferable to injections of quassia and similar remedies, which are so often used.

The treatment of the itching caused by the lesions of scratching is the same as that for the treatment of itching when the lesions are due to other causes.

SECTION VIII

THE SPECIFIC INFECTIOUS DISEASES

TUBERCULOSIS

Tuberculosis is caused by the bacillus tuberculosis of Koch. Infection may be due to either the human or bovine types. The relative frequency of human and bovine infection varies greatly in different localities according to the conditions in these localities. In general, glandular and abdominal tuberculosis are more likely to be bovine in origin than pulmonary, bone and diffuse tuberculosis. Practically, however, the type of infection is of no importance.

Intrauterine infection may occur, but happens so rarely that it is of no practical importance. Infection may take place at any time after birth. How soon it occurs depends very largely upon the local milk supply and the precautions taken in the community to prevent the spread of human tuberculosis. Hence the great variation in the figures from different localities. The proportion of individuals infected increases progressively with age. In round numbers, it is probably safe to say that between 5% and 10% have become infected by the end of the first year, and that at sixteen years not more than from 10% to 20% have escaped infection. Whether active tuberculosis developing in adult life is due to the lighting up of an infection acquired in infancy or infection in early life protects against further infection in later life by establishing a certain degree of immunity are academic questions and of no practical importance to the pediatrist.

It is almost certain that there is no inherited predisposition to tuberculosis. At most there is only an enfeebled resistance to all sorts of infection. Poverty, overcrowding, and lack of fresh air and sunshine, by diminishing the general resistance, undoubtedly favor the development of tuberculous infection. They act far more powerfully, however, by increasing the opportunities for direct contagion and by favoring the retention of virulence in the tubercle bacilli. Other diseases, especially those which involve the respiratory tract, predispose to the development of tuberculous infection, or, more often, light up a preëxisting infection. Among these, measles, whooping-cough and influenza are especially important.

In rare instances, tuberculous infection may occur through direct inoculation, as from a bite or during a ritual circumcision, or through some lesion of the skin. In general, however, the germ is put into the mouth with the food or on the hands, inhaled in dust, or directly conveyed into the mouth and nose as the result of coughing and kissing. Infection from the food is most often through milk, sometimes through butter and cheese, almost never through meat or beef juice.

Tubercle bacilli may enter the body through the respiratory or alimentary tracts, including in the alimentary tract the structures in the fauces and in the respiratory tract the nose and nasopharynx. The relative frequency of infection through these tracts is of little practical

importance. It is also of no practical importance, if infection takes place through the respiratory tract, whether there is a primary focus in the lung, as Gohn teaches, or the bacilli pass directly through the lungs to the tracheobronchial glands, as was until recently believed. The result is the same either way. Furthermore, when tubercle bacilli get into the tracheobronchial lymph nodes, it makes no difference whether they go directly through the bronchi and lungs or indirectly through the stomach and intestines. It likewise makes no difference, when the abdominal lymph nodes are involved, whether the tubercle bacilli were inhaled and swallowed or were taken in the food.

As already stated, a steadily increasing proportion of infants and children become infected with tuberculosis as the age increases. There is not, however, a corresponding increase in the proportion of children ill with tuberculosis. That is, although they have become infected, the infection is not making them ill. In other words, although they have a focus of tuberculosis somewhere in the body, that focus is inactive and is causing no symptoms That is, if disease is defined as an alteration in the state of the body or of some of its organs, interrupting or disturbing the performance of the vital functions and causing symptoms of some sort, they are not suffering from tuberculous disease. It is very necessary, therefore, to distinguish between tuberculous infection and tuberculous disease. The important thing is not so much whether they are or have been infected with tuberculosis as whether the infection is or is not active. In this connection it has always seemed foolish to me to speak of a pre-tubercular stage. I have, in fact, never been quite certain what those who use this term mean by it, whether they mean a condition of malnutrition or increased susceptibility from some cause or other before tubercle bacilli enter the body or a period after tubercle bacilli have entered the body, but in which there is no active process giving symptoms. In either case, it seems to me that the term is a poor one. A child either has or has not been infected with tuberculosis; therefore, he either is or is not tuberculous. If the infection is latent, he has no symptoms; if it is active, he has symptoms.

Tuberculin Test.—Although in the past I used the subcutaneous and eye tests, the only ones which I use now are the cutaneous, or Pirquet, test and the intracutaneous, or intradermal. The latter is the more delicate and should be tried in cases in which there is reason to suspect tuberculosis, if the Pirquet test is negative. The Pirquet test is, however, sufficiently accurate for ordinary use. The cutaneous, or Pirquet, test is performed by scratching or scarifying the skin of the forearm with some sharp instrument. A special scarifier resembling a small chisel is most suitable, but a needle or knife may be used. The wound should not be deep enough to cause bleeding. It is customary to make three abrasions about an inch apart, the middle serving as a control. On the upper and lower is placed a drop of undiluted tuberculin, which is allowed to dry. If preferred, the abrasion may be made through the drop of tuberculin. Human tuberculin may be used on one spot and bovine on the other. This is unnecessary, however, as if there is a reaction to one there almost always is to the other. Furthermore, it makes no practical difference whether the infection is human or bovine. The skin should be washed with alcohol or ether before the scarifications are made and the spots should be covered with a sterile dressing after the tuberculin has dried. When the reaction is positive, a reddened areola appears about the site of the scarification in about twenty-four hours, reaches its maximum in

the second twenty-four hours, and gradually fades out in the next two to four days. The reaction cannot be regarded as positive unless it is at least one quarter of an inch in diameter. There is, of course, no reaction about the control. In suspicious cases the test should be repeated in a couple of weeks, because of the possibility of some error in technic and also because the application of the tuberculin apparently sometimes makes the second test positive.

In the intradermal test a measured amount of diluted tuberculin is injected into the superficial layers of the skin of the flexor surface of the forearm. One tenth of a c.cm. of a 1 to 1000 dilution of old tuberculin is used.

If there is no reaction after seventy-two hours, one tenth of a c.cm. of a 1 to 500 dilution may be used. If there is still no reaction within seventy-two hours after this injection, one tenth of a c.cm. of a 1 to 100 dilution may be used, provided the symptoms are strongly suggestive of tuberculosis. A positive reaction is shown by the development of a reddened or indurated area at the site of injection. This usually appears within twenty-four hours and may increase in size for several days. It always appears within seventy-two hours. It is often painful. The discoloration may persist for weeks.

In estimating the value of the tuberculin test it must never be forgotten that a positive test means simply tuberculous infection. There may or may not be tuberculous disease as well. A positive test in infancy is more valuable than later, because at that age infection is more likely to be accompanied by disease. A positive test in a sick baby showing no other evident cause for its illness is very strong evidence in favor of the trouble being tuberculosis. A negative test is even stronger evidence against tuberculosis being the cause of the illness, although it does not positively exclude it, because the baby may be overwhelmed by the infection. This may happen not only in acute miliary tuberculosis but also in chronic diffuse tuberculosis. In this type a positive test can sometimes be obtained after repeated trials.

The test is of less value in children, and the older the child the less is the value of the test, because of the steadily increasing frequency of infection with age. A negative test is of great importance in childhood, however, and practically rules out tuberculosis, unless the child is evidently overwhelmed by some infection. Because of the severity of the infection, the test is usually negative in acute miliary tuberculosis and in tuberculous meningitis, which is, of course, a variety of acute miliary tuberculosis. I have also seen it absent several times in tuberculous peritonitis, even when it was not of the miliary ascitic type. A positive test is of much less value than a negative in childhood, because, while it shows that the child has been infected with tuberculosis, it does not show that the illness which it has is tuberculosis. The reaction may as well be due to some old or latent infection, or to the infection of some hidden gland, as to the illness from which the child is suffering. It is of some help, however, if the lesions which the child has may well be tuberculous and there is no other evident cause for the illness. Great care must be taken about calling pulmonary lesions tuberculous, however, simply because the tuberculin test is positive. I have seen many mistakes of this sort made, because physicians did not realize that physical signs merely indicate certain changes in the lung tissue and show nothing as to the cause of these changes.

Tuberculides.—The presence of tuberculides, which are small, dark red papules, which often have a scaly apex, shows evidence of an active

and usually of a generalized infection with tuberculosis. Their presence is of especial value, because they are most often found in just the cases in which the tuberculin test is negative, that is, in cases of acute miliary tuberculosis. They should be looked for, therefore, in all cases when this disease is a possibility. Their presence usually, but not always, indicates a fatal termination.

Tuberculosis at Different Ages.—A tuberculous infection in infancy may be local, never cause any symptoms and never be recognized. Contrary to the general belief of a few years ago, a positive tuberculin test does not by any means condemn a baby to death. It is certain that many babies become infected with tuberculosis that never show any evidences of this infection, except a positive tuberculin test, or, at any rate, do not show any evidences of it until later in childhood or in adult life, when the infection may become active. Nevertheless, the tendency of tuberculosis to become generalized is much greater in infancy than it is later. When it becomes generalized, it may take either one of two forms, the acute miliary or what used to be called chronic diffuse tuberculosis. A tuberculous infection in infancy, especially when it is of the chronic diffuse type, may give only the evidences of malnutrition with no localizing physical signs. Tuberculous bronchopneumonia is a very common form of tuberculosis in infancy. Tuberculous meningitis is also more common in infancy than in childhood, because of this tendency to generalization at this age. While tuberculosis in infancy which gives definite physical signs carries a very grave prognosis, it is not, as we used to think, invariably fatal. Babies with recognizable tuberculosis not infrequently recover.

In young children the tendency to localization is greater. The bones and serous membranes are much more frequently involved than the viscera, and there is also a decided predilection for the lymphatic system. Pulmonary lesions are more diffuse and progress more rapidly than in later childhood, but less rapidly than in infancy. They seldom go on to the formation of cavities before six or eight years. The tendency to acute general infection, miliary tuberculosis, is still marked, although less so than in infancy, until middle childhood. After this time the pathologic processes and progress of tuberculosis are not very different from those in young adults.

Acute Miliary Tuberculosis

Acute miliary tuberculosis is, of course, never primary. It is due to the flooding of the organism through the circulation by tubercle bacilli which have entered it as the result of the breaking down of some old focus. Death is inevitable and occurs before the tubercles have passed the miliary stage. It is not proper to apply the term "miliary," as is so often done, to diffuse tuberculosis, in which the lesions are less numerous and life is prolonged until they have passed far beyond the miliary stage. The symptomatology depends on whether the distribution of the tubercles is quite general throughout the body or there is a marked preponderance in the lungs or meninges. When there is a marked preponderance in the meninges the symptoms of meningeal inflammation overshadow those caused by the tubercles in the other organs and the condition is known as *tuberculous meningitis*. (See Tuberculous Meningitis.) The meningitic form of acute miliary tuberculosis is especially common in infancy and diminishes progressively in relative frequency with increasing age.

The *pulmonary type* is about equally common at all ages. It is especially likely to follow measles and whooping-cough. The most characteristic thing about the symptomatology is the marked discrepancy between the severity of the symptoms pointing to disease of the lungs and the paucity of physical signs in the chest. The respiration is rapid and there is usually some cyanosis, which, toward the end, often becomes very marked. Cough is troublesome, but usually unproductive. The temperature is irregularly elevated. The percussion note is, however, not changed, except that at times it is slightly tympanitic, presumably from emphysema. The respiratory and voice sounds are normal. In some instances no râles are audible. In most cases, however, dry or crepitant râles are heard. Toward the end, when the heart begins to fail, moist râles also develop. The leukocyte count is usually not increased, but may be. The tuberculin test is almost always negative. Tuberculides can sometimes be found, if looked for carefully. The Roentgen ray shows a very characteristic, generally distributed mottling of the lungs. The child is evidently very ill from the beginning, and always appears much sicker than the physical signs warrant. Death usually occurs in less than two weeks, but may be delayed some weeks longer.

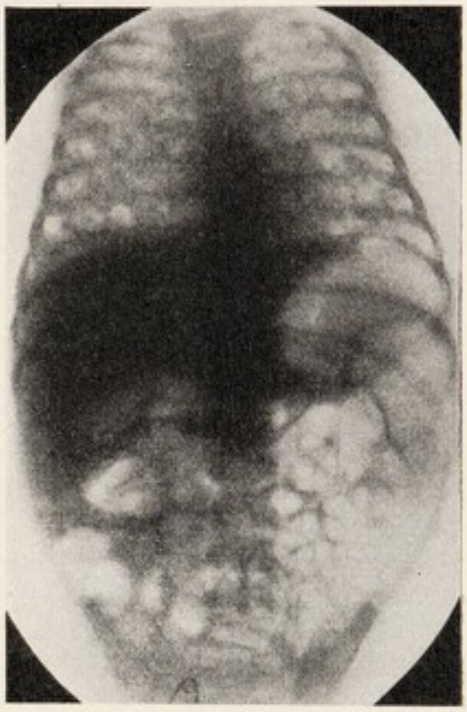

FIG. 75.—Acute miliary tuberculosis of lungs. Infant.

This type is almost always mistaken in the beginning for acute bronchitis, but after a short time it becomes evident that there is some-

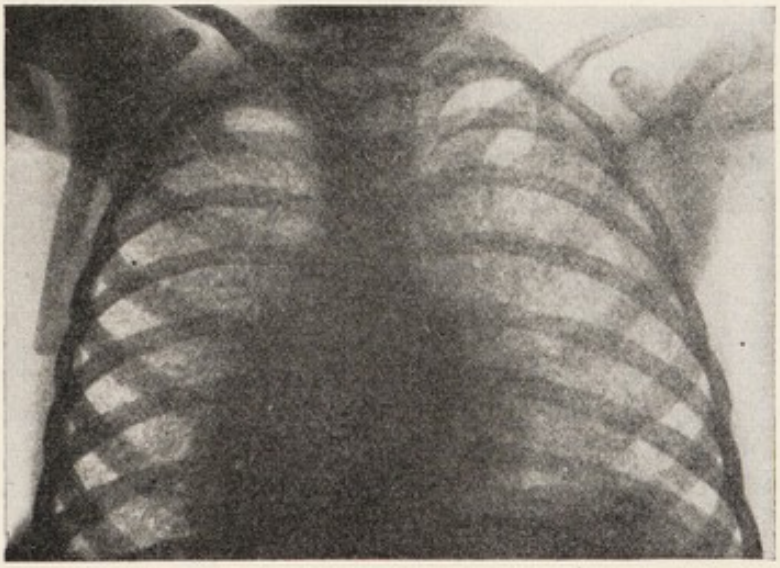

FIG. 76.—Acute miliary tuberculosis of lungs. Child.

thing more serious the trouble than simple bronchitis. The diagnosis depends chiefly on the discrepancy between the severity of the general symptoms and of those pointing to involvement of the lungs and the paucity of the physical signs in the lungs. The presence of an old focus

of tuberculosis somewhere in the body is in favor of acute miliary tuberculosis of the lungs, as is also the absence of leukocytosis. The finding of tuberculides makes the diagnosis positive. The Roentgen ray picture is also almost pathognomonic.

There is, of course, no treatment. Nothing can be done except to make the child as comfortable as possible.

When there is a fairly uniform distribution of miliary tubercles throughout the body, the symptoms are those of a profound general infection, not very different from those of typhoid fever. This form, often spoken of as the "*typhoid type*," is more common in middle and late childhood than in infancy and early childhood. The onset is usually subacute, but sometimes acute. The temperature is irregularly elevated, and may or may not be high. The pulse is relatively rapid when compared with the temperature. Emaciation is rapid and loss of strength progressive. Marked nervous symptoms usually develop. There is usually no distension of the abdomen. There may be either diarrhea or constipation. There may or may not be evidences of bronchitis. The spleen is often slightly enlarged. The white count is usually not increased, but may be. The tuberculin test is almost always negative. Tuberculides can often be found. There should be tubercles in the choroid, but I have seen them in only two cases. The progress of the disease is steadily from bad to worse. Death occurs in a few weeks.

The differential diagnosis is chiefly from typhoid fever. The most important points in favor of miliary tuberculosis are an irregular course of the temperature and a relatively rapid pulse, the temperature in typhoid usually being fairly regular and the pulse relatively slow. The presence of an old focus of tuberculosis is in favor of it. So also is a leukocytosis. The presence of rose spots is strongly in favor of typhoid, while the presence of tuberculides is almost conclusive proof of miliary tuberculosis. So also are tubercles in the choroid. Marked enlargement of the spleen is in favor of typhoid, but does not rule out miliary tuberculosis. A negative tuberculin test and a low white count are of no importance in diagnosis. Bronchitis of any grade may occur in either condition and symptoms of intestinal disturbance may be present or absent in both. A positive Widal test, if it has previously been absent, and the finding of typhoid bacilli in the feces or blood are, of course, conclusive evidence in favor of typhoid.

Treatment, as in all forms of miliary tuberculosis, can be only for comfort.

Chronic Diffuse Tuberculosis

Chronic diffuse tuberculosis, like acute miliary tuberculosis, is always secondary. The tubercle bacilli are distributed throughout the organism in the same way as the result of the breaking down of a tuberculous focus and the entrance of bacilli into the circulation. The difference between these conditions is presumably simply in the number of bacteria. In acute miliary tuberculosis large numbers of bacteria are set free and death occurs before the process which they set up has had time to progress beyond the stage of the miliary tubercle. In chronic diffuse tuberculosis a relatively small number are set free and they have time to develop and cause fairly extensive changes before death occurs. In chronic diffuse tuberculosis the lesions vary roughly in size from that of a small shot to that of an English walnut, most of them being about the size of peas or marbles. Many of them are broken down and caseous. They

are most numerous in the lymph nodes and lungs, next in the liver, spleen and kidneys, and may be found in any of the organs. There may be one or several in a single organ. This form of tuberculosis is almost never seen after infancy.

Symptomatology.—The earliest symptom usually noted is failure to gain in weight and strength without apparent cause. Loss of weight and strength continue without sufficient evidences of disturbance of the digestive tract to account for them, and in spite of careful regulation of the diet. The appetite usually drops off. Signs of disturbance of the digestion may or may not develop. There is often some cough. The color becomes pale, but the blood does not show anything more than the changes of mild secondary anemia. The babies are usually quiet and inclined to be apathetic rather than fussy and irritable. The temperature is ordinarily irregularly, but slightly, elevated. It is seldom high and often normal or subnormal for considerable periods. The course is progressively downward and death occurs in a few months, or at any rate within a year.

It is evident that the lesions are too small to cause any appreciable enlargement of the liver, spleen or kidneys. The liver is often enlarged, however, from coincident fatty change, and the spleen may be somewhat enlarged from simple hyperplasia. Enlargement of the peripheral lymph nodes is, of course, easily recognizable, but, even when the lymph nodes are enlarged, the enlargement is more often due to malnutrition than to tuberculosis. Enlarged retroperitoneal and mesenteric glands can sometimes be felt and enlargement of the tracheobronchial lymph nodes gives the usual signs. The lesions in the lungs are, as a rule, not large enough to give any physical signs. If they do, they are like those of bronchopneumonia. The Roentgen ray, however, often shows areas of solidification which cannot be made out on physical examination. In some instances the lesions in the lungs develop much more rapidly than in others, so that the lung condition becomes the marked one and so dominates the picture that the disease is really a tuberculous bronchopneumonia rather than chronic diffuse tuberculosis. The blood is usually negative except for the evidences of mild secondary anemia and sometimes a moderate polynuclear leukocytosis. The urine is usually normal. The tuberculin test is almost always positive during the early and middle stages of the disease, but toward the end is often negative. Tuberculides are occasionally seen, but never in large numbers.

Diagnosis.—This condition should always be suspected when babies fail to do well without apparent cause or do not respond to proper food and hygienic measures. A continued slight elevation of temperature, even if intermittent, is suggestive, but that is all, because slight elevation of the temperature occurs in many obscure disturbances at this age. Enlargement of the peripheral lymph nodes is usually not of importance, as they are enlarged in all disturbances of nutrition in infancy. Enlargement of the internal lymph nodes, which can be recognized, is of importance, but may be due to other causes as well as to tuberculosis. Enlargement of the liver and spleen is of no special diagnostic importance, as both are likely to be enlarged in all disturbances of nutrition in infancy. Even the presence of evidences of solidification in the lungs is not conclusive proof, as bronchopneumonia is a common occurrence in all feeble babies. The tuberculin test is of great importance and, when positive under these conditions, in infancy is almost certain proof that the baby has chronic diffuse tuberculosis. It is not possible, however, to rule out

chronic diffuse tuberculosis because the tuberculin test is negative, if the baby is very weak and prostrated. The finding of tuberculides makes the diagnosis practically positive. The Roentgen ray may be of assistance early in the course of the disease in increasing the probability of this diagnosis by showing enlargement of the tracheobronchial lymph nodes and areas of solidification in the lungs otherwise undiscoverable.

Treatment.—There is no curative treatment. The most that can be done is to prolong life by great attention to the details of the diet and care.

Tuberculosis of the Lymph Nodes

(Glandular Tuberculosis)

Any or all of the lymph nodes of the body may be attacked by tuberculosis. Glandular tuberculosis is always a secondary lesion. In many instances, however, the primary lesion is insignificant or has been healed, so that the disease of the gland appears to be primary.

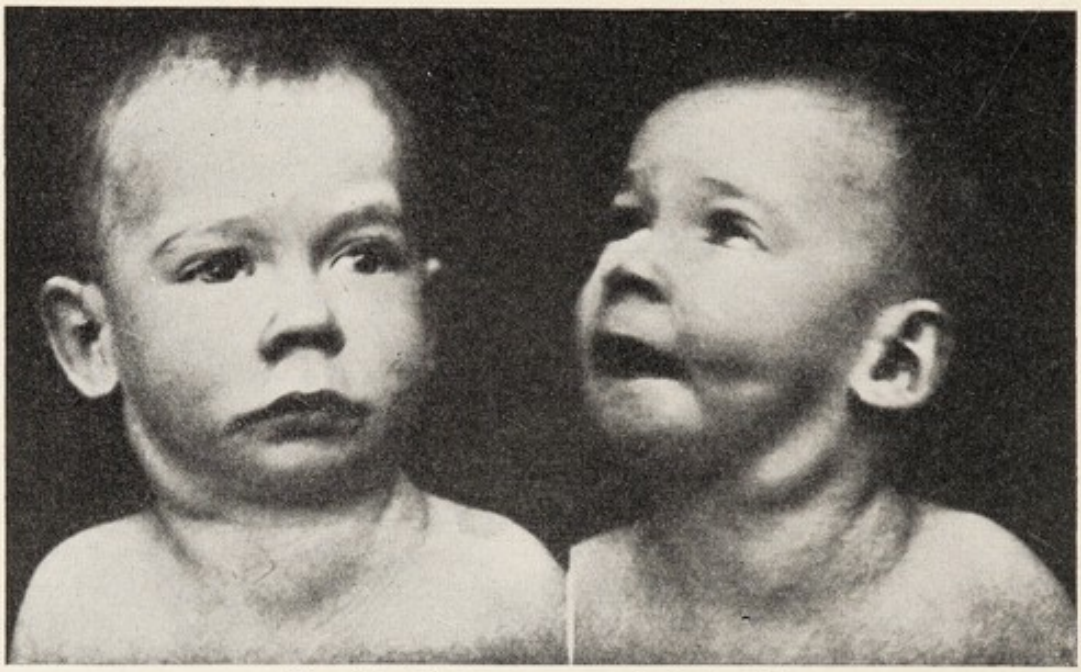

Fig. 77.—Tuberculous cervical adenitis.

General enlargement of the peripheral lymph nodes is almost never tuberculous, while local enlargements frequently are. It is true that general enlargement of the peripheral lymph nodes is often found in chronic diffuse tuberculosis and also in connection with advanced tuberculosis in other organs. The enlargement is, in most instances, however, simply a manifestation of the malnutrition resulting from the tuberculosis. It is due to simple hyperplasia of the glands, which show no tuberculous changes.

Tuberculosis of the Cervical Lymph Nodes.—This has been discussed under the head of cervical adenitis.

Tuberculosis of the Tracheobronchial Lymph Nodes.—These nodes are more often affected in tuberculosis in early life than any other portion of the body. It is safe to say that they are involved in every case in which the infection takes place through the respiratory tract and in most instances in which it takes place through the alimentary tract. They are involved in every case of chronic diffuse tuberculosis, and are

the starting point of almost every case of pulmonary tuberculosis. In mild tuberculous infections, in which the symptoms are entirely general and no evidences of local disease can be found, the seat of the trouble is most often located in these glands.

Physical Examination.—Enlargement of the tracheobronchial lymph nodes is almost always first shown by a change in the character of the respiratory and voice sounds heard over the dorsal vertebral spines. Under normal conditions in infancy and childhood, the bronchial sound of the spoken and whispered voice and of the respiration changes to vesicular at or above the first dorsal spine. The whispering sound heard after the spoken voice is also not normally heard below the seventh cervical spine. When there is an increase in the density of the tissues between the trachea and bronchi and the anterior surface of the vertebrae,

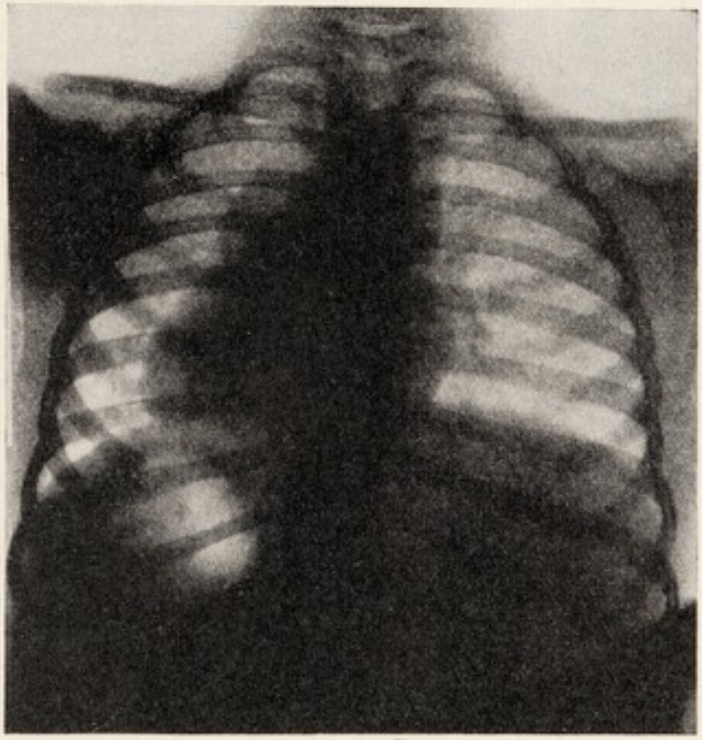

FIG. 78.—Tuberculous tracheobronchial adenitis.

the bronchial sound of the spoken and whispered voice and of the respiration is heard at and below the first dorsal spine. The whispering sound after the spoken voice is also heard below the seventh cervical spine. This persistence of the whispering sound after the spoken voice below the seventh cervical spine is what was described by D'Espine and is that to which the term, *D'Espine's Sign*, should be limited. In some instances one of these changes occurs earlier than the others; in other instances, the sequence is different. All have the same value. They merely show that there is an increase in the density of the tissues between the trachea and bronchi and the vertebrae. This increase in density is usually, but not always, due to enlargement of the tracheobronchial glands. This enlargement may or may not be due to tuberculosis. Dullness over the dorsal spines usually appears later than the changes in the voice sounds and respiration, and is of the same significance. Dullness between the scapulae, interscapular dullness, is a late manifestation and denotes considerable increase in the newly-formed tissue. Dullness under the manubrium means extreme enlargement or the formation of an abscess.

When there is increase in the size of the tracheobronchial glands, the Roentgen ray shows a broadening of the shadow at the base of the heart and shadows at the sides of the heart, unless the increase in size is behind the heart. If the glands are calcified, the shadows are, of course, denser. Great care must be taken, however, not to confuse the shadow of the thymus with that of the tracheobronchial glands. Furthermore, the normal variations in the breadth of the shadows at the base of the heart must not be forgotten. It is also very easy to mistake the shadows of the bronchial tree for those of enlarged nodes. In my experience it is very difficult, even for an expert, to determine with the Roentgen ray whether the glands are enlarged or not, unless the enlargement is considerable. Marked enlargement is, of course, easily recognized. So also is a mediastinal abscess when the glands have broken down.

If the enlargement of the glands is considerable, it may exert pressure on the trachea or one or both bronchi and result in diminution of the respiratory sound on one or both sides of the chest. When the sound is diminished to the same extent on both sides, the diminution is, of course, unrecognizable. The pressure may be so great as to cause dyspnea, which is usually more marked in expiration. The head is also sometimes hyperextended. Pressure on the superior vena cava may also cause cyanosis, and occasionally edema, of the face and upper extremities, while pressure on the esophagus may cause difficulty in swallowing. Pressure on or irritation of the pneumogastric and recurrent laryngeal nerves may cause cough, dyspnea and change in the voice. These more marked signs of enlargement and pressure are, however, very uncommon and seldom develop unless the glands have broken down and an abscess has been formed.

Symptomatology.—The symptoms of tuberculosis of the tracheobronchial lymph nodes are, as a rule, simply those of a mild disturbance of the nutrition and general well-being. The child does not seem as well as usual, is not as lively, is easily tired, irritable and hard to live with. It does not get on as well at school. Its appetite drops off and it fails to gain or loses weight. The digestion is not infrequently somewhat disturbed. A baby does not smile and coo as much as usual and is less active. Pallor or sallowness of the skin develops, and the skin and hair are dry. The temperature is a little elevated, especially at night, sometimes continuously, sometimes intermittently. There is usually no cough and also never any sweating. Incidentally, sweating is a very uncommon symptom in tuberculosis of any sort in early life.

If the enlargement of the glands is greater and there is pressure on the trachea, bronchi or nerves, there is often cough. There is usually nothing characteristic about it. It is almost always dry. Sometimes it is very persistent and occasionally paroxysmal, resembling very much that of whooping-cough. Dyspnea may also develop. This also may be paroxysmal. Sometimes it is like that in croup; at others, it suggests asthma.

The glands may break down and form an abscess in either the anterior or posterior mediastinum. When this happens the physical signs are more marked, as are also the symptoms of pressure. In rare instances, however, the abscess may be small and cause no symptoms of pressure. The constitutional symptoms are, of course, more marked and the temperature higher. The abscess may break through either into the trachea or a bronchus and cause death by suffocation, although in rare instances recovery ensues after the pus is coughed up. It may also break into

the lungs, esophagus or one of the vessels. It may occasionally rupture externally.

Prognosis.—It is very unusual, however, for the glands to become large enough to cause these marked symptoms of pressure or to break down and form an abscess, because death usually occurs as the result of extension of the disease to the lungs or of acute miliary or diffuse tuberculosis before sufficient time has elapsed. It is probable that in the vast majority of instances of involvement of the tracheobronchial lymph nodes the symptoms are never noted, and recovery takes place without the condition ever having been suspected. When the condition is suspected, and even when there are definite physical signs, recovery is the rule, if the child is properly cared for. Indeed, recovery often takes place, even when little or no care is taken. In many instances, however, the tuberculous process extends from the glands to the lungs. In fact, in infancy and early childhood, tuberculosis of the lungs is almost invariably a sequela of tuberculosis of the tracheobronchial lymph nodes. Tuberculous tracheobronchial lymph nodes are also the most common source of the infection in acute miliary tuberculosis and chronic diffuse tuberculosis.

Diagnosis.—Whenever a baby or child is not thriving, does not seem well, and there is no apparent cause, tuberculosis of the tracheobronchial lymph nodes should be thought of as a possible explanation of the symptoms. If there is also a little fever, it should be considered more seriously. It must not be forgotten, however, that there are many other causes for disturbance of the nutrition and general condition; insufficient or improper food, lack of air and sunlight, late hours, excitement, fatigue from both physical and mental overexertion, as well as other obscure diseases, especially those of the fauces, nasopharynx and their adnexa. It must also be remembered that it takes but little to cause an elevation of the temperature in early life. Fatigue, excitement, slight indigestion, slight chronic inflammation of the nose and throat, are all sufficient. Furthermore, it must not be forgotten that the temperature in the mouth is almost never exactly 98.6° F. Under normal conditions it is almost always lower than that in the morning and frequently reaches 99° F. in the afternoon. The temperature, when taken in the rectum, cannot be considered abnormal unless it is over 99.6° F.

The presence of any of the physical signs of enlargement of the tracheobronchial lymph nodes or of findings in roentgenograms suggestive of their enlargement, under these conditions, is suggestive that the enlargement is due to tuberculosis. It does not prove it, however, because enlargement of the tracheobronchial lymph nodes may be due to other causes, such as simple hyperplasia from acute or chronic inflammation in the tracheobronchial tract from any cause. It is very common in connection with and after measles, whooping-cough and influenza.

If the tuberculin test is positive, there are no other evident foci of tuberculosis in the body and no other cause for the symptoms can be found, there is a reasonable probability that the trouble is tuberculous tracheobronchial adenitis, even if there are no physical signs of such enlargement. If there are such signs, or the Roentgen ray shows evidence of the enlargment, the chances are that this is the trouble. Even under these conditions, however, it is possible that the enlargement of the tracheobronchial glands is not tuberculous and that the positive reaction is due to some hidden focus elsewhere. If the tuberculin test is negative after

several trials, it is certain that the symptoms are not due to tuberculous tracheobronchial adenitis.

When a paroxysm of cough is due to whooping-cough, it usually ends with the expulsion of a large amount of mucus or in vomiting; when it is due to enlarged tracheobronchial glands, there may be vomiting, but never the expulsion of a large amount of mucus. When it is due to whooping-cough, there is also likely to be a history of exposure to whooping-cough or of other cases in the family or vicinity. Evidences of enlargement of the tracheobronchial glands are not of much help, because they are also likely to be enlarged in whooping-cough. The value of the tuberculin test has already been discussed. Paroxysmal cough may also be due to non-tuberculous enlargement of the tracheobronchial lymph nodes. Such enlargement is likely to occur in whooping-cough, measles and influenza or in the course of simple bronchitis and bronchopneumonia.

There is nothing pathognomonic about the symptoms resulting from pressure on the neighboring organs. They may be caused also by enlargement of the glands from simple hyperplasia, leukemia or pseudoleukemia, or by new growths of any sort in the mediastinum. Abscesses in the mediastinum may be non-tuberculous, or, if tuberculous, may originate in disease of the bones as well as in tuberculosis of the glands. The physical signs are of no help in diagnosis. The Roentgen ray may be, if it shows disease of the bones. The diagnosis must depend on the findings in the blood, the tuberculin test and the presence or absence of evidences of malignant disease or pseudoleukemia elsewhere.

Treatment.—The treatment is, in general, the same as that of tuberculosis anywhere in early life. It is possible that the ultraviolet rays may be of some assistance. It is very difficult to tell whether they do good or not, because other methods of treatment are always also used at the same time. They are, however, worthy of trial. Little or nothing can be hoped for from surgery, even when pressure symptoms are very marked, because of the situation of the glands. Sometimes, however, it is possible to open and drain an abscess in the mediastinum.

Tuberculosis of the Retroperitoneal and Mesenteric Lymph Nodes.—Tuberculosis of the abdominal lymph nodes is much less common than that of the tracheobronchial, but occurs far more often than is usually thought. When it is not a part of chronic diffuse tuberculosis, the infection presumably almost invariably comes through the intestinal tract. It is also presumable that, as in the case of the lungs in tuberculosis of the tracheobronchial lymph nodes, there is a primary focus in the intestines, which later heals.

Symptomatology.—The symptomatology of tuberculosis of the abdominal lymph nodes is, in most instances, much the same as that of tuberculosis of the tracheobronchial lymph nodes when they have not enlarged enough to cause pressure, that is, the symptoms are those of general disturbance of the nutrition. It is very seldom that tuberculosis of the abdominal glands causes any symptoms pointing directly to disease of the digestive tract. They seldom become enlarged enough to cause any interference with absorption through the lymphatics. Not infrequently, however, they cause pain and abdominal discomfort. In rare instances they may be enlarged sufficiently to mechanically interfere with the passage of the intestinal contents and produce constipation. In still rarer instances they break down and form a retroperitoneal or mesenteric abscess.

Physical Examination.—Enlarged tuberculous mesenteric glands are usually easily palpable, if the abdominal wall is relaxed. It is impossible to feel either them or enlarged retroperitoneal glands unless it is. There may be only two or three glands or many. They vary in size from that of a small bean or pea to that of an English walnut. They feel elastic or hard, are usually regular in outline, are almost always somewhat movable, and are seldom tender. They may be felt anywhere in the abdomen. Enlarged retroperitoneal glands are most often felt in the right iliac fossa or in the median line in the neighborhood of the navel. They are, of course, always deep down. It is difficult to feel them unless they are at least as large as marbles. They are usually missed, moreover, unless there are a number together. They are not movable, but are more often tender than are mesenteric glands. In rare instances they may be felt through the rectum. The Roentgen ray seldom shows anything definite, unless the glands are calcified. When an abscess has been formed, the signs are those of an encapsulated collection of fluid; usually in the median line and fairly superficial, if in the mesentery; deep down and usually in the right iliac fossa, if retroperitoneal.

Diagnosis.—Symptoms of disturbance of nutrition and of the general condition, as well as elevation of the temperature, should suggest the possibility of tuberculosis of the abdominal lymph nodes just as of the tracheobronchial lymph nodes. The value of such symptoms and of a slight elevation of the temperature is the same in both instances and has been spoken of in discussing the diagnosis of tuberculosis of the tracheobronchial lymph nodes. If no enlargement of either the abdominal or tracheobronchial glands is made out, no other cause for the symptoms is detected and the tuberculin test is positive, the chances are that, if the symptoms are due to glandular tuberculosis, it is of the tracheobronchial rather than of the abdominal lymph nodes. If there is pain or discomfort in the abdomen, the chances are rather in favor of tuberculosis of the abdominal glands. If the enlarged glands can be felt and the tuberculin test is positive, a probable diagnosis of tuberculosis is justifiable. If the tuberculin test is negative, it is not justifiable, and the enlargement of the glands must be attributed to some other cause. Such causes are inflammation of the intestines, resulting in simple hyperplasia, malignant disease, leukemia and pseudoleukemia. There is nothing about the feeling of the glands which is in any way different from that when the enlargement is due to other causes. Masses of feces in the intestine may also be mistaken for enlarged mesenteric glands. Masses of feces are usually more movable and less regular in outline than enlarged glands. They can sometimes be changed in shape by pressure, while enlarged glands can not. They disappear after the bowels are thoroughly cleaned out, while enlarged glands usually become more evident.

When enlarged tuberculous retroperitoneal glands cause pain and are tender on palpation, the condition may be mistaken for either acute or chronic appendicitis. The diagnosis is often very difficult, if the enlargement of the glands has not been previously recognized. In general, when the symptoms are acute, there is more likely to be a polynuclear leukocytosis in appendicitis than in tubercular adenitis. The temperature is likely to be higher and the child to appear sicker. When the symptoms are not acute, the tuberculin test is of great assistance, if negative, and of some assistance, if positive. Roentgenograms after an opaque meal are often of considerable help in showing whether there is or is not trouble about the appendix.

When tuberculous mesenteric glands have broken down to form an abscess in the mesentery, it may be mistaken for other cysts in the same location. The diagnosis is practically impossible without an operation. The disturbance of nutrition is likely to be greater, however, in the case of tubercular abscesses than with other cysts. It may also be confused with an ovarian cyst. In general, an abscess is nearer the median line and higher up than an ovarian cyst. Rectal examination may also help in determining the origin of the tumor. When a mass of retroperitoneal glands has broken down and formed an abscess, it may be mistaken for an appendicial abscess or a psoas abscess. When the trouble is a psoas abscess, evidences of disease of the spine are easily made out both on physical examination and with the Roentgen ray.

Prognosis.—The prognosis of tuberculosis of the abdominal lymph nodes is essentially the same as that of the tracheobronchial lymph nodes. In the vast majority of cases recovery takes place without any suspicion of the condition having arisen. This is also the case when it is suspected, but no enlarged glands are felt. Even when the glands are palpable, most cases recover under careful treatment, and many without any special treatment. I have known many glands to disappear. Some of these were in cases in which they had been proved at operation to be tuberculous. In other instances they become calcified. I have seen this proven by the Roentgen ray and by operations for other conditions in after years. In other cases the process extends to the peritoneum, and in still others a broken down gland may be the source of infection in acute miliary or chronic diffuse tuberculosis. It is very seldom that a retroperitoneal or mesenteric abscess develops. Tuberculosis of the abdominal glands is almost never, of itself, a fatal disease.

Treatment.—The treatment is essentially the same as that of tuberculosis of the tracheobronchial lymph nodes. Exposure of the abdomen to sunlight and the ultraviolet rays probably does some good. It is very difficult to determine whether or not it does good, however, because other methods of treatment are also always employed. It is advisable in laying out the diet to be certain from the examination of the stools that there is no interference with the absorption of fat as the result of the involvement of the mesenteric nodes. If there is, the fat in the food must be limited. Removal of the glands is inadvisable, unless they are causing pressure. Abscesses should, of course, be operated upon.

Tuberculosis of the Peritoneum

(Tuberculous Peritonitis)

Tuberculous peritonitis is not common at any period of early life, but is rarer in infancy than later. It is always secondary to some tuberculous focus elsewhere. Except when tuberculosis of the peritoneum is a part of acute miliary tuberculosis, when it causes no symptoms, the infection is directly from neighboring organs through the lymphatics, not through the blood. The focus is usually in the mesenteric or retroperitoneal lymph nodes, not in the intestines, tuberculous ulceration of the intestines being very unusual in early life. For practical purposes the condition is always chronic.

The pathologic process is of three types. In the first, the peritoneum is studded with miliary tubercles and there is a profuse serous exudation. In the second, the process is more chronic and is characterized by the formation of fibrous tissue, which results in firm fibrous adhesions which

bind the coils of intestines to each other and to the omentum, other abdominal viscera and walls. In this form there is usually little or no exudation. In the third, large caseous masses are formed which tend to break down and form a purulent exudate. This type is often mixed with the fibrous, or adhesive, type, so that the exudate is usually encapsulated or sacculated. The symptomatology, physical signs, and prognosis differ considerably in the three types, which are spoken of as the ascitic; adhesive, or fibrinous; caseous, or ulcerative.

Ascitic Type.—This type is said to be less common than the others, but I have seen it at least as often. In some instances the first thing noticed is enlargement of the abdomen. In most cases, however, the

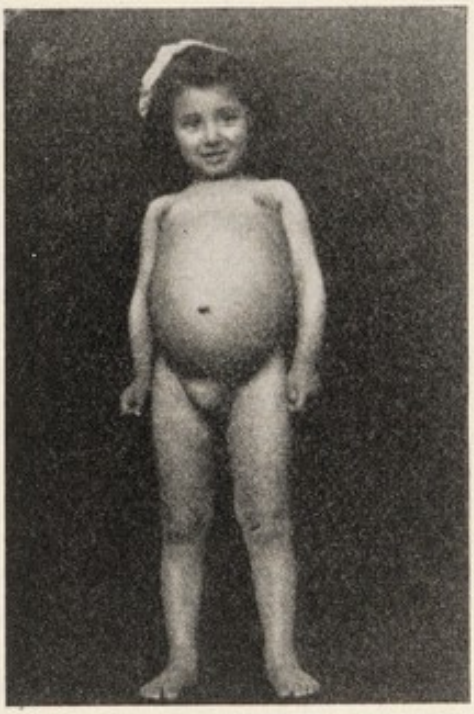

Fig. 79.—Tuberculous peritonitis. Ascitic type.

Fig. 80.—Tuberculous peritonitis. Ascitic type.

enlargement has been preceded by some weeks, or even months, of failing health, the symptoms being much like those of tuberculosis of the tracheobronchial or abdominal lymph nodes. Usually they do not point toward the abdomen as the seat of the trouble. With the increase in the size of the abdomen, there is likely to be loss of appetite and abdominal discomfort, but seldom pain. Vomiting is unusual. The bowels may move normally or there may be either diarrhea or constipation. There is loss of weight, strength and color, but the loss of weight is not as rapid as would be expected. There is nothing constant about the temperature. It may be irregularly high, more or less continuously and moderately elevated, or practically normal for considerable periods. The leukocyte count is normal or slightly increased. The blood usually shows a mild secondary anemia. The urine is normal, except that it may show the changes due to passive congestion from pressure. The tuberculin test is positive.

Physical Examination.—The physical signs are those of free fluid in the abdominal cavity, varying, of course, in accordance with the amount

of fluid in the cavity. The fluid, being an exudate, has a specific gravity of above 1,015 and contains, as a rule, more than 4% of albumin. It contains many cells, most of which are lymphocytes. Tubercle bacilli may often be found in it and animal inoculations are almost always positive. It is almost invariably straw colored and serous, but may sometimes be slightly hemorrhagic.

Diagnosis.—This type of tuberculous peritonitis ought not to be confused with a hydronephrosis or an ovarian cyst, because in them the fluid is not free in the abdominal cavity. In hydronephrosis the dullness is on one side and does not change with change in position. With an ovarian cyst the fluid is in the centre or a little to one side, there is very little change in the dullness with change of position and the upper border of the dullness is convex, not concave as when the fluid is free.

When there is free fluid in the abdomen, the physical signs are the same, whatever the cause. If the ascites is the result of passive congestion from disease of the heart or lungs, there are other evidences of disease of these organs and usually edema elsewhere. If it is due to obstruction to the portal circulation, other evidences of disease of the liver or of pressure on the portal vein are always present. The ascites due to the peculiar form of cirrhosis of the liver resulting from adherent pericardium, which often develops very insidiously and is the first symptom noted, must always be kept in mind. With it the liver is always much enlarged and the spleen usually, although the evidences of an adherent pericardium may be very slight. When the ascites is due to disease of the kidneys, there is almost always edema elsewhere and the urine shows the characteristic changes. The tuberculin test is positive in tuberculous peritonitis. It may or may not be positive in these other diseases, according to whether there is or is not a tuberculous focus somewhere in the body. The presence of a recognizable focus of tuberculosis elsewhere is, of course, evidence in favor of tuberculous peritonitis. Furthermore, in all these other conditions the fluid is a transudate with a specific gravity below 1,015 and contains, as a rule, less than 2% of albumin, while in tuberculous peritonitis it is an exudate with a specific gravity above 1,015 and contains, as a rule, more than 4% of albumin.

The physical signs of *chronic, non-tuberculous peritonitis* are the same as those in this type of tuberculous peritonitis. The course is usually slower and the temperature lower. These are only differences in degree, however, and are not of much value in diagnosis. A negative tuberculin test practically rules out tuberculous peritonitis. A positive test is somewhat in favor of it. The fluid is an exudate in both. That in tuberculous peritonitis may contain tubercle bacilli, that in chronic non-tuberculous peritonitis never does.

Chronic indigestion with marked dilatation of the intestines and distention of the abdomen is sometimes mistaken for tuberculous peritonitis with a small amount of fluid, because, if there is a considerable amount of liquid feces in the intestines, it often causes dullness in the flanks and lower abdomen. If the abdominal walls are relaxed, the dullness may be shifting. I have, in a few instances, also obtained an indistinct fluid wave. The development of the enlargement of the abdomen is always slower in indigestion than in tuberculous peritonitis and there are always marked symptoms of indigestion. The signs of fluid disappear after the bowels are freely moved. The tuberculin test is negative in indigestion, unless there is a tuberculous focus somewhere in the body, while it is positive in tuberculous peritonitis.

Prognosis.—The prognosis is not as bad in tuberculous peritonitis as is usually supposed. The younger the patient, however, the smaller are the chances of recovery. It is better in this type than in the others. Provided the child does not die of acute miliary tuberculosis or of some tuberculous process elsewhere, there is a reasonable chance of recovery. In my experience, recovery is as likely to occur under medical as under surgical treatment. Cases that do badly are likely to die within a few months. In a general way, the longer they live the better are the chances for ultimate recovery.

Treatment.—The treatment is, of course, mainly that of tuberculosis in general. Care must be taken in regulating the diet to determine from the stools that the capacity for the absorption of fat is not overstepped. Cod liver oil and other fats may do much harm if given in excess. Heliotherapy, both local and general, is especially useful in tuberculous peritonitis. I am very sceptical as to the value of the injection of nitrogen gas into the abdominal cavity in these cases. Furthermore, my own experience leads me to believe that the chances of recovery are as good, if not better, without operation. Unless the distention of the abdomen is extreme, it is wiser not even to tap it. If it is extreme, it should be tapped and a part of the fluid allowed to escape. I doubt if it is advisable to let it all run off. If the fluid continues to accumulate after several tappings and the pressure causes much discomfort and passive congestion in other organs, it is probably advisable to open the abdomen. It is not at all certain, however, that the fluid will not return after the operation.

The Adhesive and Caseous Types.—The onset is usually slower in these than in the ascitic type. There are the same general indications of malnutrition and of indefinite illness. Abdominal pain is more common, especially in the adhesive form. It is probably more often due to interference with the passage of gas than to inflammation. There is almost always loss of appetite, but seldom vomiting. Constipation is more common than diarrhea. In rare instances there may be ulceration of the intestines, sometimes tuberculous and sometimes not, and, as the result, blood, mucus and pus in the stools. In other cases adhesions may cause intestinal obstruction. In still other cases the caseous masses may break down to form abscesses, which may discharge into the intestines or externally. If they discharge externally, it is most often through the navel. The temperature is usually not much elevated in the adhesive type, and may be normal for long periods. It is generally higher in the caseous type. It is almost always irregularly high, if abscesses have formed. The tuberculin test in almost invariably positive, but in rare instances may be negative. The leukocyte count is usually normal, but may be moderately elevated. The urine is normal, unless there is passive congestion of the kidneys from pressure or secondary amyloid changes in them.

Physical Signs.—The abdomen is usually enlarged, but seldom as much as in the ascitic type. The enlargement is not infrequently somewhat irregular. The irregularity may be due to encapsulated collections of fluid, large, caseous, tuberculous masses or dilated coils of intestines. In many instances the dilated coils can be seen through the abdominal wall. Not infrequently there is visible peristalsis. Occasionally the abdomen is retracted as the result of intestinal adhesions and compression of the bowels. I have seen the whole of the intestines so bound together and retracted that they could be held easily in one hand. There may be a protrusion at the navel, with redness, if an abscess is pointing there.

In many of the cases the rolled up and thickened omentum can be felt running across the epigastrium. The lower border is usually at about the level of the navel. It may be mistaken for the liver. The surface of the liver, however, is smooth and the edge sharp, that of the tuberculous omentum is irregular and the lower border rounded. The liver moves downward with inspiration, the omentum does not. Careful examination, moreover, almost always shows that there is a space between the upper border of the omentum and the ribs or lower border of the liver. Tuberculous masses, probably originating in the mesenteric or retroperitoneal glands, may be felt anywhere in the abdomen. They are more common in the left half, however, than in the right. They are irregular in outline, not tender, do not pit on pressure, are seldom movable and may be either superficial or deep. Sometimes they fill up from one third to one fourth of the abdominal cavity. They may be confused with masses of feces, tumors of the kidneys or an enlarged spleen. Masses of feces are more movable, may pit on pressure and disappear when the bowels are cleaned out. Tumors of the kidney can always be felt in the loin and can be pushed forward; tuberculous masses do not extend as far back and can not be pushed forward. An enlarged spleen is superficial, smooth, rounded and comes out in the abdomen from beneath the ribs; tuberculous masses are deep, irregular in outline and do not extend under the ribs.

The percussion note is very variable. There is dullness or flatness over tuberculous masses on light percussion, if they are superficial; dullness on hard percussion, if they are deep. There also may be dullness or flatness from collections of fluid. This may be anywhere. Shifting dullness is very unusual, as the fluid is almost never free. All varieties and modifications of the tympanitic note may be elicited over the intestines, according to the difference in tension in the different coils.

Tuberculous disease of the testicle or epididymis is not uncommon in tuberculous peritonitis of these types and should always be looked for. Signs of disease of the testicle are very strong evidence in favor of tuberculous peritonitis in doubtful cases. Tuberculides are seldom present in these types. Evidences of tuberculosis in other organs are not uncommon.

Diagnosis.—The general principles of diagnosis are the same as in the ascitic type. These types are, however, more likely to be mistaken, in the early stages, for indigestion because of the abdominal pain and the tendency to constipation. The pure adhesive type is often unsuspected for a long time, because of the lack of physical signs. When the omentum is rolled up and enlarged and there are palpable masses in the abdomen the diagnosis is usually easy. Possible causes of error have been discussed under physical examination. Malignant disease in the abdomen is so rare at this age that it hardly needs to be taken into consideration in differential diagnosis.

Prognosis.—The prognosis is not as good in these types as in the ascitic. It is better in the adhesive than in the caseous or mixed types. The course is longer in them than in the ascitic type. Death seldom occurs under six months, especially in the adhesive type, and may be delayed for several years. Recovery is not very unusual in both types. I have repeatedly seen large tuberculous masses and what were almost certainly enlarged, rolled up omenta entirely disappear in the course of months or years. Moreover, the symptoms presumably due to adhesions of the intestines have entirely ceased in other cases. Their disappearance must have been due to the absorption of the adhesions. Recovery almost never occurs, however, if there is ulceration of the intestines or

abscesses develop. Recovery is most unusual if adhesions have formed which cause obstruction. I have, however, known cases to recover after operations to relieve the obstruction. The presence of tuberculous lesions in other organs, especially in the lungs, makes the outlook practically hopeless.

Treatment.—The treatment is essentially the same as that of the ascitic type. There being more often interference with the absorption of fat in these types, it is especially important not to overstep the limit of tolerance in the diet. Opening the abdomen cannot possibly do any good in the adhesive form, unless there is obstruction from bands or adhesions which can be cut or freed. It is impossible to break up all the adhesions and they will form again, if broken up. If the exposure of the peritoneum to the air does good in the ascitic form, which is very doubtful, it cannot do good in this form, because the adhesions prevent the exposure. Opening the abdomen cannot do any more good in the caseous form, unless abscesses have formed, which can be drained. It is impossible to remove large caseous masses, or even small ones, without doing more harm than good.

Tuberculosis of the Lungs

Pulmonary tuberculosis in infancy and early life differs materially both in its pathologic changes and its symptomatology from pulmonary tuberculosis in late childhood and adult life. The adult type is seldom seen before five years and the infantile type almost never after ten years. The explanation of the difference in type is that in early life the infection of the lungs is almost invariably secondary to tuberculosis of the tracheobronchial lymph nodes through the lymphatics, while in adults it is through the inspired air. The earliest lesions are likely to be, therefore, at the roots of the lungs in early life and at the apices in later life. The tendency of tuberculosis in early life is to form multiple and in later life single lesions. In the former it is toward the formation of caseous masses, in the latter toward fibroid changes and the development of cavities. The course of pulmonary tuberculosis in middle and late childhood, being essentially the same as in adult life, does not need to be described. What follows applies, therefore, only to tuberculosis of the lungs in infancy and early childhood.

Symptomatology.—It is utterly impossible to either intelligibly or adequately describe the manifold manifestations of tuberculosis of the lungs in early life. It is safe to say that tuberculosis may closely resemble any of the other diseases of the lungs and that, when there is disease of the lungs, the chances are that it is not tuberculosis. *Acute miliary tuberculosis of the lungs* has already been described. *Tuberculous bronchitis* is not at all common. There is nothing about either the symptoms or physical signs to distinguish it from any other form of bronchitis. In general, the symptoms are not very severe nor the physical signs very marked. The tuberculin test is usually positive and there is, as a rule, none, or only a slight leukocytosis. The value of these points is the same as in other tuberculous conditions. If tubercle bacilli are found in the sputum, the diagnosis is, of course, evident. If they are not, the nature of the trouble is almost never suspected. It is probable that in the great majority of cases tuberculous bronchitis amounts simply to the first stage of tuberculous bronchopneumonia. It is likely that recovery sometimes takes place. How often this occurs is problematical, because the sputum is seldom examined for tubercle bacilli in

bronchitis in early life. It is also likely that death may result before the development of bronchopneumonia.

Tuberculous Bronchopneumonia.—This may be acute, subacute or chronic. The acute type occurs almost entirely in infancy and the subacute in infants and young children. The chronic may occur at any age. The subacute is by far the most common form. The acute type usually appears clinically to be a primary disease. The subacute type is quite likely to follow, either immediately or after a short time, measles, whooping-cough and influenza. It apparently may also be engrafted upon a simple bronchitis or bronchopneumonia. In such instances, however, the chances are that the process was tuberculous from the beginning. The same is also true of the chronic form.

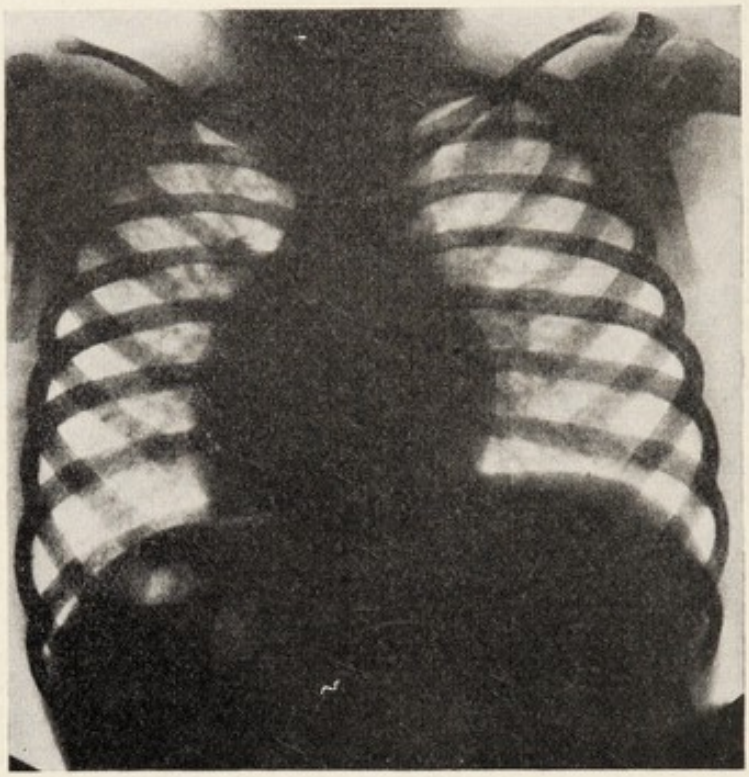

Fig. 81.—Early tuberculous lesions at roots of lungs.

Acute Tuberculous Bronchopneumonia.—There is nothing about the symptoms and physical signs of acute tuberculous bronchopneumonia to justify a positive diagnosis from simple acute bronchopneumonia. It is perhaps true that, in general, the temperature is more irregular and the remissions in the fever more marked than in non-tuberculous bronchopneumonia. It may also be true that the symptoms of intoxication are less marked until toward the end. These are only differences in degree, however, and, while they may be true in general, are of little help in the individual case. The presence of evidences of enlargement of the tracheobronchial lymph nodes is somewhat in favor of tuberculous bronchopneumonia but does not prove it, because there is often enlargement in simple bronchopneumonia. The absence of such evidences does not count against tuberculous bronchopneumonia. A positive tuberculin test is in favor of it in infancy, but does not prove it. Its absence is not important, because it is often absent in acute tuberculosis. The absence of leukocytosis is in favor of tuberculosis, but is also the rule in influenzal

bronchopneumonia. The presence of a polynuclear leukocytosis is against tuberculous bronchopneumonia, but, again, there often is a leukocytosis in tuberculous bronchopneumonia. The presence of tubercle bacilli in the sputum proves, of course, that the disease is tuberculous bronchopneumonia. They are not present, however, in most cases and are almost never sought for.

The end is probably almost always in death after one or two weeks. No one knows how many recover, because the sputum is almost never examined, and, if tubercle bacilli are not found in the sputum, there is no way of positively determining whether a case of bronchopneumonia is tuberculous or not. The treatment can be no different from that of simple bronchopneumonia.

Subacute Tuberculous Bronchopneumonia.—As in the case of acute tuberculous bronchopneumonia, there is nothing definite enough about either the symptoms or physical signs of subacute tuberculous bronchopneumonia to distinguish it from non-tuberculous bronchopneumonia. It is probably true that the temperature is, as a rule, somewhat more irregular and that it shows greater remissions than in simple bronchopneumonia. It is also probably true that, as a rule, the toxemia is less and the disturbance of nutrition not as marked, at any rate for a time, as in simple bronchopneumonia. Infants and children with tuberculous bronchopneumonia are also likely to be more alert mentally than those equally sick with simple bronchopneumonia. These are, however, only differences in degree, and such differences never justify a positive diagnosis. The bronchopneumonic areas are somewhat more likely to be largely unilateral and to be near the roots of the lungs in tuberculous than in simple bronchopneumonia. More important, however, are the persistence and steady increase in size of the areas, because in non-tuberculous bronchopneumonia the areas of solidificaiion are constantly changing and it is unusual for one to steadily increase in size. Large areas of this sort are likely to be very dull or flat on percussion and to give a more marked sense of resistance than is usually the case in non-tuberculous lesions. Cavities are often found at autopsy, but they are almost never large enough to give any physical signs. The value of evidences of enlargement of the tracheobronchial lymph nodes is even less than in acute bronchopneumonia, because these nodes become enlarged in the course of every prolonged bronchopneumonia. A positive tuberculin test is strong, but not positive, evidence that the bronchopneumonia is tuberculous, if the patient is a baby. It is of less value in a child. A negative test is very strong evidence against the process being tuberculous, but not absolute proof that it is not. It should always be repeated, if negative.

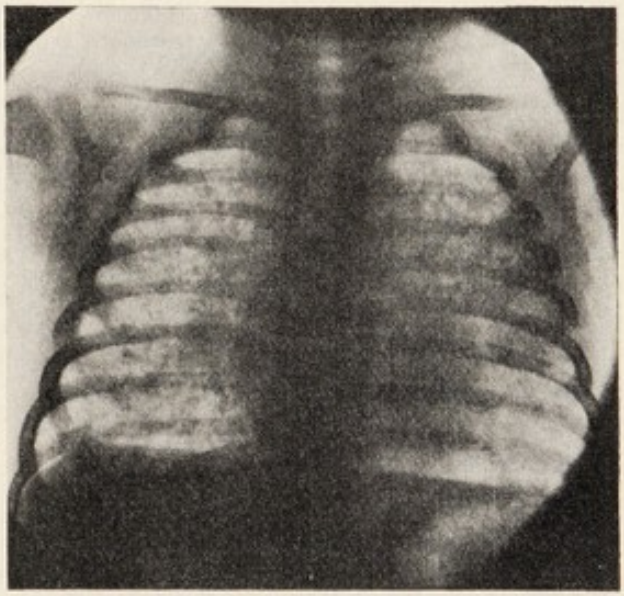

FIG. 82.—Subacute tuberculous bronchopneumonia in an infant of eight months.

In most instances there is progressive failure of strength and weight, until toward the end the baby becomes extremely emaciated and exhausted. Death occurs in from one to three months. Occasionally, however, improvement begins after a time and finally ends in recovery after many months or even years. In such cases the physical signs gradually diminish, but some evidences of disease in the lungs almost always remain. It is presumable that the diminution in the signs, as well as the cure, are due in part to resolution of the affected areas and in part to fibroid changes. Unless tubercle bacilli have been found in the sputum, there is always a question as to whether the child really did have tuberculosis.

The treatment is necessarily a combination of that for tuberculosis in general and of that for simple bronchopneumonia.

Chronic Tuberculous Bronchopneumonia.—The symptoms and physical signs are much the same as those of subacute tuberculous bronchopneumonia, except that their development is slower and their increase more gradual. The duration of the disease is, of course, much longer. The end is almost always in death, but it is probable that the process may stop at any time and recovery finally occur. In all such cases, however, there must be some doubt as to the accuracy of the diagnosis, unless tubercle bacilli have been found in the sputum. In chronic tuberculous bronchopneumonia the pathologic process and, in consequence, the physical signs are likely to be much more marked in one lung than in the other. Just as in subacute tuberculous bronchopneumonia, when the area of solidification is large, it is flat and resistant. It is sometimes mistaken for an empyema. It ought not to be, however, because the location of the area is usually different and there is no displacement of organs. Hemoptysis is very rare, even in this form of pulmonary tuberculosis, in early life. Profuse sweats are unusual. If they occur, it is toward the end.

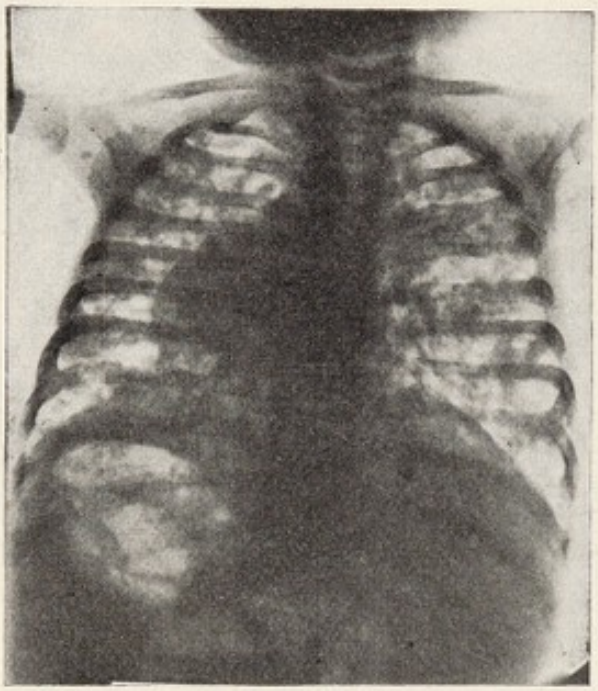

FIG. 83.—Chronic tuberculous bronchopneumonia in an infant of six months.

When recovery takes place, there is some resolution, but it is chiefly through fibrous change. Dilatation of the bronchi and bronchiectatic cavities are likely to result from the contraction of the scar tissue. The physical signs are thus much the same as when organization of the lungs and bronchiectatic cavity formation have followed pneumonia. When there is a question as to the origin of such changes in the lung, the chances are much against tuberculosis. The physical signs and the Roentgen ray are of little assistance in diagnosis. The value of the tuberculin test is as usual—very important, if negative; less so, if positive. The white count is of very little help, because there is almost always secondary infection, even if the original cause is tuberculosis.

The treatment of chronic tuberculous bronchopneumonia is that of tuberculosis in general. If fibroid and bronchietatic changes have developed in the lung, the treatment is the same as when they are due to other causes.

Acute Tuberculous Pneumonia.—This form of pulmonary tuberculosis is very rare, but may occur at any age. I have never seen a case in early life.

Tuberculosis of the Intestines

It is presumable that in most cases of tuberculosis of the peritoneum and mesenteric and retroperitoneal glands the infection occurs through the intestines. It is also probable that in most instances there is, as when infection occurs through the lungs, a primary focus in the intestines. This focus, however, is almost always small, causes no symptoms and heals without leaving any macroscopic traces.

Tuberculous ulceration of the intestines in early life is not at all common, even in advanced cases of pulmonary and abdominal tuberculosis. When it occurs, it usually causes no symptoms, unless, perhaps, a little diarrhea. The classical symptoms of blood, pus and mucus in the stools are unusual. When these symptoms develop in children known to have tuberculosis elsewhere, they are usually due to tuberculous ulceration of the intestines. When they occur in children without obvious tuberculous lesions, they are almost never due to tuberculosis of the intestines. The treatment consists mainly in regulation of the diet. If the ulcerations are low down, they sometimes can be helped a little by the measures used in the treatment of infectious diarrhea.

Treatment of Tuberculosis

The general principles of the treatment of tuberculosis in early life are, of course, the same as in adult life. In many ways it is easier to carry out the treatment satisfactorily in children than in adults, because the child does not have to support itself or its family, can give up all the time necessary, makes less objection to the details of treatment and, in general, is a better patient.

Prophylaxis.—Since tuberculosis in early life may be caused by both the bovine and human types of the tubercle bacillus, infants and children must be guarded against both. Infection with the bovine bacillus can be prevented by the elimination of tuberculous cows with the aid of the tuberculin test and by the pasteurization or boiling of all milk which does not come from non-tuberculous cows. Pasteurization at 140° F. for twenty minutes or boiling for three minutes destroys tubercle bacilli.

Infection with the human type of organism can be prevented by the separation of infants and young children from adults with open tuberculosis and diminished by instruction in and enforcement of the proper prepautions to be taken to prevent contagion. It is, of course, not always possible, no matter how advisable it may be, because of social or financial conditions, to send a child or a tuberculous member of its family away from home. It is possible, however, to prevent a baby or child from associating with people with open tuberculosis who are not members of the family. It is not necessary to allow it to be kissed promiscuously or to hire people to take care of it concerning whose health nothing is known. This is not the place to take up the details of the precautions to be taken to prevent infection from a member of the family or from outsiders.

They are well known to all physicians and familiar to many of the laity. They are all based on the fact that the tubercle bacilli are in the sputum of patients ill with pulmonary tuberculosis and in the discharges from other open tuberculous lesions. If the sputum and discharges are destroyed, the infective agent is destroyed with them. If no precautions are taken, infection is almost certain to occur. No matter how many are taken, lapses are sure to occur and infection may result. The more that are taken, the less are the dangers of infection.

Rest and Quiet.—Babies and children with tuberculosis should be kept off their feet, preferably in bed, as long as the temperature is above normal. If the temperature rises, they should be put to bed. They must be guarded against overfatigue. No matter what it is they do, whether work or play, if it tires them or puts up the temperature, it must be stopped. It is impossible to overemphasize the importance of rest and the avoidance of overexertion. It is much wiser to err by keeping a child with tuberculosis quieter than is necessary, than by allowing it to overexert itself even a little.

Outdoor Air.—There is no doubt that both childern and adults with tuberculosis do better when they are out of doors than when they are in the house. Why they do better no one knows, although many explanations have been given. It may be that the additional sunshine has more to do with it than the pureness or "freshness"of the air. The ideal to be aimed at is twenty-four hours in the open air in a climate where there are no discomforts or inconveniences incident to spending all of the time out of doors. This ideal is, of course, unobtainable in most instances, but the attempt should be made to approach it as nearly as possible. How nearly it can be approached must depend on the circumstances in the individual case.

Sunlight.—There is also no doubt that sunlight is of benefit to children with tuberculosis. It is probable that the benefit is due largely to the rays at and beyond the violet end of the spectrum. Sunlight, to do good, must fall directly upon the naked body. Glass filters out those rays which do good. The best method of using sunshine in the treatment of tuberculosis is that recommended by Rollier. When it is not possible or not advisable to use this method, much help can be obtained by letting the sun shine on the body whenever and wherever possible. It is somewhat doubtful whether sunlight has any local effect, the chances being that its action is entirely, or almost entirely, general. The *Rollier treatment* is carried out as follows: The body is divided into five zones. The first includes the feet and ankles, the second extends from the ankles to the knees, the third from the knees to the hips, the fourth from the hips to the xyphoid process, and the fifth from the xyphoid process to the chin. The head is not exposed. Two exposures a day are given, one in the early morning, the other in the middle of the afternoon. On the first day both the ventral and dorsal surfaces of the first zone are exposed to the sun's rays for two and one half minutes each time. On the second day the first zone is exposed on both surfaces for five minutes each time and the second zone for two and one half minutes each time. The third day the first zone is exposed on both surfaces for seven and one half minutes each time, the second zone for five minutes on both surfaces each time, and the third zone for two and one half minutes on each surface each time. Each successive zone is exposed in this gradual manner until the patient receives a maximum of one and one half hours in the morning and one and one half hours in the afternoon,

a total of three hours a day. Care must be taken not to burn the patient If the skin is very sensitive, the progress must be slower. It is believed that the effect is better when the skin becomes deeply pigmented than when it does not.

When children have become deeply tanned, are used to exposure and are able to be up and about, it apparently not only does them no harm but actual good to play out in the sun, even in the winter, without any clothes. Such a procedure is, however, hardly feasible, except in institutions. The Rollier treatment can, however, be carried out satisfactorily in the home.

It is probable that the ultraviolet rays from either the mercury vapor or carbon arc lamps have the same action as sunlight. They may be used when it is impossible to employ sunlight.

Tuberculin.—I have had no personal experience with tuberculin in the treatment of tuberculosis in early life. What I have seen of its use in the hands of others leads me to believe, however, that it very seldom does any good and not infrequently does real harm. It is far wiser, it seems to me, to trust to outdoor air, sunlight, food and care than to tuberculin.

Food.—There are no special indications as to the character of the food which should be given to children with tuberculosis. The diet should be regulated according to the age of the child and its individual digestive capacity. It must, of course, in order to do well, get more calories than are necessary to cover its caloric needs. It is very important, however, in the effort to give a large number of calories not to disturb the child's digestion. Fats have no specific action in tuberculosis. They are, moreover, likely to upset the digestion. Furthermore, in tuberculosis of the mesenteric glands and peritoneum the absorption of fat is often interfered with and, in consequence, fat may cause serious disturbance of the digestion. Fat should be looked upon simply as a food element containing a large number of calories. Cod liver oil is no better than any other fat as a food for children with tuberculosis. It has no specific action.

Drugs.—There are no drugs which have any specific or curative action in tuberculosis at any age. They are useful simply for the relief of symptoms. The same drugs should be given in childhood as in adult life, but, of course, in doses suitable for the age.

SYPHILIS

In my experience I have found that in Eastern Massachusetts syphilis is a very uncommon disease among the well-to-do. I have also found it much less common in hospital practice than it apparently is in some other localities. There are several possible explanations for my experience. The first is that I do not recognize syphilis when I see it; another, that syphilis is really less common in early life in Eastern Massachusetts than in some other portions of the United States; and the last, that some of the other observers have called things syphilis which were not. Syphilis in childhood, as at other ages, is an unnecessary and, therefore, a preventable disease. If people would behave themselves they would not have syphilis and would not transmit it to their children. The prophylactic treatment of syphilis is, therefore, more religious and moral than medical. Syphilis in early life may be either hereditary or acquired. In the vast majority of instances it is hereditary. If acquired, it is almost always acquired innocently. The manifestations of acquired

syphilis are the same in early life as later and will not, therefore, be considered.

Syphilis is caused by the treponema pallidum. It is present in the lesions of syphilis at all stages and can almost always be found in discharging lesions of the skin and in scrapings from condylomata and lesions of the mucous membranes. It is best demonstrated by mixing a drop of the suspected material with a drop of fluid india ink and spreading thinly on a glass slide. When the smear is dried and examined with an oil immersion lens the organisms appear as glistening, silvery, corkscrew-like threads against a black background.

Infection of the fetus may occur from a syphilitic mother without syphilis in the father. If the father is syphilitic, the fetus cannot be infected unless the mother is also. Under these conditions, however, many of the mothers are apparently healthy. They all, however, have syphilis in a latent form.

Infection of the fetus may occur at any time during pregnancy, except during the first few weeks before the formation of the placenta. It is possible that it may occur during birth. If so, the syphilis follows the course of acquired, not of hereditary, syphilis.

The manifestations of the disease depend upon the period of pregnancy at which infection takes place and also, of course, upon the severity of the infection. When infection occurs early in pregnancy, the fetus is likely to die in utero and abortion results. When this happens the fetus usually shows no gross evidences of syphilis. Maceration of the fetus is not a sign of syphilis. If death does not occur, the infant may be born alive, very likely prematurely, without showing any objective evidences of syphilis, but malnourished and feeble, and die shortly. It may also show in addition to malnutrition definite syphilitic lesions. If the infection occurs later in the pregnancy the infant may or may not be poorly nourished, but shows no objective signs of the disease. These usually appear within a few weeks, almost always within three or four months and seldom after six months. In other instances in which the infection occurred probably very late or was very mild, the babies are apparently normal at birth and develop normally for a long time, the symptoms sometimes not appearing until middle childhood. In still other instances no symptoms of syphilis develop during childhood, although the Wasserman test shows that infection has taken place.

Syphilis is not the cause of congenital malformations. It is also almost never the cause of feeblemindedness.

Symptomatology and Differential Diagnosis.—When symptoms outside of malnutrition are present at birth, they are snuffles, a vesicular eruption on the hands and feet and enlargement of the spleen. When only one symptom is present, it is likely to be snuffles. If not present at birth, snuffles is likely to be the first symptom to develop. The eruption on the hands and feet seldom appears after the fourth week. Enlargement of the spleen is almost always present at birth, but may not appear until later.

Syphilitic Rhinitis.—The cause of the snuffles is a cellular infiltration of the nasal mucous membrane. This is more marked posteriorly. At first there is but little discharge, the chief symptom being obstruction to breathing. I have seen this so marked that it was necessary to put catheters through the nostrils in order to enable the baby to breathe. As a rule, the snuffles disappears after a few weeks; in other instances, however, a profuse discharge develops, which may persist for

months. It is often purulent and may be bloody. It is very irritating to the skin. The differential diagnosis between syphilitic, diphtheritic and simple chronic rhinitis has been discussed under the head of chronic nasopharyngitis. On general principles, unless there are other definite evidences of syphilis, a chronic nasal discharge in a baby is not syphilitic. Syphilitic rhinitis does not lead to the marked deformities of the nose sometimes seen in later childhood. These are due to syphilitic disease of the bones and cartilages. In some instances the larynx is also involved. The chief symptom is hoarseness of the cry.

The vesicular eruption on the hands and feet is usually spoken of as *syphilitic pemphigus.* It consists of rounded vesicles varying in size from that of a split pea to that of half a cherry on an inflammatory base. The vesicles often change to pustules. These lesions are always present on the palms and soles. They are usually limited to the palms and soles, but sometimes appear on other portions of the body. They always denote a very severe type of infection. They must be distinguished from the lesions of pemphigus neonatorum. In this disease there are almost never any lesions on the palms and soles. They are never present at birth. They are associated with other signs of acute infection and there are no other evidences of syphilis. These are almost always present with syphilitic pemphigus. The nutrition is much impaired with syphilitic pemphigus, while it is normal at first in pemphigus neonatorum.

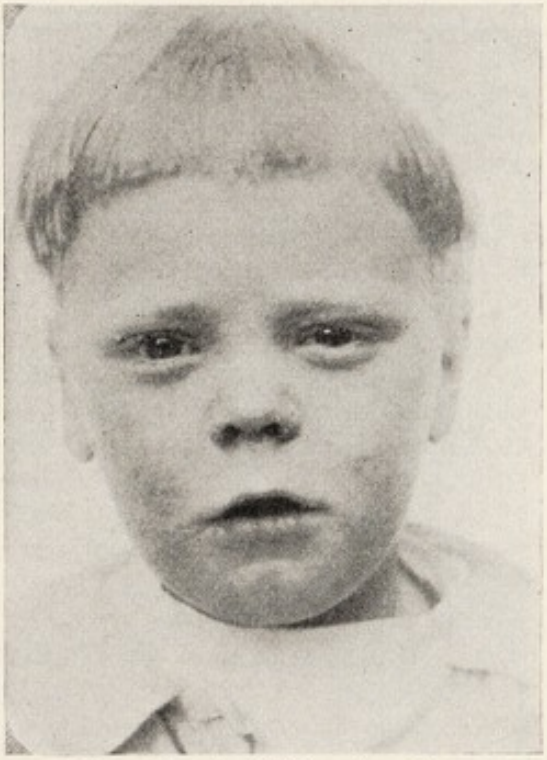

FIG. 84.—Syphilitic rhagades.

The *enlargement of the spleen,* when present at birth, is usually not marked, although later it is often very marked. Enlargement of the spleen at birth is almost pathognomonic of hereditary syphilis. It is usually associated with enlargement of the liver. In some cases both may be very marked.

Another lesion of the skin which usually appears early and is due to cellular infiltration is *thickening and discoloration of the palms and soles.* They are a little swollen, dark red or reddish purple, and shiny. When the infiltration is greater, the thickening is also greater and there is, in consequence, *desquamation of the palms and soles.* The skin usually comes off in large flakes. When this stage is reached the discoloration is usually much less marked. The whole skin may be involved and may everywhere be cracked and desquamating. If the face is much involved, it becomes expressionless and mask-like. The face is also likely to be of a peculiar yellowish, waxy color. When this thickening and discoloration of the skin is associated with emaciation, as it usually is, the picture is very characteristic and almost pathognomonic. If the skin is thickened about the mouth, cracks and *fissures* are likely to be formed. When these have healed, radiating lines or depressions are likely to remain. These are known as *rhagades.* While fissures and rhagades are very

strongly suggestive of syphilis, it must be remembered that they may be due to other causes and that their presence does not necessarily prove that a child has syphilis. When the skin of the scalp and eyelids is involved, the disturbance of nutrition may be so great as to cause *alopecia*. The nutrition of the nails may also be disturbed. This may result in suppuration and exfoliation of the nail. In other instances the dorsum is arched and the nail looks as if it had been pinched by a pair of forceps.

Another characteristic lesion of the skin which may appear at any time is the *maculo-papular syphilide*. This eruption appears in spots varying in size from that of a split shot to a dime. These spots are rose red in the beginning, but later change to a yellowish brown or copper color. They are usually not elevated, but may be slightly. They are most marked on the hands, feet and extensor surfaces of the arms and legs. They may, however, appear on any portion of the body. They usually come out slowly. Traces of them may persist for weeks or even months. There is no inflammation about them and they do not itch. After a time, fine scales develop on the top of these spots. In some instances this eruption is papular. If so, it is most likely to be on the palms and soles. It is said that, if the maculo-papular syphilide is the first symptom to appear, the prognosis is more favorable than usual.

Wherever the skin is moist the lesions may slough and *condylomata* develop. These are reddened, moist, slightly elevated areas, varying in size from one eighth to one inch in diameter. They are most common about the anus and genitals. They must not be confused with the erythema which results from the irritation caused by wet diapers, and which, in severe cases, is associated with denudation and the formation of superficial ulcers. When the lesions are due to wet diapers they are strictly confined to the area covered by the diapers, there are no other evidences of syphilis and they yield promptly to proper treatment.

Lesions of the mucous membranes are rare in early infancy, but common later. They consist of *ulcerations* and *mucous patches*. These are found upon the mucous membrane of the lips, cheeks, tonsils and soft palate and on the sides and under surface of the tongue. There is some, but not much, infiltration of the edges. The mucous patches are slightly elevated. It must never be forgotten that ulcerations of the mucous membrane of the mouth may be due to many other causes than syphilis. Among the most common causes are mechanical irritation in cleaning the mouth and irritation from foreign bodies or too hot food. Ulcerations are especially likely to occur over the tips of the hamular processes of the sphenoid bone, which are very near the surface. These ulcerations are known as *Bednar's Aphthae* and are due simply to mechanical injury. The mucous patches of syphilis are more sharply localized than those of thrush, are not as white and cannot be removed mechanically. They are whiter and less sharply defined than the circular ulcers of herpetic stomatitis. They are not as deep as the lesions of ulcerative stomatitis.

Pathologically there is almost constantly a lesion of the bones, which is usually spoken of as the *epiphyseal osteochondritis* of hereditary syphilis. These changes are present in the fetus, as well as later. Macroscopically there is a broad yellow line at the junction of the epiphysis and diaphysis. There may be simply an inhibition of bone formation or, in addition, a development of granulation tissue, which may result in the separation of the epiphysis. These lesions are usually most marked at the lower end of the femur and radius and the upper end of the humerus and tibia.

The Roentgen ray shows broadening, irregularity and indentation of the epiphyseal line, a lighter zone between this and the diaphysis and an irregular shadow at the end of the diaphysis, representing the irregular bone formation in this area. Clinical evidences of these lesions in the bones are, in my experience, quite uncommon. When there is swelling, it is in the region of the epiphyseal line or of the lower end of the diaphysis. It is rounded, smooth, often tender, seldom red or hot. When there is a separation of the epiphysis, there is, in addition, *pseudoparalysis*. This may also occur when there is no separation of the epiphysis, apparently as the result of the extension of the process to the muscles.

The diagnosis is not difficult, if the possibility of syphilis is kept in mind. Confusion with rickets is only likely to occur when the swellings are at the wrists or ankles. The swelling in syphilis is at the epiphyseal line and above it, while in rickets it is at the epiphyseal line and below it. The swelling in rickets usually develops later than that in syphilis. There are almost always other signs of rickets, if the swelling is due to rickets, and other signs of syphilis, if it is due to syphilis. In scurvy the swelling is over the diaphysis, the epiphysis is never involved and there is never separation of the epiphysis, unless the hemorrhage has been very large and the periosteum broken through. In such cases there are always other marked signs of scurvy, which are, of course, absent in syphilis. Scurvy usually develops later than syphilis. Rheumatism does not occur in infancy. If it did, the lesions would be about the joints, not at the epiphyseal line and above it. When there is pseudoparalysis involving the arm, the condition may be mistaken for obstetrical paralysis. In obstetrical paralysis the paralysis is present at birth; in syphilitic pseudoparalysis it develops later. There is no swelling, tenderness or pain on passive motion in obstetrical paralysis. All of these symptoms are present in syphilis. Furthermore, the inability to use the arm in obstetrical paralysis is due to lack of power, while in syphilis it is due to the pain which motion causes. It hardly seems possible that syphilitic pseudoparalysis could be mistaken for infantile paralysis, but I have known it to be. In infantile paralysis the onset is sudden, there is no swelling and there is a true paralysis. There are, of course, no other signs of syphilis. A positive diagnosis can always be quickly and easily made between all of these conditions with the help of the Roentgen ray.

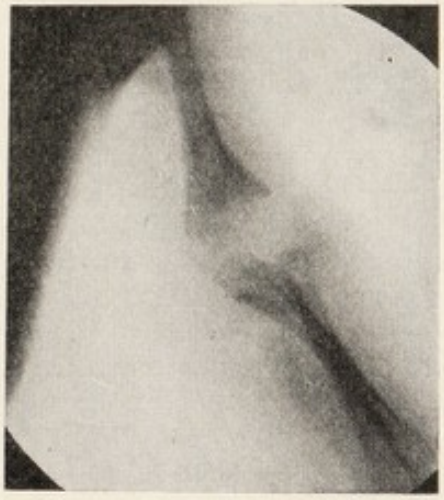

FIG. 85.—Syphilitic epiphysitis.

The shaft of the bone may also be involved. The pathological process is an *osteoperiosteitis*. It is likely to be general, all the long bones being involved. It seldom causes any symptoms, but is shown in the X-ray by a thickening of the shaft. A special manifestation of this condition is syphilitic *dactylitis*. This most often develops during the middle half of the first year. The middle and proximal are more often involved than the distal phalanx. The enlargement develops slowly and causes no pain or interference with use. The joints and soft parts are not involved. There is no redness, heat or tenderness. In tuberculous dactylitis the soft parts are, sooner or later, involved and suppuration often occurs. This never happens in syphilitic dactylitis.

The teeth are usually poorly formed, decay early and show the general manifestations of disturbance of the nutrition during fetal and early infantile life. There is nothing whatever characteristic about the teeth of the first dentition. The characteristic changes are seen only in the second teeth.

Plastic iritis may occur in infancy or even before birth. It is, however, very rare. The other syphilitic changes in the eyes, which are far more common, develop later.

General enlargement of the peripheral lymph nodes is not uncommon. It is probably almost never syphilitic in character, but is simply a manifestation of the general disturbance of nutrition. General enlargement of the peripheral lymph nodes is likely to occur in all disturbances of nutrition in infancy. It, therefore, is of no importance in differential diagnosis. Even enlargement of the epitrochlear glands is not pathognomonic of syphilis at this age.

The only definite manifestation of syphilis of the nervous system which I have seen in infancy is *syphilitic meningitis*. It resembles closely in its symptomatology and course tuberculous meningitis. The diagnosis is very difficult unless there are other definite evidences of syphilis. The findings in the cerebrospinal fluid are the same in both conditions, except those with the colloidal gold test. A positive Wasserman test is, of course present in syphilitic meningitis and absent in tuberculous meningitis. Theoretically, *hydrocephalus* might be expected to develop as the result of syphilitic adhesions at the base of the brain; practically, I think this almost never occurs.

Hemorrhages are rare as the result of congenital syphilis. They are usually from the mucous membranes, especially that of the nose. In rare instances the skin lesions may be hemorrhagic.

Malnutrition and failure to gain in weight without apparent cause are very common manifestations of syphilis. When they are not associated with other more obvious symptoms, they are likely to be attributed to some other cause. There is nothing characteristic about them. Syphilis should be thought of whenever babies do not thrive and there is no apparent reason for it. In the vast majority of cases, however, it will be found that syphilis is not the cause. A positive diagnosis can only be made, in the absence of obvious signs of syphilis, with the aid of the Wasserman test.

Anemia, secondary in type, is a constant manifestation of hereditary syphilis. Other quite characteristic symptoms are *restlessness* and *sleeplessness*, often associated with crying out during sleep. These symptoms, when there is no evident cause, should always suggest the possibility of syphilis. They usually, however, are due to some other cause. A positive diagnosis can only be made with the aid of the Wasserman test.

Enlargement of the veins of the scalp is a somewhat unusual manifestation of hereditary syphilis, but when seen should always suggest it as a possibility. It is usually due, however, to a thin skin or rickets.

If hereditary syphilis, which showed early symptoms, has been insufficiently treated, symptoms of a different sort are likely to develop during childhood. These symptoms of **late hereditary syphilis** may be the first to appear, no evidences of the disease having shown themselves during infancy. They seldom appear after puberty. The most characteristic symptoms are in the eyes and bones.

The most common manifestation in the eyes in childhood is interstitial keratitis. At this age it is almost always syphilitic in origin. The first

sign is a cloudiness of the cornea with ciliary congestion. The cloudiness gradually increases until the whole cornea is markedly hazy. There is usually no pain and no congestion of the conjunctiva. Both eyes are always involved, although one eye is usually affected earlier than the other. The chances for complete recovery are fairly good with proper treatment. Other less common manifestations are retinitis, choroiditis and optic neuritis. Their syphilitic nature is easily recognized by an opthalmologist. *Deafness* is an occasional symptom of late hereditary syphilis. It is usually due to labyrinthine disease and is incurable. It most often develops slowly and progresses steadily, but sometimes comes on acutely.

The usual lesion in the bones is a *chronic osteoperiosteitis*. It is most common in the tibiæ, but may involve any or all of the long bones. It is

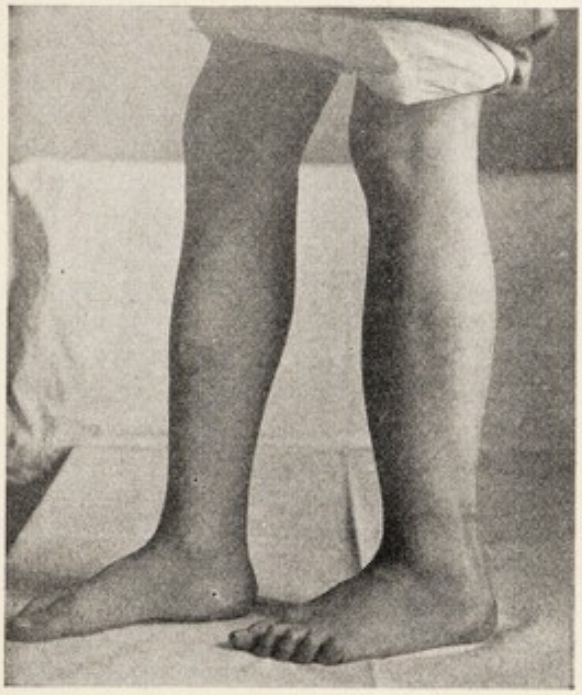

FIG. 86.—Chronic syphilitic osteoperiosteitis. Sabre tibiæ.

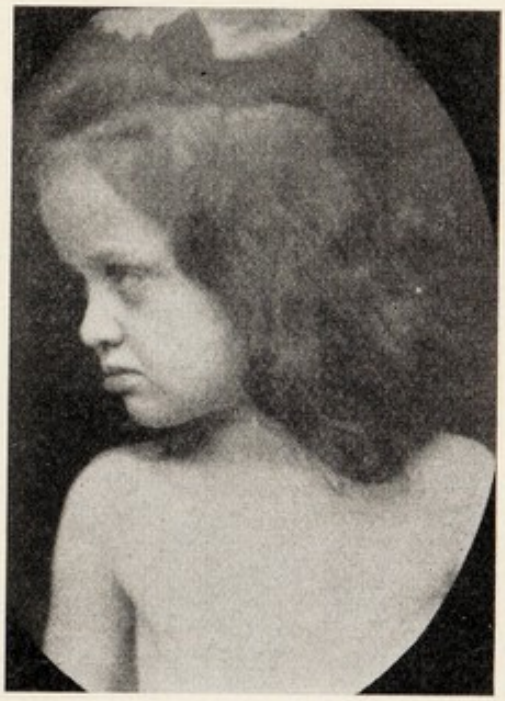

FIG. 87.—Syphilitic head. "Olympian brow."

almost always symmetrical and is usually confined to the shafts. The bones of the cranium are also often involved.

The process in the long bones results in thickening of the cortex, which may be either general or local. The enlargement may be, therefore, either uniform or irregular. The newformed tissue almost never breaks down. The deformity is usually the first thing noticed. It may, however, be preceded or accompanied by pain and tenderness. The pain is usually worse at night. Tenderness is not marked and there is never heat or redness. When the lesions are in the *tibiæ*, the deformity is characteristic and is known as the *sabre deformity*. The anterior border curves forward. Sometimes the bones are bent backward and inward in the lower portions. When this occurs the position suggests that in old deformities from rickets. In rickets there are always other evidences of rickets, which are not present in syphilis. In syphilis there are almost always other evidences of syphilis. The deformity is always regular in rickets and may be irregular in syphilis. The diagnosis between the two

conditions is easily made with the Roentgen ray. The thickening of the cortex is on the concave side of the curvature in rickets and on the convex side in syphilis. In rare instances the enlargement may occur at the ends of the bones. This enlargement may cause limitation of motion at the joints, although the joints themselves are never involved.

When the cranium is attacked, the frontal bones are involved more often than the parietal. The enlargement is not unlike that in rickets, but is usually more marked. It sometimes is largely in the median line. The resulting deformity is sometimes spoken of as the *Olympian brow*. In rickets the swelling occurs in infancy and comes on quite quickly, while in syphilis it occurs in childhood and develops slowly. There are also almost always other evidences of rickets, when it is due to rickets. It must also be remembered that many children have peculiarly shaped heads which are normal variations, not the result of syphilis. The enlargement of the head in hydrocephalus is uniform, not irregular, and there is likely to be bulging of the eyes from pressure on the supraorbital plates. Sarcoma of the skull is almost always unilateral and the swelling develops rapidly; while in syphilis the swellings are bilateral and develop slowly. Irregular, rounded nodes, usually multiple, sometimes develop on both the frontal and parietal bones. Here again it must be remembered that exostoses of the cranial bones may develop from other causes than syphilis. *Gummata* may also develop in the bones. They are more common in the cranial than in the long bones. They may break down and discharge. If so, sinuses persist until the sequestra are thrown off. When the cranial bones are involved, the process may extend inward and cause meningitis and syphilitic disease of the brain.

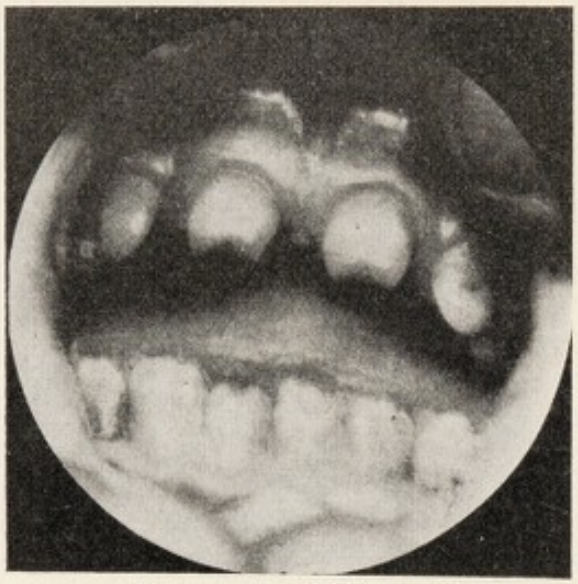

FIG. 88.—Syphilitic teeth.

There may also be gummatous disease of the bones of the nose. The mucous membrane is likely to be involved at the same time. The results are the same as in tertiary syphilis in the adult. These changes are, however, very uncommon in childhood.

Changes in the shape of the second teeth are not uncommon. Their absence does not exclude syphilis. They are most common in the upper incisors. The most typical change is the hollowing out of the edge of the teeth, making it concave. Teeth deformed in this way are known as "*Hutchinson's teeth*." They are not always due to syphilis. Other syphilitic deformities of the incisor teeth are the "screw-driver" and "peg" teeth, which justify their names by their shape. These teeth are also not pathognomonic. The enamel of the second teeth is likely to be deficient and their surfaces irregular. They decay easily.

Arthritis is an occasional symptom. It is most often in the knees and bilateral. It is not accompanied by pain, heat, redness, tenderness or fever. It sometimes, however, causes discomfort and limitation of motion. The swelling may be intermittent. The joints are symmetrically enlarged.

General enlargement of the peripheral lymph nodes is of more importance than in infancy, because enlargement of the peripheral lymph nodes is not as common in disturbance of nutrition in childhood as it is earlier. Enlargement of the epitrochlear glands is very suggestive. Both enlargement of the epitrochlear and of the peripheral nodes in general, however, may be due to other causes.

Subcutaneous gummata are not very uncommon. They occur most often on the face, thigh and upper legs. The appearances are the same as in the adult. When they break down they form ulcers which may be confused with tuberculous ulcers. The syphilitic ulcer is deeper than the tuberculous. The edges are rounded, thickened and indurated. Those of the tubercular ulcer are soft and flat. The syphilitic ulcer

FIG. 89.—Syphilitic hydrops of knees.

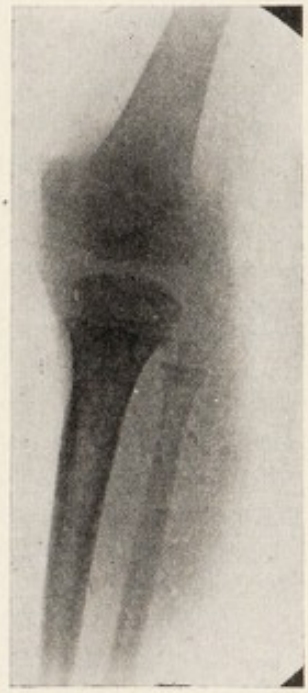

FIG. 90.—Syphilitic hydrops of knee.

is more regular in outline than the tuberculous. The scar of the syphilitic ulcer is smooth and white; that of the tuberculous red or purple, which slowly fades out.

Laryngitis is a rare manifestation. It may cause obstruction and necessitate operation. In rare instances it may go on to ulceration.

Enlargement of the liver and spleen is very constant and of considerable importance in diagnosis. Sometimes one, sometimes the other, is the larger. Syphilis has been discussed among the diseases of these organs.

Disturbance of the nutrition is likely to be a prominent symptom in the late, as well as in the early cases, of hereditary syphilis. It is shown chiefly by failure to gain properly in height and weight. When this failure is marked, the term "*infantilism*" is often applied to it. Retardation of the mental development may or may not accompany that of the physical.

Hereditary syphilis has not been, in my experience, as common a cause of disease of the nervous system as it apparently has been in that of others. It may, of course, as in adult life, be the cause of isolated paralyses of the cranial nerves, of systemic disease of the cord or of chronic cerebral disease. It is very seldom, however, in my experience, that these results are manifest before adult life.

Diagnosis.—The differential diagnosis between hereditary syphilis and other conditions with which it may be confused because of single symptoms has already been taken up under symptomatology. A history of repeated miscarriages is always suggestive of hereditary syphilis, especially if the duration of each pregnancy has been longer than that of the preceding one, and justifies further investigation. It must not be forgotten, however, that there are many other causes for repeated miscarriages. In this connection it is well to remember that, while no one is above suspicion, most people are decent, and that only a small minority have syphilis.

When there is any reason to suspect syphilis, a *Wassermann test* should be done, not only on the patient but also on its parents. It must not be forgotten that a positive reaction in an infant at birth is not certain evidence of syphilitic infection at birth, because the reacting substances may pass from mother to fetus in utero without the passage of the spirochete. The reaction may be negative for weeks after birth and then become positive, because of inability to form fixing antibodies. A negative reaction during the first few weeks or months of life, if the baby shows evidences of syphilis and the mother gives a positive reaction, does not count, therefore, against syphilis. It does not prove, moreover, that, if the baby shows no signs of syphilis, it has not been infected and will not show them later. If negative at this time, if the parents are syphilitic, it should be repeated after a few months. A positive reaction is present in at least 95% of the cases of early hereditary syphilis and in from 60% to 95% of the late cases. It is true that a positive reaction is sometimes obtained in other conditions, the most important of which in early life are tuberculosis, scarlet fever and prolonged ether and chloroform narcosis. It is easy to avoid making the test during scarlet fever or after prolonged narcosis. In early life tuberculosis apparently less often gives a positive reaction than in adults. A positive Wassermann reaction in an infant or child is, therefore, for practical purposes, conclusive proof that it has syphilis, while a negative reaction, provided it has not had treatment, is very strong evidence against it, especially in infancy. While this is true, it must never be forgotten that the technic of the Wassermann test is complicated and that mistakes are made even in the best regulated laboratories. I have, for example, known two reputable laboratories to give opposite reports on the same specimen of blood, part of which was sent to one laboratory and part to the other. I have also known a reputable laboratory to give opposite reports on the same specimen of blood sent in two parts under two different names. Mistakes of this sort being liable to occur, the results of the Wassermann test ought not to be given any more weight in differential diagnosis than the clinical findings.

At least 5 c.cm. of blood are required for the Wassermann test. This may be obtained in an infant from any of the superficial veins that can be entered or from the longitudinal sinus. It is usually less difficult to get into the veins of the scalp and the external jugular than the veins at the elbow. In a child it is usually possible to get the blood from the veins at the elbow. It may also be obtained by incising the lobe of the ear.

The longitudinal sinus is reached by introducing a hypodermic needle, attached to a syringe, at the posterior angle of the anterior fontanelle in the median line, after shaving and sterilizing this area. The needle is passed forward and a little downward through the skin and dura. There is a sudden diminution in the sense of resistance when it enters

the sinus. Care must be taken not to introduce it too deeply. The piston of the syringe is then drawn and the syringe fills with blood. It is unnecessary to use any of the special needles devised for this purpose, although there is, perhaps, less danger of going too deeply, if one of them is used.

I have had no experience with the luetin and other tests for syphilis. The concensus of opinion, however, seems to be that they are inferior to the Wassermann test.

Prognosis.—It is useless to give figures as to the mortality or the chances of complete recovery in hereditary syphilis. They give no information as to the chances in the individual case. The prognosis in the individual case depends on the development and vitality of the infant, the severity of the infection and the character of the treatment. The prognosis is always grave in premature infants and in those that are poorly nourished at birth. It is very grave when syphilitic manifestations are present at birth. The longer their appearance is delayed, the better is the outlook. The prognosis is grave when the liver and spleen are much enlarged at birth. Syphilitic pemphigus is an evidence of severe infection. When the maculopapular syphilide is the first symptom, the infection is not a very severe one. Late syphilis is far more resistant to treatment than early. The earlier the treatment is begun, the better is the outlook. Complete recovery is impossible, unless the treatment is very thorough.

Treatment.—*Prophylaxis.*—As already stated, syphilis is an unnecessary disease. It would disappear, if people were continent. The first element in prophylaxis is the religious and moral training of the public. The frequency of the occurrence of hereditary syphilis would be diminished in the children born legitimately by allowing only those who had negative Wassermann reactions to marry. Its frequency would also be diminished, if a Wassermann test was done on all pregnant women and treatment instituted, if it was positive. If treatment is begun not later than the fifth month of pregnancy, the majority of children escape infection. If it is begun after the seventh month, infection is not prevented, but the manifestations of the disease are milder.

Feeding and Care.—Babies with hereditary syphilis should be breast-fed, if possible. Breast-fed babies often thrive in spite of obvious lesions, while artificially-fed infants do not do as well, even when getting anti-syphilitic treatment. A syphilitic infant cannot infect its mother. There is, therefore, no reason why she should not nurse it. It is not right, however, for a syphilitic baby to nurse a non-syphilitic woman, even if it shows no lesions, because it can infect her. The milk can, however, be drawn and fed to the baby in a bottle. If breast milk is not available, more than the usual care should be taken in the preparation of the artificial food, as the prognosis is influenced very largely by the state of the nutrition.

It is also most important for syphilitic infants to have a large amount of outdoor air and sunlight. They are especially subject to disturbances of nutrition, such as rickets, and need all the protection and help possible. Unless they can get an ample amount of sunlight, it is advisable to give them cod liver oil or yolk of egg to prevent the development of rickets.

Medicinal.—There is at present much difference of opinion as to the best method of treating syphilis in infancy and childhood. All agree that it is irrational to attempt to treat syphilis in an infant by the administration of drugs to the mother. All agree, also, that the iodide of potash

is useful in those late manifestations which correspond to the tertiary manifestations of acquired syphilis. Some believe that the arsenicals are far superior to mercury and that it is not necessary to combine mercury with them. Others believe that mercury is superior to the arsenicals, although they admit that in very severe congenital cases the arsenicals must be used first, because the action of mercury is too slow to prevent death. Others believe that both the arsenicals and mercury should be used.

I have had very little personal experience in the treatment of hereditary syphilis since the introduction of the arsenicals. I have, in the past, seen the symptoms disappear in many cases under treatment with mercury and have followed these children for many years without the appearance of any new manifestations. Nevertheless, recent experience shows that the Wassermann test is often positive in these children. It is reasonable to believe, therefore, that, although they are free from symptoms, they may not be cured. Having had so little personal experience in the use of the arsenicals, I do not feel competent to give advice as to the best method of using them. The method in use in the clinic for the treatment of hereditary syphilis at the Children's Hospital in Boston, in charge of Dr. Philip H. Sylvester, is as follows:

Neoarsphenamine is used intravenously and sulpharsphenamine intramuscularly. Neoarsphenamine is given intravenously once a week for twelve doses. For small and feeble babies the initial dose is one twentieth of a gram. For others it is one tenth of a gram. This is dissolved in 1 c.cm. of sterile water. If the dose is increased, the relation of 1 c.cm. of water to one tenth gram of the drug must be maintained. The drug must be given within twenty-five minutes after the solution has been prepared. The dose is increased each time one quarter of one tenth of a gram until a dose of two tenths of a gram is reached. After this the dose is kept at two tenths of a gram.

After a month a Wassermann test is done. If the test is negative, another series of twelve doses, one half the strength of the initial series, is given intravenously. If the test is positive, the series is repeated at a slightly higher dosage, but the dosage should never exceed two and one half tenths of a gram in babies under six months of age.

The Wassermann test is repeated every six months. If it is positive, another course of treatment is given. No mercury is used. The experience in this clinic has shown that, if treatment is begun before the baby is two months old, a permanently negative Wassermann can be obtained. If it is begun later, a permanently negative Wassermann is very unusual.

In late hereditary syphilis the method of treatment is the same, but larger doses are given, up to four and one half tenths of a gram. Experience has shown that it is extremely difficult to get a permanently negative Wassermann test in late hereditary syphilis. Experience has also shown that in many instances there is an excess of cells in the spinal fluid, when there are no symptoms of any sort. How many of these children later develop demonstrable lesions of the nervous system it is impossible to say.

Great care must be taken in the use of neoarsphenamine intravenously not to inject the drug outside of the vein. If this is done, there is always marked induration, often sloughing. It is safer and wiser to make the injection into one of the veins of the scalp or at the elbow than into the longitudinal sinus. This, in my opinion, should only be used when it is impossible to get into one of the superficial veins.

The dose of sulpharsphenamine usually recommended is about twice that of neoarsphenamine, or twenty milligrams per kilo. of body weight. In the clinic at the Children's Hospital, however, forty milligrams per kilo. of body weight is the usual dose. This is dissolved in the same way as neoarsphenamine, that is, in the proportion of one tenth of a gram to 1 c.cm. of water. It is injected deeply intramuscularly, usually into the buttocks. The injection is practically painless. Twelve doses are given at weekly intervals in the same way as with neoarsphenamine. The indications for repetition are the same. It apparently is just as effective intramuscularly as neoarsphenamine is intravenously. It also apparently has more effect on the spinal fluid than neoarsphenamine. It is much easier to use sulpharsphenamine intramuscularly than neoarsphenamine intravenously. Sulpharsphenamine intramuscularly seems, therefore, more practicable for the general practitioner than neoarsphenamine intravenously.

There is much difference of opinion as to the form of mercury to use. The investigations of Ramsey & Groebner (American Journal of Diseases of Children, 1920, Vol. 20, Page 199) as to the rapidity of absorption and elimination of the different mercurial preparations is enlightening. They found that 50% mercurial ointment is to be preferred to the less concentrated forms, and that the inunctions need not be given more often than twice weekly, instead of daily. They also found that the quantity of mercury absorbed is much increased by friction. They found that, when mercuric chloride is given hypodermically, it continues to be eliminated for six or seven days. They also found that its use is frequently followed by the appearance of protein in the urine. They believe, therefore, that it should not be used in the treatment of syphilis in children. Other authorities think that the appearance of protein in the urine is unimportant and still recommend its use. Calomel by the mouth is absorbed in small amounts and continues to be eliminated for a considerable time, so that it is probable that it is sufficient to give it at intervals of several days, thus avoiding diarrhea. Gray power is absorbed to a small degree and eliminated rather rapidly, so that large doses, repeated daily, are probably necessary to maintain mercury in the circulation. Granting that these observations are correct, the best way to administer mercury in hereditary syphilis is by rubbing 50% mercurial ointment into the skin twice weekly. The usual amount is from ten to twenty grains. It should, of course, be rubbed into a different spot each time. Calomel appears to be, from their experiments, a rather better drug than gray powder. Calomel is usually given in doses of from one twentieth to one tenth of a grain three or four times a day and gray powder in doses of one half grain three or four times a day. Judging from Ramsey's work, equally good results should be obtained by giving calomel in grain doses twice a week.

Iodide of potash should be used in the same way as it is in tertiary syphilis in adults. It should, of course, be given well diluted either with milk or water. It is less likely to disturb the digestion if it is given in essence of pepsin.

INFLUENZA

Influenza, commonly known as the grippe, occurs in widely spread epidemics at irregular intervals, usually of many years. It is probably endemic for some years after an epidemic, and it is not unlikely that sporadic cases occur from time to time. It is a specific disease and must

not be confused with ordinary "colds," whether epidemic or sporadic, which are so common, especially in the winter. Clinically, it is protean in its aspects, but has a special tendency to attack the respiratory mucous membrane. Prostration out of proportion to the clinical manifestations is also characteristic. It occurs at all ages, although in some epidemics infants and young children are less often attacked than adults. It is as severe in early as in adult life.

Etiology.—The evidence at present seems to show that influenza is caused by a minute, filter-passing, anaerobic bacillus, known as the bacterium pneumosintes. "This bacillus, when inoculated into rabbits, induces slight lesions of the lungs, predisposing to ordinary bacterial pneumonic infections, and at the same time affects the number of circulating mononuclear leukocytes, in a manner corresponding with the leukopenia of epidemic influenza in man. It is agglutinated also by the blood serum of patients that have recovered from influenza." (Flexner.) Pfeiffer's bacillus, or the bacillus influenza, is almost certainly not the cause of influenza, as was so long believed, but is simply a secondary invader, like staphylococci, streptococci and pneumococci.

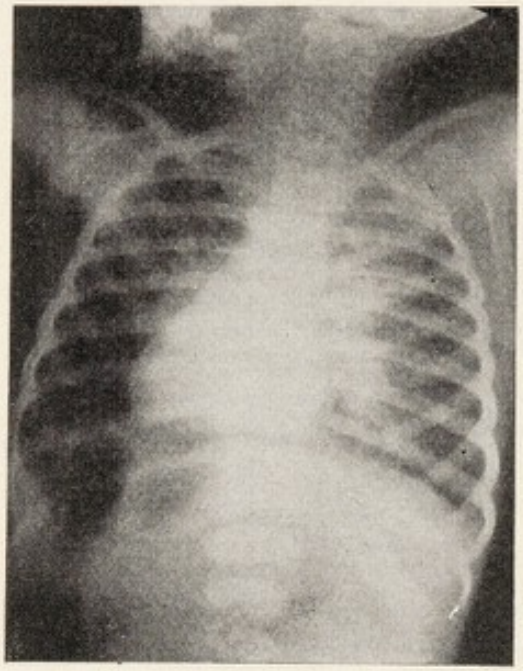

FIG. 91.—Influenza. Rales the only physical sign.

The organ is mistransmitted directly by patients or carriers by droplet infection. It is possible that it may be transmitted indirectly by contaminated articles, if only a very short time elapses between contamination and exposure.

The period of incubation is apparently between one and four days, but probably may occasionally be much longer.

Symptomatology.—The symptomatology of influenza is essentially the same in early as in adult life. There is the same sudden onset of fever and catarrhal symptoms in the upper respiratory tract and the same tendency to depression out of proportion to the local symptoms. Babies and young children are apparently less troubled by general pains and aches than older children, who complain in the same way as adults. In the majority of instances, as in adults, the temperature remains elevated, often considerably, for some days and then drops to normal by a rather rapid lysis. The catarrhal symptoms increase for a few days, after which they gradually diminish and disappear. The prostration is out of proportion to the severity of the other symptoms and convalescence is relatively slow.

Babies are quite likely to show rather marked evidences of involvement of the nervous system. They are often markedly drowsy and apathetic. In other instances they are excited, restless and twitchy. In some cases the reflexes are increased and there is more or less rigidity. Convulsions are, however, unusual.

In many instances, especially in infants and young children, the catarrhal process extends to the bronchi. There is nothing characteristic

about the physical signs of influenza bronchitis. The sputum is, however, often more purulent than in ordinary bronchitis. Further extension, resulting in bronchopneumonia, is also not uncommon. The

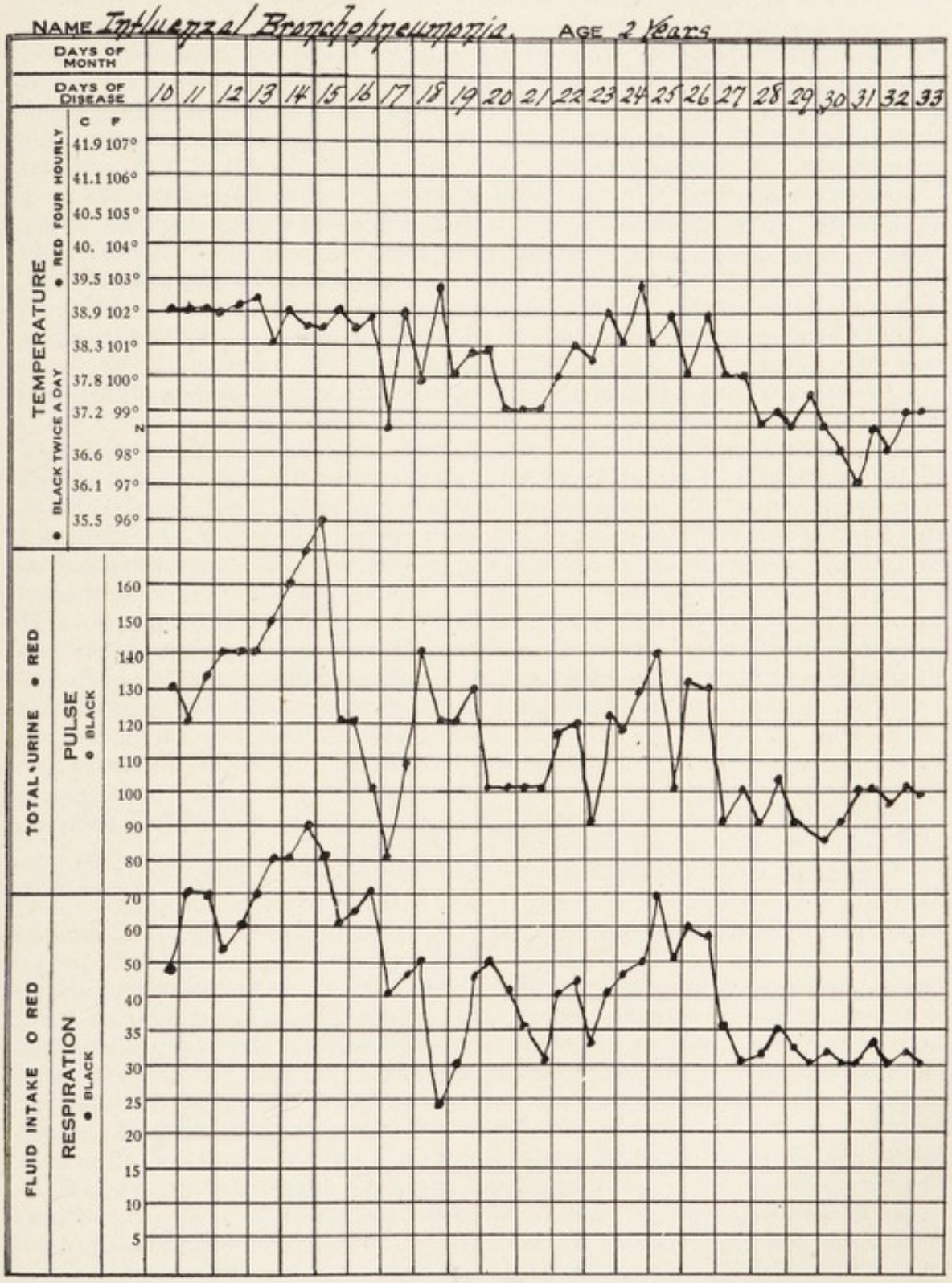

Fig. 92.—Influenzal bronchopneumonia.

bronchopneumonic process is, however, not caused by the b. pneumosintes, but by secondary invaders, especially Pfeiffer's bacillus, streptococci and pneumococci. The symptoms, physical signs, temperature

and course do not differ from those in cases of bronchopneumonia due to other causes, except, perhaps, that the number of areas involved at one time is greater and the period during which new areas continue to be involved longer than is usual in other forms.

Long continuance of the fever, without continued evidences of involvement of the respiratory tract or its adnexa, is, in my experience, very unusual in early life. The gastrointestinal type of influenza is also comparatively uncommon in early life, although there are, of course, as in all the acute febrile diseases of childhood, usually more or less symptoms referable to the digestive tract.

Blood.—In uncomplicated influenza there is a leukopenia. There is apparently nothing constant about the differential count of the whitt cells in early life, although there is considerable evidence to show thae there is an absolute diminution in the number of mononuclear cells. When there is a complicating bronchopneumonia, there is a tendency to a slight leucocytosis. It is said that the prognosis is better when there is a leucocytosis.

Urine.—The urine is either normal or shows the changes of acute degenerative nephritis.

Complications.—Otitis media is a very common complication, especially in infancy. It may develop without the appearance of any symptoms pointing to the ears and is the most common cause of an increase in or persistence of the fever. The ears should, therefore, be examined daily. Involvement of the accessory sinuses is more common than in other infections of the upper respiratory tract. Bronchitis and bronchopneumonia have already been referred to. Lobar pneumonia and infections of the pleura are uncommon. Myocardial degeneration is not uncommon, but endocarditis and pericarditis seldom develop. Acute nephritis is a rare complication. I have never seen involvement of the peritoneum or joints. Meningitis sometimes develops late in the course of the disease. It is usually caused by Pfeiffer's bacillus, but may be due to any of the pus organisms. Encephalitis is not due to the same organism which causes influenza. It is, indeed, very doubtful if there is any connection between influenza and encephalitis.

Prognosis.—The prognosis in uncomplicated influenza is good. Death seldom results at any age. When the disease is complicated by bronchitis or bronchopneumonia, the outlook is more serious and depends largely upon the severity of these complications. In general, the prognosis is not quite as good as when bronchitis and bronchopneumonia are due to other causes. Secondary inflammations of the ears and accessory sinuses are also likely to be more serious and more obstinate than usual. Those cases in which the fever persists for a long time usually eventually recover. In my experience, influenza is just as serious a disease in infancy and childhood as in later life. Furthermore, bronchitis and bronchopneumonia, whether or not the results of influenza, are far more dangerous in infancy and early childhood than in late childhood and adult life. Influenza and its complications may, like other infections of the respiratory tract, light up a quiescent tuberculous process in the lungs or tracheobronchial lymph nodes. It may also, like other diseases, by weakening the resistance, increase the susceptibility to infection with the tubercle bacillus.

Diagnosis.—The diagnosis of influenza during an epidemic is easy. At other times, in view of the present lack of any rapid and simple method of bacterial diagnosis, it is very difficult. It is important to bear in mind

that catarrhal inflammations of the upper respiratory tract, no matter how acute, how severe or how contagious, are, except during an epidemic of influenza, almost never influenza. It pleases people under such conditions to be told that they have the grippe, but the diagnosis is almost always wrong. The points most strongly in favor of influenza are a very acute onset and constitutional symptoms and prostration out of proportion to the severity of the local symptoms. The most important single point in diagnosis is the white blood count, there being a leukopenia in influenza and a polynuclear leukocytosis in those catarrhal inflammations with which it is likely to be confused.

Treatment.—There is no specific treatment for influenza. Preventive inoculations with the bacillus pneumosintes have been unsuccessful. There is no excuse for using vaccines made from Pfeiffer's bacillus and other organisms, when the disease is not caused by them. Furthermore, the results obtained from their use in the hands of competent and unprejudiced observers have been negative. There is no drug which has any effect on the course of the disease. Treatment must, therefore, be symptomatic and for the prevention of complications. It is the same as for other catarrhal conditions of the upper respiratory tract and is described in the treatment of these diseases. It should go without saying that every child with influenza should be put to bed and kept there until it is well, and then several days longer. The treatment of complicating otitis media, bronchitis and bronchopneumonia is the same as when they are due to other causes and is described in the chapters on these diseases.

Children ill with influenza should be isolated and the discharges from the nose and throat destroyed, just as in diphtheria and scarlet fever. Influenza should be a reportable disease and the same precautions taken to protect the public as with other contagious diseases.

TYPHOID FEVER

The etiology of typhoid fever and the methods of infection are the same at all ages. The pathologic changes and symptomatology are essentially the same, but are modified somewhat by age. The principles of diagnosis are also the same.

Fetal and Congenital Typhoid

Abortion occurs in from 50% to 70% of all pregnant women who contract typhoid fever. Among the possible causes of abortion are the high temperature, the accumulation of toxins in the maternal blood and the death of the fetus. The last is probably the most common cause. It may be the result of the high temperature, the passage of toxins through the placenta or intrauterine typhoid, probably most often of the last.

Under the head of fetal typhoid are included the cases which are born dead or die at birth, while under the head of congenital typhoid are included the cases which are born alive and suffering from typhoid. If infection of the fetus occurs, death usually takes place in utero. If it is born alive, it usually succumbs rapidly to an acute cachexia without special characteristics. If it lives longer, it may develop some of the classic symptoms of extrauterine typhoid and may possibly survive the disease. There is some evidence to show also that the fetus may have typhoid in utero, recover and be born alive and well.

The pathologic lesions in intrauterine infection are general rather than local. No changes are found in the intestines and mesenteric lymph nodes, unless the infant has lived for some time after birth. The explanation of the lack of intestinal changes is probably in part the nature of the infection, through the blood, and in part the fact that the intestine is not functionating before birth. It is probable that the septicemic nature of the infection accounts for the high mortality.

Infantile Typhoid

Typhoid fever is relatively less common in infancy than in childhood. Its relative infrequency during the first year, when the infant is fed on breast milk or some food which has been heated sufficiently to destroy typhoid bacilli and the water boiled, is easily explainable. After the first year, when the exposure, through food, at least, becomes the same, it is hard to see why it should not be as common as in childhood. Nevertheless, it apparently is not.

The symptomatology is essentially the same as in early childhood, except that diarrhea and distention are more common. The mortality, however, is apparently considerably higher, being about 50% in the reported cases. It is probable that the mortality is not as high as this, as many mild cases are presumably unsuspected and overlooked.

Typhoid Fever in Childhood

Typhoid occurs with about equal frequency at all periods of childhood and is apparently about as common as in adults. The course is shorter and the prognosis better than in adults, the mortality being only about 6%, or one half that in adults. The younger the child, the shorter on the average is the duration of the disease, the less the severity of the symptoms and the lower the mortality.

Pathology.—The relative mildness of the disease is probably to be explained by the comparatively slight development of the intestinal lesions. Those of the solitary follicles and Peyer's patches seldom go beyond hyperplasia. Ulceration is unusual and, in consequence, hemorrhage and perforation extremely rare. The severity of the lesions, as a rule, varies directly with age. The involvement of the bronchial mucosa is, however, more marked than in later life. The changes in the other organs are the same as in later life, but usually less marked.

Symptomatology.—The following description is based on an analysis which I made some years ago of about three hundred cases treated at the Boston City Hospital and a considerable personal experience at the Children's Hospital, Boston, at a time when typhoid fever was much more common than at the present day.

Onset.—The onset is far more often acute in early life than later. The younger the child, the more often is the onset acute. The apparent acuteness of the onset may be due in part, however, to the failure of the young child to speak of the early symptoms or of its parents to notice them. A chill at the onset is very uncommon. Vomiting is not infrequently the first symptom noted, however, especially in young children.

Epistaxis.—Nose bleed is as common as in adults, occurring in about 50%. It is, however, less often an early symptom.

Fever.—The average duration of the fever is less than in adults, being about twenty-one days between the ages of five and ten and twenty-five days between ten and fifteen years. The proportion of cases in

which the duration of the fever is less than ten days is twice as great in the younger children, 7% as against 3%. Not only the average duration of the fever but also the average height of the temperature varies directly

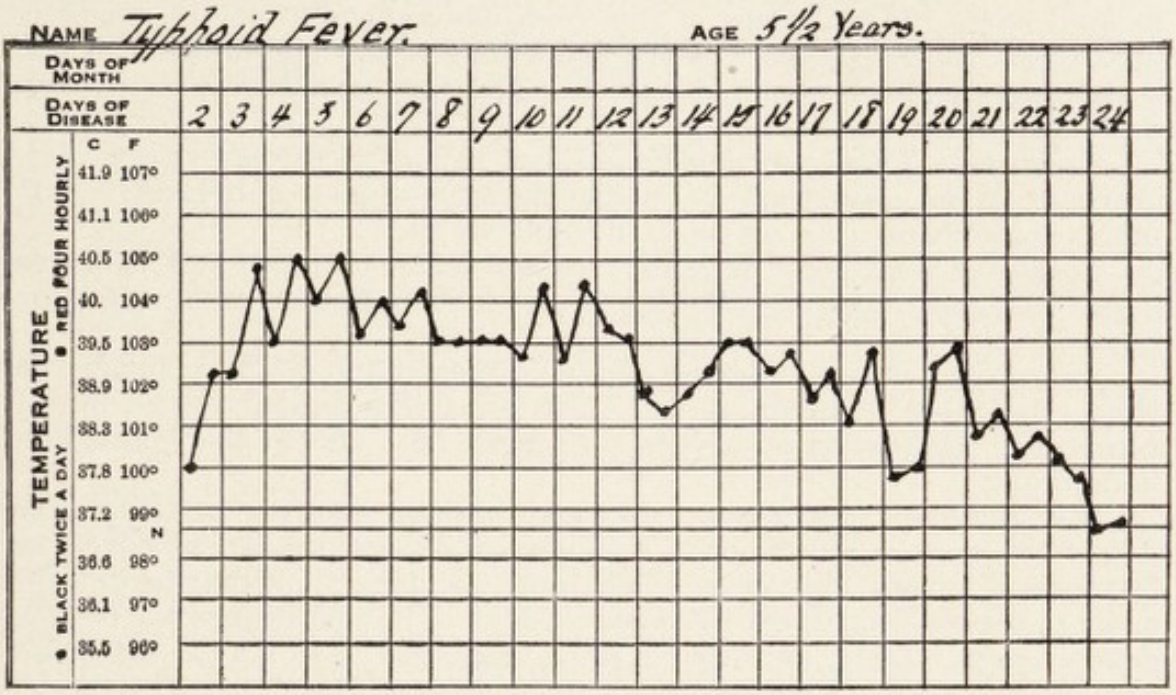

FIG. 93.—Typhoid fever.

with the age of the child, the average temperature ranging lower in the younger children. The rise of the temperature to the maximum is usually much more rapid in children than in adults, the height usually being

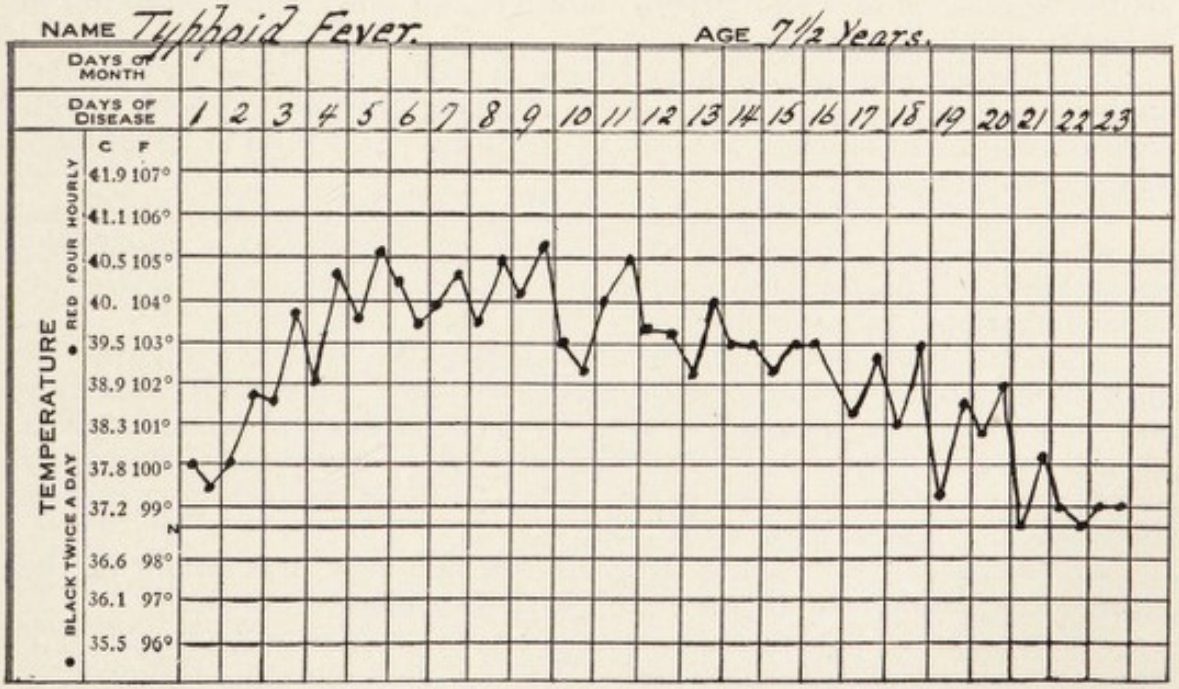

FIG. 94.—Typhoid fever.

reached in four or five days and not infrequently in two or three days. Remissions in the temperature during the height of the disease are more common than in adults. They are usually not marked. The remittent

and intermittent stages, so marked in the temperature of typhoid fever in adult life, are, however, absent in about one half of the cases in childhood. They are more often absent in the younger children and are always less marked. The temperature usually comes down to normal by a rapid lysis. The absence of the remittent and intermittent stages is usually attributed to the absence of ulceration and suppuration in the lymphatic structures of the intestines. This may or may not be the true explanation.

Headache.—There is headache in about 75% of all cases. It is severe, however, in not more than 5% of the cases in which it is present. It is more common and more severe in the older children.

Tongue.—The tongue is usually coated but seldom dry and brown as in severe adult typhoid. Sordes is also uncommon in well cared for cases.

Vomiting.—This is far more common in early life. The younger the child, the more common it is. It is especially common as an initial symptom. Unless very severe, it does not affect the prognosis unfavorably.

Diarrhea and Constipation.—Marked diarrhea does not occur in over 2% of the cases in childhood. It is almost always present, however, in fatal cases. Constipation is much more common than diarrhea, especially in the children under five years of age. The condition of the bowels depends very largely, however, in most cases, just as in adults, upon the care which is taken in the regulation of the diet and the attention given to the bowels.

Tenderness and Distention of the Abdomen.—Tenderness of the abdomen is not common and is almost never marked. Pain is unusual. There is usually some distention, or at least fullness, of the abdomen. It is more likely to be marked in the younger children. Like the condition of the bowels, it depends largely, however, on how much attention is given to regulation of the diet.

Hemorrhage.—Intestinal hemorrhage is very uncommon in children under ten years of age. It occurs in children between ten and fifteen years of age about one half as often as in adults. It is fatal in about 50% of the cases in which it occurs.

Perforation.—Perforation of the bowels is most unusual. It almost never occurs in children under five years of age, but after this, age apparently plays no part in the frequency. The symptoms are apparently less characteristic than in adults. I have never seen a case.

Roseola.—Rose spots are as common in early life as later, being present in from 60% to 70% of all cases, regardless of age. In certain cases they are very abundant, so much so that the French describe them under a special name,—typhoide exanthemique. They claim that the other manifestations are less severe in this type. It must not be forgotten that the spots may appear first on the back.

Erythematous rashes and urticaria are not very uncommon in the course of typhoid in childhood. Herpes also develops occasionally.

Splenic Tumor.—The spleen is palpable in between 80% and 90% of the cases of typhoid in childhood. It is not infrequently considerably enlarged, especially in the younger children. Percussion is unreliable in young children and it is, therefore, not safe to consider the spleen enlarged unless it can be felt.

Sore Throat.—Inflammation of the fauces and pharynx is a rather marked symptom in something more than 5% of the cases. In a number

of instances which I have seen it was so marked that it dominated the picture and for some time prevented the true diagnosis from being made.

Bronchitis.—Pathologically, there is a bronchitis in every case of typhoid in early life. Clinically, it is evident in about 40%. It is usually more marked in the younger children and not infrequently leads to errors in diagnosis. In rare instances it may be purulent. Bronchopneumonia may develop secondarily in severe cases and in feeble children. It is due to secondary invaders. In very rare instances lobar pneumonia, due to infection of the lungs with the bacillus typhosus, may develop. Recovery from it is the rule. I have never seen dry pleurisy in typhoid in children.

Pulse and Heart.—One of the most characteristic symptoms of typhoid in early, as in adult life, is that the rate of the pulse is relatively slow compared with the increase in temperature. The value of this relation, to which much importance was attached in the past, is not sufficiently appreciated at present. It is one of the most important points in diagnosis. The fullness and compressibility of the pulse is less marked in childhood than later.

There is nothing characteristic about the heart. It shows the changes common in all febrile diseases. Endocarditis almost never develops. Murmurs when present are, therefore, almost always inorganic or due to muscular weakness.

Cerebral Symptoms.—These are present in about one quarter of the cases, independent of the age. From 15% to 25% of these cases show various grades of stupor. The others are more or less delirious, the delirium being active in from two-thirds to three-quarters of them. A rather characteristic manifestation is crying out sharply at intervals, especially during the night. Severe nervous manifestations, such as coma vigil, subsultus and picking at the bed clothes, almost never develop in childhood.

Aphasia, while very uncommon at any age, occurs much more often in childhood than later. It may be due to an embolus, but is almost always functional. It develops during convalescence. Recovery is the rule. The average duration is three weeks.

Urine.—The urine shows the usual evidences of acute degenerative nephritis. Evidences of acute nephritis are very rare.

Blood.—The white blood count is not increased and in many cases is somewhat diminished. This is a very important point in differential diagnosis. There is also likely to be a slight relative increase in the lymphocytes. This is less constant and of less importance in diagnosis. An increase in the number of white corpuscles in the course of typhoid means the presence of some complication, usually inflammatory.

The Widal reaction occurs under the same conditions and with the same limitations in children as in adults. There is some evidence to show, however, that in them the reaction appears earlier, is feebler and persists for a shorter time than in adults. The Widal reaction appears in infantile typhoid as in that of older children and adults. The presence of the reaction in infancy, especially in early infancy, is of less diagnostic value, however, than in adult life, as the reaction may be transmitted from the mother through the placenta or through the milk. The agglutinating power may be transmitted to the infant through the placenta, not only during the course of or convalescence from typhoid but even when pregnancy takes place years after recovery. It is probable that it may also be transmitted through the milk after many years. When

transmitted through the milk, however, it does not persist more than a week after the cessation of nursing. In early infancy, therefore, a positive Widal reaction is of somewhat less diagnositic value than in older children and adults. If the mother has had typhoid and especially if she is nursing the infant, it should be looked on with some suspicion unless associated with other characteristic signs of typhoid. Examination of the mother's blood and milk and the cessation of breast feeding will then assist in estimating the true value of the reaction in the infant.

Complications and Sequelæ.—Many of what are usually considered complications have already been spoken of as symptoms. Some of them, however, deserve further mention, especially as regards diagnosis. It must not be forgotten that a little blood in the stools, especially if it is bright, does not necessarily mean an intestinal hemorrhage. It may come from a crack at the anus or be due to the irritation caused by the thermometer, when the temperature is taken repeatedly in the rectum. It must also be remembered that perforation of the intestines is very uncommon in childhood and that there are other conditions which give symptoms very much like it. The most common of these is inflammation of the mesenteric and retroperitoneal lymph nodes. Acute appendicitis in the course of typhoid is probably as common as perforation. Impacted feces may also at times cause symptoms suggestive of perforation.

Otitis Media is a common complication of typhoid, as of all other severe febrile diseases in childhood. It occurs more frequently in the younger children and in those seriously ill. It should always be thought of when there is a rise in the temperature without evident cause. As a rule, there are no subjective symptoms or, at any rate, they are not mentioned. The inflammatory process is due to secondary invasion by the pus organisms. There is, therefore, usually a leucocytosis. In other instances there is deafness without any evidences of inflammation of the middle ear. In such cases recovery of the hearing during convalescence almost always takes place.

Parotitis.—Inflammation of the parotids occasionally develops and is a serious complication. It is quite likely to go on to suppuration and to require operation.

Neuritis and Inflammation of the Bones and Joints are much less common sequelæ in childhood than in adult life, but may occur. There is nothing characteristic about the symptomatology at this age.

Relapses.—These occur at least as frequently in children as in adults. In them, as in adults, the duration and height of the fever and the severity of the symptoms are usually less than in the original attack.

Diagnosis.—Typhoid fever should always be suspected when a child has a continued fever without evident cause. If the rate of the pulse is not increased proportionately to the rise in the temperature, typhoid is still more probable. A low white count is still stronger evidence in favor of typhoid and almost justifies a positive diagnosis of it. Enlargement of the spleen and the presence of rose spots make the diagnosis certain. A positive Widal test, if it has been previously absent, also proves the diagnosis. As it seldom appears before the second week of fever, and often not until later, it is of less value than the slow pulse and the absence of leucocytosis. The diagnosis ought to be evident in most instances before the appearance of the reaction. Examination of the blood and feces for typhoid bacilli is impracticable for the general practitioner and usually unnecessary, as it is possible to make the diagnosis in other ways.

Acute miliary tuberculosis and typhoid may sometimes be confused. Both have a continued fever and a low white count. The spleen may be enlarged in acute miliary tuberculosis, but is enlarged much less often than in typhoid. Tuberculides sometimes have a superficial resemblance to rose spots, but they are always slightly elevated, usually are desquamating a little on top and do not fade on pressure, while rose spots are usually not elevated, never desquamating and fade on pressure. The tuberculin test is useless, because it is usually absent in acute miliary tuberculosis and may be present in typhoid fever, if the patient has a tuberculous focus anywhere in the body. The pulse is increased in rate in tuberculosis, while it is relatively slow in typhoid. The Widal test is helpful. The course is steadily downward in acute miliary tuberculosis, while in typhoid fever it usually is not.

Malaria is sometimes confused with typhoid fever, chiefly because of the enlargement of the spleen. In malaria, however, the spleen is usually firmer and the temperature more irregular. The white count is low in both. There is no roseola in malaria. The Widal test is negative and the plasmodia can always be found in the blood.

Septic infection is sometimes mistaken for typhoid, but typhoid is seldom mistaken for septic infection. There is a continued fever in both. The temperature is more irregular, however, in sepsis. The spleen may be enlarged in septic infection. The enlargement is less frequent, however, than in typhoid fever. There is no roseola in septic infection, although there may be erythematous and urticarial eruptions. The rate of the pulse is always increased in septic infection, usually out of proportion to the rise in the temperature, while in typhoid the rate of the pulse is slow compared with the increase in temperature. The white count is normal or diminished in typhoid and always increased in septic infection, unless the patient is moribund. This point is sufficient to settle the diagnosis. Furthermore, careful search will almost always reveal the focus of infection in septic infection.

The diagnosis between typhoid fever and *paratyphoid fever* is often very difficult, the symptomatology being essentially the same in both diseases. In general, however, the symptoms are milder in paratyphoid fever. The diagnosis can only be made on the absence of the Widal reaction and the presence of a positive reaction to the paratyphoid bacillus of either Type A or B.

The bronchitis in typhoid may sometimes cause confusion and lead to an erroneous diagnosis of bronchitis, the local infection attracting the attention away from the general disease. The rate of the pulse is, however, not increased as in ordinary bronchitis, and the white count is normal or but slightly increased, while in ordinary bronchitis it is usually considerably increased. The finding of enlargement of the spleen and of rose spots, of course, makes the diagnosis plain. In other instances inflammation of the throat is the most prominent symptom for some days or even a week or more, drawing attention away from the general infection. In these cases the pulse is relatively slow and the white count low in contradistinction to the conditions usually found in inflammation of the throat. The diagnosis of typhoid fever in cases where the symptoms of inflammation of the throat and of the bronchi predominate should be possible without a Widal test.

In infancy especially the diarrheal diseases may be mistaken for typhoid fever or possibly typhoid fever for one of the diarrheal diseases. Confusion is possible, however, only when the observer is careless. The

temperature range is materially different in the two conditions. The pulse varies with the temperature or rises out of proportion to the temperature in the diarrheal diseases, while in typhoid it is relatively slow. There is always a leucocytosis, usually marked, in the diarrheal diseases, while there is no leucocytosis in typhoid fever. There are never rose spots in the diarrheal diseases while there usually are in typhoid. The spleen may be enlarged in both. Nothing can be told from the stools, unless they are those characteristic of infectious diarrhea. Such stools never occur in typhoid.

Treatment.—Typhoid fever is a preventable disease. The precautions to be taken to avoid it are the same in early as in adult life. If it has developed, the treatment is, of course, along the same lines as in adults. Certain modifications are, however, necessary. Antityphoid vaccine should be used as a prophylactic in childhood as in adult life. The dose for a child of ten years is one half of that for an adult. That for younger children is proportionately smaller.

A child ill with typhoid fever must be put to bed and kept there until at least a week after the temperature has reached and remained normal. It may be necessary to keep it in bed much longer than this. It is inadvisable to allow a child to begin to get up as long as the spleen is palpable, because relapses are very likely to occur when the spleen remains enlarged.

It is very important to give a child ill with typhoid considerable amounts of food. When sufficient food is given, the marked emaciation and weakness, so commonly seen during the latter part of typhoid and convalescence in the past, are very unusual. The child should get at least as many calories as it requires when it is well and should also get at least enough protein to cover its minimum protein need. It is wiser for it to get more, because of the increased destruction of protein when there is fever. It is impossible to properly cover the caloric needs when the diet is exclusively milk and broth, as was formerly the rule. Furthermore, an exclusive milk diet is very likely to cause marked constipation. There is, of course, no nourishment in broths. There is no reason why a child with typhoid should not take any of the starches, as well as eggs and milk. There need be no fear that things like toast and crackers will be hard and irritating when they reach the lower part of the small intestine and the large intestine, where the lesions of typhoid fever are located. In my experience it is inadvisable, however, to give meat, green vegetables or fruit, although many physicians give orange juice freely. The following diet is a suitable one:

Milk	Oatmeal	Butter
Cream	Cream of Wheat	Junket
Milk Soups	Wheat Germ	Baked Custard
Soft Boiled Eggs	Ralston	Corn Starch Pudding
Dropped Eggs	Farina	Bread Pudding
Scrambled Eggs	Rice	Rice Pudding
Toast Bread	Hominy	Blanc Mange—Plain
White Bread	Baked Potato	Gelatines
Whole Wheat Bread	Mashed Potato	Ice Cream
Plain Crackers	Plain Macaroni	
Milk Toast	Plain Spaghetti	

It is very easy to lay out a suitable diet for a child with typhoid and to say that it should take so many calories and so many grammes of protein per day. It is another thing, however, to make the child take it. I defy anybody to make certain children take as much food as they

should theoretically during the height of the disease. Anybody can get them to take it as they become convalescent. In fact, the difficulty then is to prevent them from taking too much. When it is difficult to get in the desired number of calories, the addition of milk sugar to the milk and sometimes to other foods may help, because the child does not notice it.

It is advisable, as in all febrile diseases, to give considerable amounts of water. The amount of water taken should be charted in the same way as the amount of food.

The temperature in typhoid fever in early life is seldom high enough or of long enough duration to require any active treatment. Treatment for the temperature should not be instituted simply because it happens to reach some arbitrary point. It should be given whenever the temperature of itself causes depression or disturbance of the nervous system, whether the point at which this occurs is 102° F. or 104° F. It is inadvisable to attempt to lower the temperature by the administration of any of the antipyretic drugs. It is far wiser to use cold externally. Sponge baths of alcohol and water, equal parts, at 90° F., are usually sufficient. If they are not, fan baths are almost certain to be effectual. Fan baths are given in this way: The child is stripped and wrapped in cheese cloth. This is then wet with water at 100° F. and the patient fanned. The fanning may be done with an electric fan or with palm leaf fans. The temperature is reduced by the evaporation of the water. The cheese cloth is wet from time to time as the water evaporates. Children seldom object to this form of bath. If this is ineffectual, cold packs at from 60° F. to 70° F. may be used. Children seldom bear tub baths well. I have in the past seen many children injured by them at the time when it was the fashion to give tub baths to everyone ill with typhoid fever. In fact, if the application of any form of cold disturbs the child a great deal, it probably does more harm by depressing it and weakening it than it does good by lowering the temperature. Cold should not be applied regularly once in so many hours regardless of the symptoms, as it so often is, but whenever it is necessary to relieve the symptoms caused by the high temperature.

There are no drugs which have any effect on the activity of the typhoid bacilli in the intestines or elsewhere in the body and no drugs which have any appreciable affect on either abnormal fermentative or putrefactive processes in the intestines. If diarrhea develops, it is due to abnormal fermentative or putrefactive processes in the intestinal contents and should be treated by regulation of the diet on the same lines as in other diarrheal conditions in early life. If there is abnormal fermentation with acid irritating stools, the proportion of carbohydrates in the diet should be diminished and that of the proteins increased. If the stools are alkaline and foul, the protein in the diet should be diminished and the carbohydrates increased.

Constipation is seldom troublesome in typhoid, if a sufficient amount of food and water is given. If it does develop, it is usually better to treat it with enemas from below than by drugs by the mouth. If laxatives are given, the mild salines in small repeated doses are the best.

Distention is seldom troublesome, if the diet is properly regulated and the bowels are looked after. If it develops, turpentine stupes and turpentine enemas are the most useful remedies. The rectal tube seldom affords much relief. The treatment of hemorrhage and perforation is, of course, the same as in adult life.

Stimulation is seldom required. If it is, it is along the same lines as in pneumonia and other acute diseases. I have had no experience with the use of vaccines in the treatment of typhoid.

MALARIA

The etiology and pathology of malaria is the same at all ages. The symptomatology is less definite in early than in adult life, however, and the diagnosis, therefore, rather more difficult. Malaria is very uncommon in Eastern Massachusetts, and is becoming more so every year. I have never seen a case of malaria originating in this part of the country, which was not due to the tertian organism. The infection is more often single in children and double in infants. My youngest patient was only a few days old, and the infection was apparently congenital.

Symptomatology.—My experience with malaria in early life having been limited to infection with the tertian organism, what I have to say regarding its symptomatology necessarily applies only to such infections. In middle and late childhood, typical malarial paroxysms are not uncommon. In early childhood and infancy, however, chills are likely to be replaced by vomiting, and occasionally by convulsions. Infants, especially, are often apathetic or somnolent, sometimes collapsed and cold. Not infrequently they are cyanotic at the onset, but not as often as during the third stage. Pallor and collapse are more common during the third stage in infancy and early childhood than sweating. In some instances, however, profuse sweating is the most marked symptom. There is nothing characteristic about the temperature in early life. In some instances, the symptoms during the paroxysms are chiefly respiratory, the breathing being rapid and the chest full of râles. In other instances, the chief symptom is diarrhea. Pain in the head and abdomen is less common in early life than later. In general, there is a definite periodicity in the symptoms, but the younger the individual, the less marked is the periodicity likely to be. In infants and young children the development of the disease may be very insidious.

Anemia develops very rapidly in most instances, especially in infants. It is secondary in type. There is no leukocytosis. There is said to be a relative increase in the proportion of large mononuclear cells. This increase is not great enough, however, to be of any practical importance in diagnosis. Enlargement of the spleen is almost invariable, occurs early, and is usually marked, sometimes being enormous. Tenderness over the spleen is not uncommon.

Diagnosis.—The diagnosis of malaria is easy when the typical paroxysms are present, but is often difficult in early life, when they are absent. The most characteristic thing about the symptoms, when there are no typical paroxysms, is the periodicity of their occurrence. No matter what the symptoms are, malaria should always be thought of, therefore, whenever there is any periodicity about them. Malaria should always be thought of as a possibility when children have an irregular fever or become debilitated or anemic without apparent cause. Enlargement of the spleen, under such conditions, increases the probability of malaria as the cause. In Eastern Massachusetts, however, malaria is the least common cause of such symptoms. The more usual explanations are pyelitis, toxic absorption, or tuberculosis.

Whenever there is any possibility of malaria, the blood should be examined at once for plasmodia. The question can be quickly and

positively settled in this way. The diagnosis of malaria is not justified unless the plasmodia are found in the blood. Incidentally, the presence of a leukocytosis is very strong evidence against malaria. It does not positively exclude it, but shows that if there is malaria, there is also something else the matter.

It is far easier to find the tertian organisms in smears of the fresh blood than in stained specimens, because of the movement of the pigment granules in the organisms when they are alive. The organisms are larger and easier to see a few hours before a paroxysm than at other times. Care must be taken not to confuse vacuoles in the red cells with the young hyaline forms. If vaseline is smeared around the edge of the cover glass, the specimen will not dry for several hours. The easiest stain to use is Wright's. It does not give as clear pictures, however, as some of the old double stains with eosin and methylene blue after baking. If quinine has been given, several examinations may be necessary before the organisms can be discovered.

Prognosis.—Tertian malaria in early life yields quickly to treatment. Recurrences may occur under the same conditions as in adults. The anemia gradually disappears when the cause is removed. The splenic tumor quickly disappears in acute cases and eventually in those of longer duration.

Treatment.—The plasmodium of malaria is destroyed by quinine. It is more susceptible to quinine when it is outside than when it is inside the red corpuscles. It is outside of the corpuscles at the onset of the paroxysm, but quickly reënters them again. Quinine should be given, therefore, long enough before the paroxysm so that it will be in solution in the blood when the organisms are extracorpuscular. Quinine appears in the urine in thirty minutes after its exhibition by mouth. About one half of the quantity ingested is excreted within six hours, and the whole in twenty-four hours. The best time to give the drug is, therefore, one or two hours before the time at which the paroxysm is expected. Theoretically, one dose for each group of organisms should be sufficient; practically, it is wiser to give two doses for each group in order to catch the second time those which escaped the first. Further doses are ordinarily unnecessary. If there are no definite paroxysms, the quinine may be given three times daily for four days. As a matter of fact, tertian malaria in Eastern Massachusetts yields promptly to quinine, however given.

Children, as a rule, bear quinine well. In general, the dose is one grain for each year of age. Twice these doses, however, may be safely given. Quinine should not be given in the form of pills. Fresh triturates and capsules are allowable, but not as good as solutions. The tannate, which has less taste, is feeble and untrustworthy. I object to putting quinine in chocolate and to other attempts to disguise its taste. The chocolate and other messes upset the digestion, and, as a rule, the amount of quinine taken in this way is insufficient. Quinine is most effective when given in solution. No matter if it is bitter. The child should be made to take it because it is good for it. In any event, only a few doses are required.

The acid salts of quinine are much more soluble than the neutral salts. It is advisable, therefore, to use the bisulphate or acid hydrochlorate rather than the sulphate or hydrochlorate. The bisulphate is dissolved in ten parts of water and the acid hydrochlorate in less than its own weight. Eight grains of the sulphate will dissolve in one ounce

of water, if twelve minims of dilute sulphuric acid or of aromatic sulphuric acid are added. The dose of the bisulphate is 25% greater than that of the sulphate.

If the quinine is vomited, it may be given in a suppository, the dose being twice as large as that by mouth. It is less reliable, however, when given in this way. It may also be given subcutaneously. The form usually employed is the hydrochloride of quinine and urea, which is soluble in an equal weight of water. I have never found it necessary to use quinine subcutaneously.

Children recover from malaria more quickly, if they are kept in bed as long as there are any organisms present in the blood, or, at any rate, until at least two days have elapsed without any paroxysms or fever. It must not be forgotten, moreover, that the child is not well, even if the plasmodia have been destroyed. More care is necessary during convalescence than is usually given. It is advisable to give iron during the convalescence to aid in overcoming the anemia. The saccharated oxide is a good form, the dose being about one grain three times daily for each year of age.

WHOOPING-COUGH

(PERTUSSIS)

Whooping-cough is a serious and fatal disease in infancy and early childhood. It is not a trifling affair as it is usually considered to be by the laity, but a condition of the utmost gravity. "Any disease which kills ten thousand children per annum is," as Rucker says, "a serious one. If bubonic plague were to kill that many children in the United States in one year, the whole world would quarantine against our country. A child dead of whooping-cough is just as dead as a child dead of plague." The mortality from whooping-cough is, moreover, much greater than it appears, because a considerable proportion of the deaths in infants and young children from bronchitis and bronchopneumonia are really due to whooping-cough, the bronchitis and bronchopneumonia simply being complications of this disease. Roughly, 55% of the fatalities are in the first year and 25% in the second year. It is a comparatively mild affection in healthy children over five years of age, and after puberty it is rarely fatal.

Immunity.—There is no inherited immunity to whooping-cough. It is true that it is less frequent in the first few months of life than later in infancy. This is not due to immunity, however, but simply to the exposure to contagion being less at this time. No one escapes whooping-cough. Most individuals contract it the first time that they are exposed. This accounts for the age incidence. One half of the cases are in the first two years of life. Most children have had it before they are seven years old. No one is too old to contract it. Permanent immunity is almost always conferred by an attack. A second attack practically never occurs in childhood. It is probable, however, that occasionally a second attack may occur in adult life. If so, it is usually in women who are taking care of children with whooping-cough and consequently are very thoroughly exposed.

Etiology and Pathology.—Whooping-cough is caused by the Bordet-Gengou bacillus. This is a minute, ovoid, gram-negative cocco-bacillus, which stains lightly by the ordinary methods. It is about the size of the influenza bacillus. Staining of smears of the sputum by the proper methods gives fairly satisfactory results early in the disease. It can be

cultivated on a potato-blood-agar medium, but the isolation and recognition of the organism by cultures is too complicated a procedure for everyday use. It is probable that there are many strains of this organism. An agglutination reaction is present in many cases, but is not constant and is usually not very high. Its presence is proof of whooping-cough; its absence does not count much against it. A complement fixation reaction is present in a considerable proportion of children with whooping-cough and convalescing from it. The frequency of the reaction increases with the duration of the disease. Both of these tests are, however, too difficult of performance to be of practical utility for everyday use. This organism is present in the sputum from the beginning of the catarrhal stage. It is most abundant at this time and in the first two or three weeks of the spasmodic stage. It has been found in the sputum as late as the eighth week of the spasmodic stage.

There is a more or less marked catarrhal inflammation of the larynx, trachea and bronchi. The mucous membrane of the pharynx and nose is also sometimes involved. This inflammation is due to the Bordet-Gengou bacilli, which are present in large numbers between the cilia of the epithelial cells lining the trachea and bronchi. They are also found free in the bronchial secretions. They are never found in the alveoli. The bronchopneumonia, which is so common a complication of whooping-cough, is due to other contaminating organisms. The other lesions found at autopsy are due to the mechanical effects of the disease and to complications.

The action of this organism is largely mechanical. It is present in such large numbers that it interferes with the normal movements of the cilia. This keeps up a constant irritation, which causes the coughing. The paroxysm of coughing lasts until the mucus is coughed up. The Bordet-Gengou bacillus secretes a mild toxin, as is shown by a slight inflammatory exudate in the trachea and bronchi, a lymphocytosis and, as mentioned above, the production of a specific antibody.

Blood.—There is an increase in the total number of white corpuscles with an absolute and relative increase in the number of lymphocytes in the catarrhal stage of whooping-cough. The leukocytosis and lymphocytosis increase with the severity of the disease, reaching their highest points in the paroxysmal stage. They are, however, not always present and may be absent, if there is some complication which is accompanied by a polynuclear leukocytosis.

Contagious Period.—It is fair to assume that, as the organism is most abundant in the sputum during the catarrhal stage and the first two or three weeks of the spasmodic stage, that whooping-cough is most contagious during the catarrhal and early weeks of the spasmodic stage. As it is also present in the sputum, although in much diminished numbers, during the rest of the spasmodic stage, it is fair to assume that the disease is also contagious throughout the whole of the spasmodic stage.

Kittens, puppies and monkeys may be infected with the disease. This probably happens so seldom under ordinary conditions, however, that infection from animals can, for practical purposes, be disregarded. The Bordet-Gengou bacillus is very easily destroyed outside of the body. Indirect contagion is, therefore, very unusual and of little importance. I have never known of a case contracted in this way. It is probable, however, if an adult held a child in its arms during a paroxysm of whooping-cough and then, within a short time, held another child or a baby in its arms, that the disease might be carried in this way. It is

also possible that, if a child used the same spoon or cup immediately after a child with whooping-cough and the utensil had not been washed, it might contract the disease. The exposure must be very marked, however, for contagion to take place.

The paroxysmal cough, often associated with whooping, which develops so frequently in children when they have a cold or bronchitis after recovery from whooping-cough is not an evidence of reinfection and is not contagious.

Period of Incubation.—This is not positively known. It is usually between one and two weeks, but may be as short as four days and possibly as long as three weeks. A safe rule to follow, and the one which is usually adopted, is that, if no catarrhal symptoms have developed in two weeks from the date of the last exposure, the child has escaped infection.

Symptomatology.—It is customary to divide the disease into three stages: the catarrhal, paroxysmal or spasmodic, and the stage of decline. This division is, of course, an arbitrary one, as the stages run into and overlap each other.

Catarrhal Stage.—There is nothing characteristic about the catarrhal stage of whooping-cough. The symptoms are those of an ordinary nasopharyngitis, with occasionally symptoms pointing to involvement of the larynx or bronchi. There may or may not be a little fever. The cough is usually somewhat out of proportion to the other symptoms. As it goes on, it becomes intermittent and paroxysmal. It usually is worse at night. The most characteristic thing about it is that it resists treatment. Examination shows simply the evidences of a catarrhal inflammation in the nose and throat. The throat is usually somewhat reddened from coughing. In most instances nothing is to be heard in the lungs, in spite of the hard cough; occasionally, however, there may be a few râles.

Spasmodic Stage.—The paroxysmal character of the attacks becomes more marked and finally the characteristic attack appears. This is easy to recognize, but hard to describe. Adults say that there is a sense of suffocation at the beginning of the attack. Children always try to get hold of something to lean on as the attack begins. If they cannot find anything, they bend over and place their hands on their knees. There is then a series of short, barking coughs without any inspiration between them. Finally there is a long, noisy inspiration, which is the whoop. This is immediately followed by another series of coughs and a whoop. There may be six or eight of these spells of cough with whoop before the paroxysm ends with the expulsion of mucus. This is often accompanied by vomiting, especially if the attack occurs soon after a meal. While the attack is going on the face at first becomes red and, if it is severe, markedly cyanotic. The eyes bulge and saliva often runs out of the mouth. After a severe attack the child is often much exhausted, sometimes so much so that it can hardly stand. In other instances, it is confused and dazed. Babies are sometimes unable to breathe and have to be helped by artificial respiration. Not all the attacks are, however, as severe. There may be no whoop or only one or two in a paroxysm.

The number of paroxysms varies from three or four in twenty-four hours to one every half hour or so. I have known a child to have as many as seventy-two in twenty-four hours. The attacks are usually more numerous and more severe at night than during the day. They are

rather less frequent when the child is out of doors. Eating and drinking often bring them on. There is undoubtedly a certain nervous element at the base of many of the paroxysms. They are likely to come on if the child is excited. If one child has a paroxysm, another is likely to. Suggestion may also play a part. I remember that when I had it myself I never got by a certain street corner without losing my last meal.

The average duration of the spasmodic stage is three or four weeks. It may be as short as one week or as long as three months. The recurrence of the paroxysmal cough after it has once stopped, however, does not signify whooping-cough.

There may or may not be a little fever during the spasmodic stage. There usually is none, however, unless there are complications. In most instances in children, nothing abnormal can be heard in the chest, although in babies there are almost always a few râles.

Stage of Decline.—After the cessation of the whoop, which marks the end of the spasmodic stage, the cough still remains and is often somewhat paroxysmal. It gradually diminishes and usually ceases in about three weeks. It may persist for many weeks, however, especially if it is winter.

Atypical Cases.—In some instances, whooping-cough is very mild. There may be no paroxysmal stage at all. In other cases, while the cough is paroxysmal, there is never any whoop, or the child may whoop only once or twice. The duration of the symptoms may be as short as two or three weeks. Such cases are usually overlooked, unless other children in the family have whooping-cough or there has been a definite exposure to whooping-cough. Unfortunately, these mild cases are as contagious as the more marked ones. In other instances, whooping-cough may be very severe, even when there are no complications. Vomiting is so frequent and marked that the child gets but very little nourishment and the paroxysms are so frequent that they prevent sleep. The temperature is often high in these cases, the general condition fails rapidly, and death may occur in young children from exhaustion.

In little babies there may be no whoop in the paroxysm. They sometimes lose their breath entirely, however, and at times are somewhat convulsive and lose consciousness. In the attacks they are markedly cyanotic.

Complications.—Many of the complications, some of which are hardly more than symptoms, are the result of the venous stasis which occurs during the paroxysms.

Edema of the face is very common and is especially marked about the eyes.

Dilatation of the right heart develops in most of the severe cases during the paroxysmal stage. This is shown by an increase in the rate of the pulse and widening of the area of cardiac dulness to the right. There may also be a systolic murmur at the apex as the result of a relative insufficiency of the mitral valves Endocarditis, myocarditis and pericarditis are not complications of whooping-cough.

Hemorrhages are not at all uncommon. Bleeding from the nose is the most common. With some children it occurs with every paroxysm. Bleeding may also occur from rupture of small vessels in the throat, trachea, and bronchi. In such cases, however, the bleeding is usually slight. Hemorrhage into the conjunctivae is not uncommon. Hemorrhage also sometimes takes place in the cellular tissues about the eyes, causing a typical black eye. In rare instances, intracranial hemorrhages develop. They are far more often meningeal than cerebral. The symptoms

and results are the same as from intracranial hemorrhages due to other causes. Most of the cerebral symptoms are, however, presumably due to edema, because they are so fleeting. All sorts of manifestations occasionally develop, such as temporary paralyses of the muscles supplied by the various cranial nerves, fleeting loss of vision, and temporary deafness. Convulsions are not very infrequent, especially in babies and young children. They are probably usually due to edema, but may, perhaps, be due to toxemia. When they occur apart from a paroxysm, it is probable that they are usually manifestations of spasmophilia. They are not infrequently fatal, if there is a complicating pneumonia or the babies are spasmophilic.

Bronchitis and Bronchopneumonia.—These are the most common and, in infancy, the most serious of the complications. The physical signs are the same as when they are due to other causes, *Lobar pneumonia* is a very rare complication and is somewhat less serious than bronchopneumonia.

A certain amount of vesicular *emphysema* develops in every serious case of whooping-cough. It is shown by slight hyperresonance on percussion. Occasionally one of the alveoli ruptures and interstitial emphysema develops. The air may work its way up through the mediastinum and produce subcutaneous emphysema. This shows usually first as a swelling above or at the sides of the sternum. In some instances it is so marked that it spreads over the chest and up over the neck.

Ulceration of the Frenum.—Ulceration of the frenum of the tongue is not uncommon from the irritation of the teeth as the tongue is pressed against them in coughing. It may occur in other conditions than whooping-cough, if the cough is severe.

Digestive System and Nutrition.—The digestion is not usually affected, except as it is in any disease, if there is much general disturbance of the nutrition. Sometimes there is really loss of appetite. Usually it is only apparent, the children being afraid to eat lest eating bring on an attack of coughing and vomiting. The vomiting which occurs after cough is not a symptom of indigestion. The bowels are likely to be constipated as the result of an insufficient intake of food. Sometimes, especially in babies, if the nutrition is much disturbed, there is secondary diarrhea. When there is much vomiting the nutrition suffers. Babies may get into a very serious condition of malnutrition.

Albuminuria and Nephritis.—Albuminuria occasionally occurs in uncomplicated cases. Acute degenerative nephritis may develop when there is bronchopneumonia, just as in bronchopneumonia from other causes. I have never seen acute nephritis with whooping-cough.

Measles and Tuberculosis.—Measles and whooping-cough are frequently associated. The association is, however, accidental. Neither can be regarded as a complication or sequela of the other. Tuberculosis of the lungs occasionally develops with or after whooping-cough. The tuberculosis is not the result of the whooping-cough, however, but a pre-existing tuberculous infection is merely lighted up by the whooping-cough.

Diagnosis.—There is nothing characteristic about the symptomatology of the catarrhal stage, at any rate in the beginning. It is like that of an ordinary cold. Toward the end of this stage, however, the paroxysmal character of the cough, its greater frequency at night, the discrepancy between the severity of the cough and the paucity of the physical signs, and the failure of treatment to alleviate the cough are all suggestive. A history of exposure to whooping-cough is also of considerable importance. The examination of the blood is also sometimes of

assistance, there being in the catarrhal stage of whooping-cough both a leukocytosis and a lymphocytosis. There is either no leukocytosis or a leukocytosis with a relative increase in the polynuclear neutrophiles in the diseases with which whooping-cough may be confused. This blood formula is a fairly constant one in whooping-cough and is, therefore, of considerable assistance in the diagnosis before the appearance of the characteristic whoop. The sputum at this time also usually contains large numbers of Bordet-Gengou bacilli. Unfortunately, unless there is some other good reason for suspecting that the cough may be whooping-cough, no one thinks of examining either the blood or the sputum.

The diagnosis is easy in the spasmodic stage, if there are typical paroxysms. It may be very difficult in those cases in which the cough is periodic, but in which there is no whoop. The tendency of the child to try to get support during an attack of coughing is very suspicious. The severity of the cough in relation to the slightness of the physical signs is also very suggestive. Children with a catarrhal involvement of the upper air passages from other causes may have, however, a paroxysmal cough, whoop occasionally and even vomit. Children with enlargement of the tracheobronchial lymph nodes also sometimes have a paroxysmal cough with drawing in of the breath and sometimes with vomiting. Even if the enlargement of the tracheobronchial lymph nodes can be demonstrated by physical examination and the Roentgen ray, it does not rule out whooping-cough, because these nodes are enlarged in whooping-cough. The onset of the symptoms is, however, usually slower in enlargement of the tracheobronchial lymph nodes than in whooping-cough. The cough after influenza often resembles to a certain extent that of whooping-cough. This cough is probably often due to enlargement of the tracheobronchial lymph nodes. The examination of the blood is just as useful in the spasmodic stage as in the catarrhal. The lymphocytosis is, however, not always present and may be absent, if there is some complication which is accompanied by a polynuclear leukocytosis. Due allowance must also be made in infants and young children for the normally high percentage of lymphocytes at this age. The examination of the sputum is also of use during the first two or three weeks of the spasmodic stage. After this the Bordet-Gengou bacilli are less and other bacteria more numerous. The complement fixation test may also be of use. Practically, however, it is seldom available.

The diagnosis of whooping-cough is not likely to be made during the stage of decline, if it has not been made previously. Recurrences of a paroxysmal cough, even if there is whooping, during convalescence and after do not mean a recurrence or relapse of the whooping-cough.

Prognosis.—The mortality of whooping-cough is roughly 25% in the first year, 15% in the second year, 3% between two and five years and less than 2% after five years. These figures are of little value, however, in the individual case. The prognosis then depends on the general condition of the infant, how it is fed and cared for, the severity of the disease in the individual instance and whether complications develop in the lungs. After infancy whooping-cough is seldom fatal, unless there are pulmonary complications. The presence of severe rickets and of spasmophilia make the prognosis much worse.

Treatment.—*Prophylaxis and Quarantine.*—The first step to be taken in the prophylaxis of whooping-cough is to convince physicians and the public in general of the seriousness of whooping-cough in infants and young children and of the importance of protecting them from it. All

regulations, no matter how well-planned, will be useless until those interested in their enforcement are so convinced. They can be convinced only by a campaign of education. When the public and physicians are properly educated, regulation will be relatively simple.

Whooping-cough must be made everywhere a reportable disease and the same penalties imposed for failure to report it as in the case of smallpox, scarlet fever and diphtheria. The house should be placarded and the inmates instructed by the health authorities as to the seriousness of the disease in infancy and the methods to be employed to prevent contagion. The sputum and vomitus should be treated in the same way as in tuberculosis.

The patients should be separated from the other children in the family, if they are under five years of age. If such separation is impossible, the patients should be removed by the health authorities to special hospitals provided for the purpose. There is no reason why children should be isolated in one room and not allowed to go out of doors, provided they are kept away from other children. They should not be allowed on the street at all during the acute and most contagious stage of the disease. Later it may be allowable for them, provided they are attended by an adult. In order to protect other children in case they do get on the street, they should be required to wear an arm band of some prescribed color and labeled "Whooping-cough" in large letters. They should not be allowed under any circumstances to visit places of public congregation or to travel in public conveyances. Children who fail to observe the quarantine regulations should at once be sent to the hospital by the public authorities, no matter what their circumstances or social position. They should not be released from quarantine until after the cessation of the paroxysmal cough or until at least six weeks have elapsed since the onset, even if the paroxysmal cough has ceased.

The vitality of the Bordet-Gengou bacillus outside of the body being slight, formal disinfection is not necessary. Thorough cleaning and airing of the premises is all that is required.

The other children in the family should not be allowed to attend school unless they have already had the disease or until two weeks have elapsed since the last exposure, provided they are free from catarrhal symptoms. Children with catarrhal symptoms, in whom there is any reason to suspect the possibility of whooping-cough, should also be excluded from school.

Fresh Air and Sunlight.—It is not necessary to keep children with whooping-cough in bed, unless the temperature is above normal or they have pulmonary complications. Fresh air and sunlight, especially fresh air, are of more benefit in whooping-cough than any other remedial measures. It is very likely that the old treatment, so much in vogue at one time, of taking children with whooping-cough to the gas house owed its popularity to the fact that the children who were carried to the gas house got out of doors while the others were kept in the house. It is not advisable, however, to take children with whooping-cough out of doors in all sorts of weather. They should be kept in when it is stormy, blowy or very cold. If it is necessary to keep them in the house they should, if possible, be changed from room to room, the room which they are not in being very thoroughly aired in the meantime.

Food.—It is very important and at times very difficult, because of the loss of appetite and vomiting, to give sufficient food to keep up nutrition. There are no special indications as to the kind of diet to be given, except

that it is advisable to give foods containing a high caloric value in small bulk and foods which leave the stomach fairly quickly. It is foolish, for example, to give broths and soups which have very little nutritive value. The basis of the diet must necessarily be milk. Soft foods are less likely to bring on a paroxysm of coughing than hard. It is advisable to give another meal, usually a small one, soon after a paroxysm in which the previous meal has been vomited. Some children are willing and glad to eat at once, others have to be urged.

Medicinal.—There is no drug which has any specific action in whooping-cough. This fact is plainly shown by the multiplicity of the drugs which are recommended.

Local applications to the nose and throat are useless, because the seat of the lesion is not in the nose and throat but in the trachea and bronchi. Furthermore, making the application is likely to bring on a paroxysm of coughing.

Local applications to the chest are also useless. It is impossible for anything which is rubbed on the outside of the chest to have any effect on the Bordet-Gengou bacilli in the trachea and bronchi or upon any pathologic process in them.

Inhalations are also practically of little utility. It is impossible to inhale any drug in sufficient quantities to have any effect on the action of the Bordet-Gengou bacilli. It is barely possible that local irritation may be slightly diminished by them. Creosote and vapo-cresolene are the preparations most often used. The chief advantage derived from them is that they make such a bad smell that everyone knows that somebody is sick, probably with whooping-cough. There is some danger with both of them that irritation of the kidneys may be set up. The chief objection to their use, however, is that the windows are entirely or partially closed when they are used and the supply of fresh air is, therefore, interfered with.

It is far better not to give any drugs by mouth, unless the case is a severe one, because, in order to do any good, they have to be given to the physiologic limit. None of them have any curative action. All of them, if given in sufficient doses, diminish, to a greater or less extent, the number and perhaps the severity of the paroxysms. The beginning dose of the tincture of belladonna for a baby is one quarter of a minim and for a child of four or five years one minim three or four times daily. The dose should be increased until there is slight dryness of the throat, flushing of the face or dilatation of the pupils. It should then be dropped down to the dose which does not give these symptoms and kept there. The dose of antipyrin for a baby of six months is one grain every three hours, for a child of two years two grains every four to six hours. It is not advisable to increase the size of the dose but the frequency may be increased. The patient should be carefully watched for symptoms of antipyrin poisoning. The dose of the bromides is, roughly, one grain for each year of age, every three or four hours. It makes but little difference what form of bromide is used. The sodium salt is, however, less likely than the other to disturb the digestion. Antipyrin and the bromides may be combined. It is usually advisable to vary the drugs used, because after a week or so they are likely to lose their efficiency.

It is often advisable to use a sedative at night, although no drugs are necessary during the day. The bromides are sometimes sufficient. In general, however, it is necessary to use some preparation of opium. The best and only safe preparation to use in babies and young children

is paregoric. It is practically impossible to do any harm with this preparation, unless it is used absolutely recklessly. The dose of paregoric is from three to ten drops for a baby and from ten drops to one half a teaspoonful for a child, the dose varying, of course, according to the age. A teaspoonful of paregoric contains about one quarter of a grain of powdered opium. For older children codeine or the phosphate or sulphate of codeine, in doses of from one sixteenth of a grain to one half a grain, may be used according to the age and individual susceptibility of the child. Whenever codeine is used a small dose should be given first, because so many children are susceptible to opium. If that is not sufficient, it may be increased later.

Vaccines.—There is much difference of opinion as to whether or not vaccines are useful in whooping-cough. It seems reasonable to believe, however, that they have a certain amount of prophylactic value and that at times they may do good in the early stages. There is also considerable evidence to show that polyvalent stock vaccines are more valuable than monovalent vaccines. The use of vaccines of the Bordet-Gengou bacilli combined with those of other organisms is irrational. The other organisms cannot possibly have any effect on the whooping-cough, and they can have no more effect on the complicating bronchitis and bronchopneumonia than they do when those diseases are due to other causes. There is also much difference of opinion both as to the dosage of the vaccines and as to the intervals between the doses. When used for prophylaxis five hundred millions may be given at the first dose, to be followed at three day intervals by doses of one billion and of two billions. When used for treatment of the disease the first injection should be of five hundred millions. An injection should be given every second day, the dose being doubled at each injection. If definite improvement is not noticed after five doses, it is useless to continue. Whether the vaccines do any good or not, it is certain that they do no harm.

Roentgen Ray.—Dr. H. I. Bowditch, of Boston, has recently recommended the Roentgen ray in the treatment of whooping-cough and claims improvement from its use. It is thought that it reduces the enlargement of the tracheobronchial lymph nodes and that, in addition, it may have a direct action on the bacteria and on the hematopoietic system in general. The apparatus used in the treatment consists of an ordinary high tension transformer, with a rectifying device, universal Coolidge tube and a treatment stand, with cones of such size and shape as would include the entire chest at a distance of 28 inches. The thyroid gland is protected by sheet lead. Three treatments are given on alternate days. A second course of treatments is given after an interval of seven days, if necessary. The ordinary dosage for a child of from five to ten years is five minutes' exposure with the tube set at 4 ma., a 1 mm. aluminum filter and a 6 to 7 inch back up spark at a distance of 28 inches being employed. Young infants are given three minutes' exposure instead of five. The first treatment is given over the front and the second over the back of the chest, the remaining treatments being alternated.

Various other methods of treatment are sometimes useful. A paroxysm can sometimes be prevented or shortened by grasping the child's lower jaw from behind and pushing it upward and forward. When the paroxysms are so frequent and severe that they prevent sleep and the taking of food, intubation is sometimes very useful. Considerable relief is often afforded and the vomiting diminished by a properly applied abdominal belt.

Complications.—The treatment of bronchitis and bronchopneumonia complicating whooping-cough is the same as when they are due to other causes. When there is ulceration of the frenum of the tongue it is sometimes useful to put a piece of plaster over the sharp edges of the lower incisor teeth. Healing is often hastened by the application of a stick of nitrate of silver.

DIPHTHERIA

Diphtheria is an acute, contagious disease due to the Klebs-Loeffler bacillus. It is primarily a local infection, with constitutional symptoms from the absorption of toxins. When the Klebs-Loeffler bacilli enter the circulation they cause no local lesions. There is usually a concurrent infection with the pus organisms. These cause the secondary inflammations and, by their toxins, increase the constitutional symptoms.

Etiology.—There can be no true diphtheria without the presence of the Klebs-Loeffler bacillus. The presence of the Klebs-Loeffler bacillus without lesions or symptoms does not, however, constitute the disease diphtheria. Persons harboring the Klebs-Loeffler bacillus without lesions or symptoms are, however, dangerous to the community. It is true that in such cases the Klebs-Loeffler bacilli may not be virulent. They should, however, be considered to be virulent until they are proved not to be. Pseudodiphtheria bacilli are of theoretical, rather than of practical, importance to the practitioner, although they occasionally disturb the bacteriologist.

Drains, sewers and bad plumbing, no matter how bad the smells which come from them, do not and cannot cause diphtheria. Cats, dogs and cows may have diphtheria and transmit it to human beings. The Klebs-Loeffler bacillus thrives in milk. Milk contaminated with diphtheria bacilli is occasionally the cause of an epidemic of diphtheria. The diphtheria bacillus cannot penetrate the healthy mucous membrane. A lesion of the mucous membrane is necessary before infection can take place. Infection is, therefore, less likely if the mouth, throat, nose and teeth are in good condition. Diphtheria may occur at any age. It is rare in early infancy, most common between two and five years, and uncommon in adults. The rarity in early infancy is due to the presence of antitoxin, transmitted from the mother through the placenta. One attack of diphtheria does not confer any immunity against further attacks.

The diphtheria bacilli are contained in the secretions of the nose and throat. The Klebs-Loeffler bacillus is easily destroyed. It is short-lived, unless conditions are very favorable for it. Infection does not occur through the air, but is almost always from mouth to mouth, or hand to mouth. Indirect contagion is unusual.

Incubation.—The duration of the period of incubation is doubtful. It is probably usually between two and five days. It may be as short as twenty-four hours, or even less. On the other hand, diphtheria bacilli may be present in the throat for a long time without causing infection. This does not occur until there is a lesion of the mucous membrane from some other cause.

Pathology.—The Klebs-Loeffler bacillus may cause a great variety of local lesions. The earliest lesion is a superficial loss of substance of the mucous membrane, with a moderate amount of reddening. This is followed in a few hours by the formation of a very thin, pearly-white membrane. This becomes thicker and, for a time, whiter. In a short time, however, it becomes a dirty white, or yellow, or even yellowish green. It is adherent to the underlying structures and when pulled off

leaves a bleeding surface. There is, of course, swelling and reddening of the rest of the throat. The color is a rather dark red, quite different from the bright red of scarlet fever. These lesions are due to the action of the Klebs-Loeffler bacilli and their toxins. They are the result of a combination of fibrinous exudation and coagulation necrosis. Other organisms may cause the same lesions. When, however, the lesions look like diphtheria, they should be treated as if they were, until they are proved not to be. In other instances there may be no false membrane formed, the only lesion being reddening of the mucous membrane with some edema. In still other instances, the membrane may be limited to small spots at the opening of the crypts of the tonsils. The membrane usually forms first on the uvula or tonsils, but may start anywhere in the fauces or pharynx. It may not extend much or, on the other hand, may extend upward into the nasopharynx and nose or downward into the larynx and thence down the trachea and into the bronchi. In rare instances it may extend through the Eustachian tubes to the ears. It almost never extends upwards to the eyes, and seldom extends forward into the mouth. Membrane is more often present on the lips than in the mouth.

In primary nasal diphtheria the membrane is usually thin. It may be on either the turbinates or the septum. There is almost never extension from the nose backward. In primary laryngeal diphtheria the membrane is seldom very thick, but there is a large amount of infiltration of the tissues. It is not as firmly adherent as in the pharynx and fauces and hence may be detached and coughed up. Its attachments in the trachea and bronchi are usually quite loose.

When the lesions are in the fauces, there is usually some enlargement of the cervical lymph nodes, most marked at the angles of the jaws. When the nasopharynx is involved, there is almost always very marked enlargement of the cervical lymph nodes with considerable edema about them. I have seen this so marked that parents coming to the hospital did not recognize their own children. There is rarely any enlargement of the cervical lymph nodes in primary nasal diphtheria, and never any in primary laryngeal diphtheria.

Bronchopneumonia is a common complication of diphtheria, especially of the laryngeal type. The diphtheria bacilli play little, if any, part in its causation. It is due to other organisms. Degenerative changes are present in all the parenchymatous organs. They are caused by the toxins of the Klebs-Loeffler bacillus and the associated organisms. Acute nephritis, an uncommon complication, is caused by the associated organisms and their toxins. Degenerative changes in the nerves, which are not uncommon, are caused by the toxins of the Klebs-Loeffler bacillus.

Symptomatology and Course.—No one who has not seen diphtheria before the days of antitoxin really knows anything about either the symptomatology or the course of the disease, they have been so modified by the use of antitoxin. I shall try to describe it as it used to be before there was any specific treatment for it. The symptomatology and course depended, naturally, in the individual case, on the virulence of the infecting organism, the local resistance and the general resistance. These three factors modified the picture. In the light of our present knowledge, both the local and general resistance depend on the amount of antitoxin present in the blood.

There is nothing characteristic about the onset of diphtheria. It comes on like any other sore throat. It is possibly true that, in general, the onset of diphtheria is somewhat less acute than that of tonsilitis.

There are so many exceptions, however, on both sides, that nothing can be told as to the nature of the lesion in the throat from the character of the onset.

There is nothing characteristic about the temperature in diphtheria. It may rise quickly and be continuously high; it may rise slowly and be only moderate. Sometimes it is regular, sometimes irregular. In some of the worst cases there may be scarcely any fever. Although the temperature in tonsilitis, as a rule, goes up quickly and is high, nevertheless it is not always so. There are so many variations in the temperature in both diseases that no reliance whatever can be placed on the temperature curve in the differential diagnosis between diphtheria and other inflammations of the throat.

In a typical case of *diphtheria of moderate severity*, not treated with antitoxin, the membrane gradually spreads from its point of origin over a part or the whole of the tonsils, the whole of the uvula and on to a portion of the soft palate. The throat is a dusky red and there is moderate swelling of the mucous membrane. The membrane is dirty white or yellowish, sometimes green. The odor of the breath is characteristic. There is moderate enlargement of the cervical lymph nodes. The temperature is not very high, 101° F. to 102° F. The child is evidently not very severely ill. After from three to five days the membrane ceases to extend. It gradually begins to diminish in thickness, especially at the edges, often rolls up at the edges and finally, in from four or five days to a week, disappears entirely. While this is going on the temperature gradually works down to normal, the glands diminish in size, and the general condition improves.

The typical *severe nasopharyngeal* or *septic* case begins in the same way, but the membrane extends rapidly over the tonsils and soft palate and then backward over the pharynx and up into the nose. There is a profuse, foul, purulent, nasal discharge and considerable interference with nasal respiration. The foul odor of the breath is very marked. Great enlargement of the cervical lymph nodes quickly develops. There is marked edema of the face and eyelids. Pallor is marked, and prostration becomes extreme. The child shows all the evidences of severe septic infection. Death almost invariably occurs within a week. In the few days before death hemorrhages from the nose and throat are not uncommon. Hemorrhages may occur into the skin. There is marked cardiac weakness and usually there are evidences of marked degenerative changes in the kidneys.

In the typical *secondary laryngeal* case of diphtheria, the membrane extends downward into the larynx and symptoms of laryngeal obstruction develop. In such cases the membrane in the throat is seldom very extensive and there is no additional enlargement of the cervical lymph nodes. After poultices and steaming have been tried, an intubation is done. This usually relieves the obstruction temporarily, fortunately sometimes permanently. As a rule, however, the membrane continues to extend. When it gets into the trachea below the point which the tube reaches, the symptoms of obstruction recur and a tracheotomy is done. This affords temporary relief and occasionally cures. In most instances, however, the membrane continues to extend downward into the bronchi and the child eventually dies of bronchopneumonia, secondary to the interference with the clearing of the bronchi.

In *primary laryngeal diphtheria* the first symptoms are usually a croupy cough and a little huskiness of the voice. In typical cases slight

interference with respiration develops. The interference is entirely inspiratory. The interference with respiration gradually increases, so that inspiration becomes noisy. There is at first retraction of the supraclavicular and lower intercostal spaces. As the obstruction increases, inspiration becomes more and more difficult, there is marked retraction of the supraclavicular, suprasternal and lower intercostal spaces, with, toward the end, retraction of the epigastrium and the lower chest. The child is unable to lie down, and becomes more and more cyanotic. If it can speak at all, it is only in a whisper. Death is almost certain, in agony, unless intubation is done. If it is, the course is usually that described in speaking of secondary laryngeal diphtheria.

The diagnosis between primary laryngeal diphtheria and acute catarrhal laryngitis is sometimes very difficult. The main differences are that in laryngeal diphtheria the symptoms of stenosis begin slowly and steadily increase in severity without remissions or intermissions while in catarrhal laryngitis the child usually has a cold, the obstruction develops suddenly, usually in the early evening, and becomes very marked at once. Under treatment the symptoms of catarrhal laryngitis quickly diminish, but recur again on the following night. Occasionally they are persistent, but, if so, always vary greatly in severity from time to time. If there is any question as to whether the disease is laryngeal diphtheria or catarrhal laryngitis, it should always be treated as if it was diphtheria. It must be remembered, moreover, that a negative culture for diphtheria bacilli does not rule out primary laryngeal diphtheria. No reliance should be placed on a negative culture.

Primary nasal diphtheria is far more common in infancy than in childhood. There are seldom any constitutional symptoms. There are usually a little nasal obstruction and snuffles. The nasal discharge is usually somewhat purulent, occasionally bloody, and irritates the upper lip. The condition is quite likely to be overlooked or mistaken for a common cold or syphilitic rhinitis. The disease almost always remains localized in the nose. The great danger is of infection of other people. In most instances the membrane can be seen, if it is looked for. In some instances, however, it is too far back to be seen easily. Cultures should always be made, therefore, whenever there is a chronic nasal discharge, especially if the patient is a baby.

Blood.—There is almost always a considerable polynuclear leukocytosis in diphtheria. This is most marked in the severe septic cases. It may be lacking in very mild cases and in the severest cases, when the child is overwhelmed by the toxemia. Secondary anemia develops very rapidly and in the severe cases is quite marked.

Urine.—The urine, except in the mildest cases, shows the evidences of acute degenerative nephritis.

Complications and Sequelae.—Bronchopneumonia is a common complication of diphtheria, especially in the laryngeal and operative cases. There is nothing characteristic about the physical signs. It makes the prognosis decidedly worse.

An interstitial emphysema occasionally develops as the result of the rupture of an alveolus in laryngeal diphtheria. This is especially likely to occur in operative cases. The air may work its way up through the mediastinum and produce subcutaneous emphysema. This shows usually first as a swelling above or at the sides of the sternum. In some instances it is so marked that it spreads over the chest and up over the neck.

Cardiac weakness is a very common complication, being present in almost all of the severe and septic cases. In the vast majority of instances it is due to myocarditis. In a certain number, however, it is probably due to pneumogastric neuritis, and in others is increased by vasomotor paralysis. I have always found it very difficult to determine, in a given case, how much of the cardiac weakness was due to degeneration of the myocardium and how much to pneumogastric neuritis, unless there were other very definite signs of pneumogastric neuritis. The points in favor of myocarditis are enlargement of the heart and marked weakening of the heart sounds. The rate of the heart is of no assistance, because it may be either slower or quicker when the pneumogastric is involved, and more or less rapid according to whether or not the sinoauricular node or the bundle of Hiss are involved, if the myocardium is diseased. Fortunately, the cause of the cardiac weakness makes no difference in the treatment.

Acute nephritis is a not uncommon complication of very severe and septic diphtheria. There is nothing very characteristic about it. In a general way, however, the amount of urine is not as much diminished, there are fewer red cells and a larger amount of albumin than in acute nephritis due to other causes.

Otitis media is a very common complication. It is almost never caused by the Klebs-Loeffler bacilli, but is due to a secondary infection. Pain is not common in otitis secondary to diphtheria.

Chronic nasopharyngeal catarrh is a common sequela of diphtheria. Anemia develops rapidly during the disease and usually persists for a considerable time. Cardiac weakness, usually due to myocarditis, often lasts for many weeks. Pneumogastric neuritis, or, at any rate, a set of symptoms which are commonly attributed to pneumogastric neuritis, may develop either toward the end of the acute stage or at the beginning of convalescence. The earliest symptoms are usually refusal to take food and vomiting. The vomiting is at first only occasional, but finally everything taken is lost. Associated with the vomiting is pain in the epigastrium, which usually is independent of the vomiting. The pulse is weak and often irregular. It may be either increased or diminished in rate, but is usually diminished. The face is pale or slightly cyanotic, and there is moderate dyspnea. The mind is clear. These symptoms usually increase steadily in severity and end in death, sometimes within twenty-four hours, but usually after several days. In other instances the symptoms are less severe, but of the same general type. They gradually diminish, and finally cease after three or four weeks.

Peripheral neuritis is also a not uncommon complication. It has not diminished in frequency since the introduction of antitoxin, because, although antitoxin, when given early, prevents its development, it saves a great many children to have neuritis who would have died without developing it, if they had not had antitoxin. Peripheral neuritis develops usually in from one to five weeks after the acute stage is over. The symptomatology of myocarditis and diphtheritic neuritis is taken up elsewhere.

Diagnosis.—The clinical diagnosis of diphtheria depends almost entirely upon the appearances in the throat and the symptomatology. These have already been described, as has also the differential diagnosis between diphtheria and other conditions, except Vincent's angina, with which it may be confused. Vincent's angina is usually unilateral, while both tonsils are ordinarily involved in diphtheria and there is more swell-

ing and inflammation of the tonsils as a whole. The membrane in diphtheria is grayer, smoother and the edges better defined, and, when it is taken off, the ulceration is not as deep. A positive diagnosis is easily made by examining a smear from the deeper parts of the membrane. Vincent's bacillus is easily recognizable in Vincent's angina.

It must never be forgotten that any inflammatory process in the throat, nose or larynx, however atypical, may be due to the Klebs-Loeffler bacillus and be, therefore, diphtheria. The only safe method to pursue, therefore, is to take cultures from the nose and throat in every inflammatory condition of these organs. If the local conditions suggest diphtheria, antitoxin should always be given without waiting to take cultures. An unnecessary dose of antitoxin can do no harm, while delay, if the disease is diphtheria, may mean the loss of the child's life. It must also be remembered that bacteriologists, like physicians, are only human, and that, if their reports do not coincide with the clinical appearances, they are just as likely to be wrong as the physician. If the clinical appearances suggest diphtheria, antitoxin should be given in spite of negative reports.

Great care must be exercised in taking the cultures. The child must be made to gag, so that the throat can be seen properly, and the swab rubbed directly on the suspicious area. In taking cultures from the nose the swab must be introduced deeply. In taking cultures when there is a question of laryngeal diphtheria, the swab must be introduced far into the back of the throat and, if possible, down to the larynx. Mistakes are certain to occur, if the swab is only put into the mouth or into the nostrils. It is also most important to use fresh culture media. Diphtheria bacilli will not grow on old and dry media.

In many cases an immediate diagnosis can be made from smears without waiting for cultures. In almost all such cases, however, the diagnosis can be made almost as well from the clinical appearances.

Prognosis.—Before the days of antitoxin from 45 to 50% of all cases of diphtheria died, and from 70 to 75% of the operative cases. The mortality has been very materially reduced since the introduction of antitoxin, being now well under 10% for all cases and under 25% in the operative cases. The number of operative cases has, moreover, been very materially reduced. No one can appreciate what a dreadful disease diphtheria is, unless they have seen it before the days of antitoxin. I have seen every child admitted to a ward of forty-five beds during a month die. I have seen also five children which had been operated upon die in one night in that same ward. Every child in a diphtheria ward was seriously ill, pale, septic and uninterested. The children in a diphtheria ward now, almost without exception, do not appear sick, have a good color, and are sitting up playing, talking and laughing. A diphtheria ward now is like a convalescent ward rather than a death-house as it used to be.

The prognosis with antitoxin depends very materially on the stage of the disease in which it is given. When it is given on the first day, the mortality is less than 1%. The mortality increases, the later the antitoxin is first given. The mortality is not as low as it should be, because the laity delay in calling a physician, because physicians do not always appreciate that any inflammation of the throat may be diphtheria, because physicians depend too much on the bacteriologic diagnosis and too little on the clinical manifestations, and because of the presence in the community of various deluded classes—such as the Christian Scientists, and antivivisectionists. When the Schick test and toxin

antitoxin immunization are universally employed, as they should be, the morbidity and, in consequence, the mortality can be reduced to practically nothing.

Bad signs in diphtheria are pallor, prostration, hemorrhages, vomiting, fetor, purpura, albuminuria, an irregular, rapid or slow pulse, and weak heart sounds. None of these signs will develop if diphtheria is recognized early and antitoxin given in sufficient doses.

Cardiac weakness may persist for a long time and the danger of sudden death is not passed until the heart is normal. The prognosis of the peripheral paralysis which follows diphtheria is good, except when the pneumogastric is involved.

Treatment.—*Prophylaxis.*—By the use of the Schick test and toxin antitoxin injections everyone can be protected against diphtheria. Such being the case, everyone should be protected against diphtheria. Not everyone will be so protected, however, because of the unintelligent and bigoted opposition of a certain proportion of the community to all health measures.

Schick Test.—An antitoxic content of $\frac{1}{30}$ unit per c.cm. of blood protects the individual against infection with diphtheria. It is also sufficient to neutralize and prevent the inflammatory action of a small amount of toxin injected into the skin. The Schick test is based on this phenomenon. A positive reaction shows that the individual is susceptible and a negative that he is immune to infection. The test is performed by injecting intracutaneously $\frac{1}{50}$ of the minimum lethal dose of toxin for a guinea-pig weighing 250 grams. The toxin is supplied for clinical use in a capillary tube and before being used is mixed with a fixed amount of salt solution. One tenth of a c.cm. of this mixture is the dose to be injected, and contains $\frac{1}{50}$ of a minimum lethal dose. The solution must be used within a few hours after it is made, as it quickly loses its power. The injection must be made into the skin, not below the skin. If the needle is properly introduced, with the opening upward, the opening can be seen through the skin. The injection makes a small, slightly raised, white wheal, which disappears in about half an hour. The injection causes almost no pain and no constitutional symptoms. The skin should, of course, be cleaned with alcohol or ether before the injection. No dressing is necessary.

If the individual has $\frac{1}{30}$ unit of antitoxin per c.cm. of blood, there is no reaction, showing that he is immune. If he does not have this amount of antitoxin, an area of redness and slight infiltration, varying in diameter from one to two cm. begins to appear in from twenty-four to thirty-six hours. The reaction increases gradually in severity, reaching its maximum on the fourth day. It then fades very slowly and leaves a scaly, brownish, pigmented area which persists for many weeks. This reaction shows that the individual is not immune and may contract diphtheria, if exposed to it.

A pseudoreaction occurs in some individuals, usually adults, as the result of hypersensitiveness to the protein of the diphtheria bacillus present in the toxin. This reaction appears in from twelve to eighteen hours, and fades out in three or four days. It leaves an irregular area of brownish pigmentation, which does not scale. The height of the pseudoreaction is reached at about the time that the real reaction appears. The pseudoreaction has disappeared by the time that the positive reaction reaches its maximum. A control should, therefore, be done on the other arm with an injection of $\frac{1}{10}$ of a c.cm. of a similar dilution of toxin, which

has been heated to 75° C. for five minutes. Such heating destroys the toxin, but does not affect the bacillary protein.

In some cases there is a combined reaction. In such cases, however, the reaction is more marked at the site of the Schick test than of the control, and at the end of three days the positive reaction will be quite distinct, while the psuedoreaction has nearly disappeared.

Most babies under three months of age are not susceptible to diphtheria. The susceptibility then rapidly increases, reaching its maximum at about two years. It then gradually diminishes up to twenty years, after which it is somewhat less than in infancy.

It is evident that all individuals who give a positive reaction to the Schick test should be immunized against diphtheria. The only question is as to whether it is necessary to bother to do a Schick test on young children, when such a large proportion of them are susceptible to diphtheria. The recommendations of the Massachusetts State Board of Health are practical and satisfactory. They are essentially as follows:

"Children under six months of age should have a Schick test performed and if they are negative they should be retested between six months and one year of age. If they give a positive reaction they should be immunized with diphtheria toxin-antitoxin mixture. All children between the ages of six months and six years should be immunized with three injections one week apart, without having the Schick test performed on them. All children between six years and eighteen years of age should have the Schick test performed and if they give a positive reaction should be immunized. All individuals above eighteen years of age who are exposed to diphtheria or may come in contact with it, should have the Schick test performed and be immunized if the test is positive. If children above six years of age or adults show a marked combined reaction, the toxin-antitoxin mixture should be given in divided doses, beginning with 0.1 c.cm., then 0.2, 0.5, 1, and 1 c.cm. at weekly intervals."

Toxin-antitoxin Immunization.—In the toxin-antitoxin mixture the toxin is partially neutralized by antitoxin, so that it is harmless. Experience has shown that the toxin in the mixture stimulates the organism to produce antitoxin and that the organism produces enough antitoxin to change the reaction to the Schick test from positive to negative and to protect the individual against diphtheria. The immunity produced is active, not passive. It requires from eight to twelve weeks for the immunity to develop. In order to be certain that immunity has been produced, the Schick test should be repeated in six months and, if positive, the toxin-antitoxin injections should be repeated and another test made after six months. At least 90% are immunized at the first attempt. Toxin-antitoxin immunization being a recent procedure, the duration of the immunity produced is unknown. It is known, however, that it lasts at least seven years. It is probable that it lasts for life.

Although larger amounts of toxin were used at first in the mixture without doing any harm, it has been found that $\frac{1}{10}$ L+ dose is sufficient to produce immunity. This is, therefore, the amount of toxin now used at a dose. The toxin-antitoxin mixture is put up in ampules, each containing 1 c.cm. Three injections are given subcutaneously at weekly intervals. The skin should be painted with iodine before the injection is given. It is wise to put a sterile dressing over the puncture for a few hours. In the vast majority of cases there is no reaction, either local or general, from these injections. Occasionally there may be a little redness about the site of injection and sometimes there may be a very

slight rise in temperature or malaise the next day. The toxin-antitoxin mixture should be kept in a cool place, as it deteriorates if kept in a warm place. It must not, however, be frozen. Freezing apparently causes some change in the mixture which causes a marked local reaction.

Isolation.—All children with diphtheria should be isolated until the Klebs-Loeffler bacilli have disappeared from the nose and throat, as proved by three consecutive negative cultures. All those exposed should be given immunizing doses of antitoxin, unless they have been shown previously to be immune by a negative Schick test. 500 units should be given to an infant, 1000 to a young child, and 1500 to an older child. The immunity conferred is simply passive and does not last over three weeks, at the most. The antitoxin may be given subcutaneously. Other children in the family should be kept out of school until the patient is discharged from quarantine. Theoretically, they should not be allowed to return to school until it has been shown that they do not harbor diphtheria bacilli in the nose and throat. Practically, this precaution is hardly feasible. Adults of the household should not be allowed to go to work where there are children. Theoretically, they should not be allowed to leave the house, unless it is proved that they are not carriers. Practically, such a regulation is not reasonable. By far the best solution of the question of quarantine is to send the child at once to the diphtheria ward of a hospital.

The Klebs-Loeffler bacilli are present in the secretions of the nose and throat. These secretions should, therefore, be destroyed and the articles contaminated by them disinfected. Those who take care of children with diphtheria must be extremely careful not to put their hands in their mouths without washing them.

Hygiene of the Mouth and Throat.—Proper care of the teeth, gums, mouth and throat, by keeping the mucous membranes in a normal condition, diminishes the chances of infection. The removal of adenoids and diseased tonsils not only tends to prevent infection, but also diminishes the severity of the disease, if infection occurs. Spraying of the nose and throat does little to prevent infection, and, if strong and irritating solutions are used, probably favors it by irritating the mucous membrane.

Antitoxin.—Diphtheria antitoxin has a specific action in diphtheria. It neutralizes the toxin produced by the diphtheria bacilli both locally and throughout the body. It also inhibits the activity of the bacilli. It has no neutralizing action on the toxins produced by the associated organisms. It cannot undo the injury done to the tissues by the toxin which has already attacked them. It can only prevent further injury. Unfortunately, the action of diphtheria toxin on the tissues is very rapid and irreparable injury may be done in a short time. The antitoxin must, therefore, be given early and in sufficient doses, in order to do the good that it is capable of doing. The dangers of delay and the reasons why delays so often occur have been discussed under prognosis. Antitoxin should be given at once, whenever the clinical symptoms and appearances suggest the possibility of diphtheria, without waiting for a bacteriologic diagnosis. Furthermore, it should be given, even if the bacteriologic examination fails to show Klebs-Loeffler bacilli.

Antitoxin should be given in such a way that it reaches the tissues quickly. It is not sufficient to neutralize the toxin in the blood. It must also reach that in the tissues. Antitoxin given intravenously passes into the tissue fluids from the blood ten times as fast as when given under

the skin and four times as fast as when given into a muscle. When injected under the skin the major portion is not absorbed into the blood stream for twenty-four hours, and when given intramuscularly not for twelve hours. The local swelling after the injection disappears rapidly because of the absorption of the water, but the colloid portions of the serum, in which is the antitoxin, are absorbed slowly. Antitoxin should never, therefore, be given subcutaneously in the treatment of diphtheria. In early and mild cases it may be given intramuscularly. In late and severe cases it may be given either intramuscularly or intravenously, but the intravenous method is far more reliable. In malignant cases it must be given intravenously. When antitoxin is given intravenously, there is a possibility of sudden death from anaphylactic shock from the horse serum in which it is contained. This danger is, however, extremely slight and much less than in pneumonia, in which large amounts of horse serum are given intravenously. It is hardly necessary to test every child as to sensitization to horse serum before giving antitoxin in diphtheria. If, however, there is a history of asthma, eczema or urticaria, or horse serum has been given previously, the child should be tested and, if sensitive, desensitized. Sensitiveness to horse serum may be tested by injecting into (not under) the skin 0.02 c.cm. of sterile normal or immune horse serum diluted 1:10 or 1:100 with sterile physiologic salt solution. A control injection of an equal amount of sterile physiologic salt solution should be made at the same time. In sensitive children an urticarial wheal, surrounded by a zone of erythema, appears in a few minutes. The zone of erythema gradually increases in size for about an hour, sometimes becoming as large as a fifty cent piece. It then gradually fades out. No such reaction occurs at the site of the control injection. It is most important that the injection is given into the skin, not under it, because no reaction will occur even in sensitive children, if it is given subcutaneously. Although a negative intradermal test does not absolutely prove that a child is insensitive to horse serum, it is safe, for practical purposes, to assume that it is and to act accordingly. If the test shows that it is sensitive, it should be desensitized before the serum is given. The Board of Health of Massachusetts advises the following method of desensitization: 0.025 c.cm. (0.25 c.cm. of a 1:10 dilution) is injected subcutaneously. The dose is doubled every half hour until 1 c.cm. is given. 0.1 c.cm. is then injected intravenously. The dose is doubled every half hour until the desired dose can be given without untoward reaction.

The dose of antitoxin to be given depends on the age and weight of the patient and the severity of the case. A sufficient amount should be given at the first injection to obviate the necessity of its repetition. If further doses are necessary, it means that an insufficient amount was given the first time. One large dose is far more effective than the same amount given in divided doses, because, when it is all given at once, the maximum effect is obtained quickly, while, when it is given in divided doses, the maximum is reached slowly and is not as great. Nothing is gained as to keeping antitoxin in the blood by divided doses, because it remains in the blood for several days after a single dose. It is never a mistake to give more antitoxin than is necessary. Irreparable harm may be done, if the dose is too small.

The dosage and method of administration advised by Dr. W. H. Park of New York, and recommended by the Massachusetts State Board of Health, is as follows:

AMOUNT OF ANTITOXIN IN THE TREATMENT OF A CASE OF DIPHTHERIA

	Mild cases units	Early moderate units	Late moderate and early severe units*	Severe and malignant units*
Infants, 10 to 30 lbs. in weight under 2 yrs.....	2,000 to 3,000	3,000 to 5,000	5,000 to 10,000	7,500 to 10,000
Children, 30 to 90 lbs. in weight under 15 yrs....	3,000 to 4,000	4,000 to 10,000	10,000 to 15,000	10,000 to 20,000
Adults, 90 lbs. and over in weight.....	3,000 to 5,000	5,000 to 10,000	10,000 to 20,000	20,000 to 50,000
Method of administration advised.....	Intramuscular	Intramuscular	Intravenous	Intravenous

* When given intramuscularly use the larger amounts above indicated.

The skin at the point of injection should be washed with soap and water, followed with alcohol. When it is dry, it should be painted with tincture of iodine. Intramuscular injections should be given deeply, preferably into the buttocks. Intravenous injections can usually be given into one of the veins at the elbow in children. In infants it may be necessary to use one of the veins of the scalp or even the longitudinal sinus. This route should not be used, however, unless absolutely necessary. It goes without saying that the syringe should be sterile and that every precaution should be taken against infection, as in other operations. When antitoxin is given intravenously, it should be highly potent, clear and without sediment. It must be warmed to body temperature and not injected more rapidly than 1 c.cm. per minute. The point of puncture should be protected by a sterile dressing for several hours after the injection.

Although the chance of the development of any severe anaphylactic symptoms is very small, it is advisable to be ready to inject 1 c.cm. (15 minims) of the 1:1000 solution of suprarenal extract at once, if they arise. There is practically no danger of anaphylactic shock in giving repeated injections of antitoxin in diphtheria to those who have not suffered from the first. If the second injection is given intravenously after a considerable period, it should be given very slowly.

Improvement in both the constitutional symptoms and local manifestations is usually evident within twelve hours, at the most within twenty-four hours. If there is no improvement in twenty-four hours, the dose of antitoxin was insufficient or treatment was put off too long. If there is no improvement within twenty-four hours, more antitoxin should be given. If a sufficient dose is given in the first place, however, a second dose is not required. The general effect of the antitoxin is shown by a fall in the temperature and improvement in the general condition. The local effect is shown by the cessation of the spreading of the membrane and the beginning of the changes in the throat which occur when recovery is spontaneous. The improvement in the local condition is very rapid, changes occurring in hours which took days when antitoxin was not available. The improvement is especially striking when there is laryngeal obstruction. The respiration often becomes quiet and easy within a few hours in cases which were apparently heading straight for intubation. Even when the antitoxin is given too late to obviate the need for intubation, the tube can usually be removed in a much shorter time than in the past.

Serum Sickness.—The anaphylactic phenomena to which this term is applied are due to the horse serum in which the antitoxin is contained, not to the antitoxin itself. Some children show a reaction, whatever

horse serum is used. Others react to that from one horse and not to that from another, while the serum from some horses causes a reaction in almost everyone. These reactions are much less common than they used to be, because the serum is now far more refined and concentrated than in the past. They usually develop during the second week after the injection, but may come either earlier or later. Severe local reactions at the seat of injection sometimes appear within a few days. There may be marked swelling, redness and pain. I have never known an abscess to develop or sloughing to occur, however, even when the condition appeared quite serious. An icebag usually gives the most comfort. There is not infrequently enlargement and tenderness of the lymph nodes, which may be either of those nearest the seat of injection or general.

The commonest manifestation is a rash, which ordinarily appears first at the seat of injection. It is usually a blotchy erythema, but is not infrequently urticarial in character. It is almost always accompanied by itching, which may be intense. In rare instances, the eruption may be general and scarlatiniform. The diagnosis from scarlet fever is easy, however, because there is the history of the injection of the antitoxin, the tongue is not reddened, and there is no eruption in the throat. The eruption is usually accompanied by some fever, which may be high, sometimes as high as 104° F. The eruption usually lasts for three or four days, but may last a week or more. It often appears in crops. Occasionally it is hemorrhagic.

Edema of the face is not uncommon. It sometimes occurs in the fauces and may involve the larynx, causing obstruction. As there is sometimes considerable abdominal pain and vomiting, it is presumable that edema, or some condition similar to urticaria, may develop in the digestive tract. In rare instances there may be temporary suppression of the urine. In other cases there is marked pain and tenderness in the joints, which may be red and swollen. The temperature is usually high in these cases.

None of these manifestations are serious, except the edema of the larynx. They are very uncomfortable, however, and sometimes harder to bear than the original attack of diphtheria. They may follow immunizing injections as well as those used in treatment.

I have seen but little relief from calcium lactate, which was so highly recommended at one time. Injections of from five to ten minims, according to age, of the 1:1000 solution of adrenalin chloride often, however, afford much relief. The usual remedies for itching may be tried. I have found bathing with alcohol and water, equal parts, as satisfactory as anything. The various dusting powders may also be used. Stearate of zinc is the best of these, because it sticks best.

Local Treatment.—The most important thing to be remembered in the local treatment of diphtheria is that no harm must be done by it. There are no local applications which have any effect on the progress of the disease or that limit the spread of the membrane. It is possible, of course, to remove the membrane. This does no good, however, because it immediately reforms and is usually more extensive because of the injury done to the tissues in removing it. Irrigation of the throat with warm or hot physiologic salt solution usually makes the child more comfortable and by washing out the secretions may, perhaps, diminish to a certain extent toxic absorption. It may be done every few hours. All that is accomplished, however, is gross cleanliness. If the child objects to irrigation of the throat or is tired by it, it does more harm than good and should be stopped. In irrigating the throat the child should,

according to its condition, either sit up or lie on its side, with a basin under the chin or cheek to catch the fluid. The fluid should be allowed to run into the throat from a fountain syringe hung not more than two feet above the level of the throat. A hard rubber nozzle with a single opening in the end is most suitable. The child, of course, must hold its breath while the fluid is running in and out. The flow must, therefore, be interrupted frequently. If there is much secretion in the nose, the nose may be cleansed with salt solution introduced with a syringe, but with the use of very little force. It must always be remembered that washing out the nose predisposes, if force is used, to infection of the ears.

Hygiene and Food.—Children ill with diphtheria undoubtedly do better if they have an abundance of fresh air and sunlight. An old practitioner once told me, years before the introduction of antitoxin, that, if he had diphtheria, he should like to be put on top of a hill with a jug of whisky and left alone. There was considerable basis for his wish. He, in that way, would get fresh air and sunlight and nobody could do anything to him to hurt him. In the old days most of the treatment, which was largely local, unfortunately did hurt the patient. We can hardly agree with him at present, however, as to the advantage of the whisky. Nothing should be vaporized in the room. There is no kind of vapor which has any effect on diphtheria. It merely makes a more or less bad smell, and, as the windows are usually closed while it is used, interferes with the supply of fresh air.

It is also important to give children with diphtheria a liberal amount of food. It is often very difficult, however, to get them to take it. Milk and things made from milk should form the basis of the diet. No energy should be wasted in giving broths and soups, which contain practically no nourishment. The same applies to white of egg and, to a less extent, to orange juice. On account of the pain in swallowing, it is usually advisable to give small amounts often. Foods which are quite hot or cold are more easily taken than those which are warm.

Medication.—There are no drugs which have any effect on the course of diphtheria. It was thought years ago that alcohol had almost a specific action in diphtheria. I have seen enormous doses of it given, as much, even, as an ounce an hour to a child of four years. Strange to say, the children were not intoxicated by these enormous doses. When they recovered we used to think that they recovered because of the alcohol which they took. I am inclined to think now that they recovered in spite of it. Stimulation may be required in diphtheria as in other acute diseases. The stimulants to be used have been discussed in speaking of the treatment of pneumonia.

The importance of rest when there is myocardial involvement cannot be too strongly emphasized. Children with myocarditis should be kept absolutely quiet until the heart is normal. Sudden death may occur from the myocarditis of diphtheria during convalescence, even several weeks after the child is otherwise well. Myocarditis and its treatment are discussed under myocarditis.

There is no treatment which does any good in pneumogastric paralysis, unless it may be rest and care. Diphtheritic paralysis and its treatment is discussed among diseases of the nervous system.

Laryngeal Obstruction.—When antitoxin is given early, laryngeal obstruction does not develop. Even when it is given later than it should be, the obstruction is seldom as marked as it is without it and is of much shorter duration.

The old procedures of steaming, fumigating with calomel and nauseating with ipecac occasionally afford relief when the obstruction is not too great. It is doubtful, however, whether it is advisable to use them, now that antitoxin is available. If there is much obstruction, it is probably wiser to intube the child at once. In former days there was much discussion as to whether intubation or tracheotomy was the preferable operation. Now, no one, I think considers tracheotomy until intubation has been tried and has failed to relieve the symptoms. Intubation should not be put off too long. It should not be delayed until the child is exhausted by its efforts at respiration. It is far better to intube too early than too late.

Intubation.—There are no tubes as good as the original O'Dwyer tubes. The hard rubber tubes are preferable to the metal. It is far wiser to intube too early than to wait too long. In general, the size tube recommended for the age should be used. In many instances, however, it is necessary to use the next size larger in order to have it retained. I have always introduced the tube with the child lying on its back. Others, however, always intube with the child sitting up. Either position is probably satisfactory, each operator preferring the one to which he is accustomed. The child should be tightly wrapped up so that it cannot struggle. A small, hard roll should be placed underneath the neck so that the head is moderately extended. The head and body must be held firmly by assistants. The operator stands on the right side of the child. The assistant who holds the head introduces the gag into the left side of the mouth, opening the mouth widely. The operator introduces his left forefinger into the mouth, pulls forward the epiglottis and feels with his finger the two arytenoids. The tube on the introductor, held in the right hand, is then put into the mouth and the end of the tube introduced into the larynx under the finger. If the angle at which the tube is held is right, it slides into the larynx without the slightest difficulty. If it is wrong, it will not go in. No one can tell anyone else what that angle is. It must be learned by experience. The usual mistake is to put the tube into the oesophagus. This will not happen if the tip of the left forefinger is on the arytenoids and the tube is introduced in front of them. The introducer is then removed, the finger still being kept on the tube, but the thread is left in. Relief is seldom immediate. The child always strangles for a few minutes and coughs up a lot of mucus and sometimes some membrane. If membrane has been pushed down before the tube, respiration is, of course, impossible. The tube must be pulled out at once. If respiration is not resumed, a tracheotomy must be done. Incidentally, no one should do an intubation without being prepared to do a tracheotomy at once, if necessary. After the breathing has quieted down the left forefinger should be again placed over the tube, the string cut and removed. Care must be taken not to attempt to pull the knot through the hole in the tube. If this is done, the tube will be pulled out. It seems to me that it is wiser to remove the string than to leave it in. It is true that if the tube becomes obstructed and the string is out, no one but an expert can get the tube out. On the other hand, if the string is left in and fastened to the face it interferes with eating, disturbs the child constantly, and, if it grabs the string and pulls the tube out, no one but an expert can put it back. It is a choice of evils. It seems to me the less is to take out the string.

A child that has been intubed should never be left alone. There should, moreover always be someone constantly at hand able to take out

and replace the tube. Practically, however, unless the child is in a hospital, it is impossible for a physician to be constantly at hand. Sometimes, if the tube becomes blocked, it can be shaken out by holding the child up by its legs, and sometimes the tube can be loosened by pressing upward on the trachea.

It is very hard to know just when to attempt the removal of the tube. When antitoxin has been given, it can usually be taken out on the third day, sometimes earlier. It is often much more difficult to take out the tube than to put it in. The child should be wrapped up and held in the same way as for intubation. The left forefinger should be introduced in the same way, the epiglottis pulled forward and the tip of the finger placed over the top of the tube. The point of the extubator is then slid into the tube, the jaws opened and the tube withdrawn. Everything should be at hand for an immediate reintubation, if the difficulty with respiration recurs, as it often does, at once. Great care must be taken not to get the child excited before or after the extubation. If the difficulty with respiration recurs and the tube has to be put back, another attempt at removal should be made in a couple of days. It is often advisable to give a small dose of bromide or of some opiate a short time before the removal of the tube. The tube is sometimes coughed out. Under such instances it is not infrequently swallowed. It does no harm, however, and is always passed in a few days.

Occasionally it is impossible to keep the tube out. The usual reasons are ulcerations of the larynx as the result of the introduction of too large a tube or of injury to the larynx when it was introduced and paralysis of the vocal cords, either from pressure or as the result of the diphtheria. Various tubes of different shapes, which bear in different places from the ordinary tubes, have been devised for the tretament of this condition. Unfortunately, it not infrequently is impossible to remove the tube in these cases. When this happens a tracheotomy must be done and the intubation tube removed. The further treatment can only be handled by a specialist. Occasionally the tracheotomy tube has to be worn throughout life.

In most instances children are able to eat as well with the tube in as with it out. Sometimes they take liquids more easily, sometimes soft solids. If there is difficulty in taking food, it can sometimes be taken easier, if the child lies on its back with the head bent backward. If necessary, it is possible to feed the child through a tube, which may be introduced either through the mouth or through the nose.

Tracheotomy.—Far be it from me to describe the operation of tracheotomy. It is not a difficult operation, if the child is held absolutely quiet and the cut is made in the median line. If the cut is not made in the median line, it is extremely difficult. The expert surgeon can probably do the operation better with a careful dissection. The man who is not an expert is less likely to get into trouble, if he makes but two cuts, one down to the trachea and the second through the trachea. If an anesthetic is used, chloroform is preferable to ether.

MUMPS

(EPIDEMIC PAROTITIS)

Mumps may occur at any age. It is rare in infancy and most common in children between five and fifteen years of age. It usually occurs in epidemics. Many people escape it entirely. It is almost never a serious

disease in childhood. Complications are unusual at this age. The essential lesion is an inflammation of the salivary glands. The parotids are the glands most often involved. Both the submaxillary and sublingual glands may be involved either independently or in conjunction with the parotids. A second attack in the same gland is most unusual at any age, and almost never occurs in childhood.

Etiology.—Mumps is undoubtedly due to some microorganism. This microorganism has, however, never been discovered. It probably belongs in the class of the filterable viruses. It is present in the secretions of the mouth. It may be present in them for one or two days before the appearance of the symptoms and at any rate as long as the swelling persists, perhaps longer. Nothing is known as to carriers. It is probably quickly destroyed outside the body. It may cause the same disease in cats. Contagion is probably almost always direct, but it is probable that it may be occasionally transferred by third persons and contaminated objects. The causative microorganisms probably enter the glands through the ducts, but this is not proved. They certainly leave the glands through the ducts.

Incubation.—The period of incubation is variable. The extreme limits are probably ten to twenty-five days. The usual period is from fifteen to twenty-one days.

Period of Contagion.—This is from one or two days before the appearance of symptoms to the disappearance of the swelling, and perhaps some days longer. It is wise to consider a child with mumps contagious for at least three weeks from the appearance of the swelling, or for one week after the disappearance of all symptoms.

Pathology.—All parts of the gland are involved. The parenchyma is apparently involved first and the interstitial tissues later. There is marked edema and cellular infiltration of these tissues, especially about the secretory ducts.

When the testicle is involved, the inflammatory process is limited to the body and the epididymis escapes. The interstitial tissues are chiefly affected. Atrophy of the organ with loss of function may result.

Symptomatology.—Mumps has always been, in my experience, a very mild disease. I have never seen a child seriously ill with it.

In most instances the first thing noted is pain in the region of the parotid gland. It is often referred to the ear and is increased by movements of the jaw. It is not influenced by taking sweet or sour substances into the mouth. The pain is followed in a few hours, or one or two days, by swelling of the gland. The swelling reaches its maximum on the third day, remains about the same for two or three days, and then gradually subsides. When the swelling is of the parotid gland it is located over the ramus of the lower jaw. It extends upward both in front and behind the tragus, pushing it upward and outward. It extends forward to the zygomatic arch and backward to the sterno-mastoid muscle. It does not extend below the angle of the jaw. The swelling is somewhat tender, but not red or hot. It usually begins on one side. The other side is usually involved in two or three days, but sometimes not for a week or more afterward. The disease may be limited to one side. It may extend to one or both of the submaxillary or sublingual glands. The swelling then is below the jaw just to one side of the median line. It may begin in these glands and not extend to the parotids. If several glands are involved in succession the duration of the disease is, of course, longer. When the disease is in the parotids the face feels stiff, move-

ments of the jaw are painful and mastication uncomfortable. Pressure of the swelling on the external auditory canal may cause slight deafness. Pressure on the jugular veins may interfere with the cerebral circulation and cause headache and various other nervous symptoms.

There is swelling and redness about the opening of Stenson's (Steno's) duct, which is situated on the inner surface of the cheek opposite the second molar tooth of the upper jaw. There is no swelling or redness about the opening of the ducts of the other glands when they are involved. The secretion of the glands is often diminished, which causes dryness of the mouth. In other instances, however, it may be increased.

In other instances the pain and swelling may be preceded by the usual discomfort of the onset of all acute diseases—weakness, malaise, loss of appetite, pains all over, chilliness and fever. The initial temperature is usually between 100° F. and 101° F. In severe cases it may be as high as 104° F. I have never seen nosebleed, convulsions or any of the other severe nervous symptoms which have been described as occurring at the onset.

Fig. 95.—Mumps.

During the disease the temperature is usually under 100° F., although it may sometimes be higher. Not infrequently there is no elevation of the temperature. The fever lasts until the swelling begins to go down. The constitutional symptoms may be slight or moderate. Not infrequently there are none. They usually cease as the swelling begins to subside.

There is always a relative, and sometimes an absolute, lymphocytosis. The white count may, therefore, be normal, diminished or slightly increased. The urine usually shows nothing abnormal, but may contain a trace of albumin.

Complications and Sequelæ.—These are rare in childhood. Orchitis is very uncommon before the fifteenth year. It really is rather another manifestation of the disease than a complication. The symptoms are pain in and swelling of the testicle. The onset is sometimes acute and accompanied by a chill and fever. The temperature is usually higher than with the original mumps. The chief danger is of the development of fibrous change and eventual sterility. Swelling and tenderness of the ovaries and breasts is, in my experience, more common in childhood than orchitis. I have never seen any evidences of pancreatitis in childhood. Acute nephritis is an occasional complication. I have seen several cases. Facial paralysis may develop either as the result of pressure on the facial nerve or the extension of the inflammatory process to it. The paralysis is usually fleeting, but in rare instances the injury to the nerve may be sufficient to cause permanent paralysis. I have never seen multiple neuritis in connection with mumps. Symptoms of cerebral irritation are rather unusual. They are, in most instances, due simply to irritation, but sometimes there may be a serous meningitis. In other instances the process may go a little further. The spinal fluid is

under pressure, shows a very slight test for globulin and contains an excess of mononuclear cells. The prognosis is good. In rare instances there may be a secondary infection of the gland with the pus organisms and an abscess as the result. Deafness is a rare complication. It comes on suddenly during the illness. It may be either neural or labyrinthine, but is probably usually neural. It is permanent, and may be on either one or both sides. It is not the result of otitis media, which is not a common complication of mumps.

Diagnosis.—Inflammation of the salivary glands may result from infection with any of the pus organisms. The physical signs are the same as in mumps, but the glands are likely to be more tender. The constitutional symptoms are more marked. The inflammation is almost invariably confined to one gland. It may go on to the formation of an abscess. It usually develops toward the end of some debilitating disease, like typhoid. The salivary glands may be enlarged as the result of obstruction in the ducts. The physical signs are the same as in mumps, but there are no evidences of inflammation and no constitutional symptoms. The salivary glands may also be enlarged from malignant disease. This is very unusual, however, in early life. It is a chronic condition. The parotids may be enlarged in leukemia and pseudoleukemia. The onset is slow. Other glands are involved. In leukemia the blood shows the characteristic picture of this disease.

FIG. 96.—Cervical adenitis.

Enlarged cervical lymph nodes may be mistaken for mumps. They ought not to be, however, because the location of the swelling is different. It is behind and about the angle of the jaw, not over it. The tragus is not pushed up. The opening of Stenson's duct is not reddened. It follows some other inflammatory process in the nose or throat. It is associated with a polynuclear leukocytosis.

The swelling from an abscess from a tooth is lower down on the jaw and connected with the jaw. The tragus is not pushed up, there is more tenderness and often heat and redness. There is a polynuclear leukocytosis and a diseased tooth is present in the jaw.

Treatment.—The treatment is mainly for comfort. Heat, externally applied, usually gives more relief than cold. The rubbing in of some simple ointment, like cold cream, or olive oil sometimes helps. Aspirin, in doses of from two to five grains or phenacetine and salol, equal parts, in doses of from two to five grains, according to age, may be given for discomfort. Some mild mouth-wash, like the liquor alkalinus antisepticus, should be used to prevent infection.

The diet should consist of liquids and soft solids, as chewing is painful. The child should be kept in bed, if there is any fever. If there is not, it may be up and about, but must be kept warm and quiet. Orchitis is more likely to develop, if the child is active or gets chilled. If orchitis develops, the child should be put to bed, hot or cold applications applied to the testicles and the testicles supported.

Quarantine should be enforced for at least three weeks from the onset of symptoms or for one week after the disappearance of all symptoms. The secretions from the nose and throat should be disinfected or

destroyed and the contaminated utensils and clothing disinfected. The room should be thoroughly aired and cleaned.

Hess (American Journal of Diseases of Children, 1915, Vol. 10, Page 99) found that intramuscular injections of from 6 to 8 c. cm. of whole blood from children convalescent from mumps protected other children from infection. The blood possesses protective principles even before the swelling of the glands has disappeared. It is claimed that the injection of diphtheria antitoxin diminishes the severity of the symptoms and prevents complications. This treatment seems irrational, as the antitoxin is specific only against the toxin of diphtheria.

SCARLET FEVER

Scarlet fever is uncommon during the first six months of life. Immunity is transferred through the placenta, if the mother has had the disease or has acquired an immunity in some other way. If she has not, the baby is not immune. From this time on there is a rapid increase in susceptibility. About half of the cases occur in children between three and eight years of age, and 90% in children under fifteen years. Scarlet fever is rare in adults. The insusceptibility of adults is probably because they have had the disease in a mild form without its having been recognized. It is possible that immunity may have been acquired from the repeated absorption of subinfective, immunizing doses of toxin as the result of the presence of hemolytic streptococci in the throat. An attack of scarlet fever almost always confers immunity for life.

Etiology.—It seems to be proved that scarlet fever is caused by the streptococcus hemolyticus. It is probable that at least two different strains of this organism may cause the disease.

The pathologic process is apparently analogous to that in diphtheria, that is, the local manifestations in the throat are due to the action of the causative organism, while the other symptoms are due to the action of a toxin produced by this organism. An antitoxin is produced in the patient and, when recovery occurs, causes an active immunity which persists throughout life.

Method of Infection.—The streptococcus hemolyticus, presumably the cause of this disease, is present in the discharges from the nose and throat and their adnexa. It is also present in the discharges from secondary, purulent, inflammatory foci caused by them. It is not present in the urine or desquamation. It is conceivable that the streptococci might be swallowed, pass through the digestive tract without being destroyed and be present in the feces. It is extremely improbable, however, that this happens. Contagion can occur, therefore, by direct contact and indirectly as long as the causative organism remains alive in the secretions on contaminated objects. This period evidently must vary according to the amount of exposure to sunlight and air, the resistance of the special strain of the organism and other unknown conditions. It is evident that, if the streptococci get into milk or other foods which afford a suitable culture medium, they will survive longer. It is probable that, like other bacteria which live in the mouth and throat, they may be harbored by healthy persons who act as carriers. In the vast majority of instances infection is due to direct contact. It is evident that, as the organism is the cause of the inflammatory processes in the nose and throat and their adnexa, and present in the discharges from them, the patient is contagious as long as these discharges persist.

Period of Incubation.—The incubation period varies between two and eight days. In the vast majority of cases it is between two and four days. Occasionally it is as short as one day. It is said that it may be as long as twenty-one days. It is probable, however, that there is some error in observation in these cases of presumably long incubation. It may be that the patient did not contract it at the time supposed, but later. It may be, also, that the streptococcus hemolyticus of scarlet fever may be able to live in the throat, like diphtheria bacilli and meningococci, without causing infection until conditions are favorable.

Pathology.—The inflammatory lesions in the throat vary from a simple catarrhal to a membranous, or even gangrenous, inflammation. These lesions are due to the direct action of the streptococcus hemolyticus. The lesions in the cervical lymph nodes may be due simply to toxic absorption or to the direct action of the bacteria. If suppuration occurs, there has certainly been an invasion by bacteria. The secondary infections in the adnexa of the nose and throat are due to the direct action of the streptococcus hemolyticus by extension. The secondary infections in other parts of the body are due to the same organism, which is carried in the blood. The degenerative lesions found in all the parenchymatous organs are due to the absorption of toxins, not to the organism itself. The lesions in the kidneys may, perhaps, be due in part to bacteria. The general hyperplasia of the lymphoid tissues throughout the body is due to the toxins. The inflammatory cardiac complications are caused by the streptococcus. The lesion of the skin, which is an acute dermatitis, is caused by the toxins. There is at first an acute hyperemia of the skin. This is followed by an exudation of serum and polynuclear cells into the corium, most marked about the blood-vessels and hair follicles. The epidermis is destoyed and is thrown off in the desquamation.

Symptomatology.—It is very difficult to describe the symptomatology of scarlet fever because of the great variability in the severity of the disease. It may be most malignant in its onset and symptomatology, with death in twenty-four hours or less. Death may occur even before the appearance of the rash. On the other hand, it may be so mild that it is extremely difficult to know whether the disease really is scarlet fever or not. There is no doubt that scarlet fever is, on the whole, much milder in type now than it was thirty years ago, when I first knew it. Judging from the descriptions of the disease in the older text-books, it was milder then than it was one hundred years ago.

Prodromal Stage.—The prodromal stage, or stage of invasion, is short, varying, as a rule, between twelve and thirty-six hours. In rare instances the eruption may not appear until the third or fourth day. The onset is almost always sudden and is not infrequently accompanied by vomiting. Sometimes there is a convulsion. The temperature quickly rises, in most instances to 103° F. or 104° F. In mild cases it does not go as high, while in severe cases it may reach 105° F. or even 106° F. Soreness of the throat develops almost at once. The throat is everywhere bright red. Careful inspection shows that the redness is not a uniform flush but that it is caused by a great many minute, red points. It is easier to make out these points on the hard palate than elsewhere. The tonsils are almost always more or less swollen. Membranous patches occasionally develop on them during the first day. At this time there is nothing characteristic about the appearance of the tongue. It is usually coated and sometimes the papillae at the tip and edges are enlarged.

Eruption.—The eruption almost always appears in from twelve to thirty-six hours after the onset of symptoms. Its appearance is sometimes delayed, however, until the third or fourth day. It appears first upon the neck and upper chest and spreads upward on to the face and downward over the body and on to the extremities. It usually is fully developed in from twelve to twenty-four hours. The rash is usually less marked on the face than elsewhere. The area about the mouth is almost never involved. The pale area about the mouth in combination with the redness of the rest of the face is very characteristic of scarlet fever. Although the skin appears uniformly red, it is not so, as the rash is made up of very many, minute, red points, the skin between these points being relatively much less red. The rash disappears on pressure. The duration of the eruption varies in most cases between three and seven

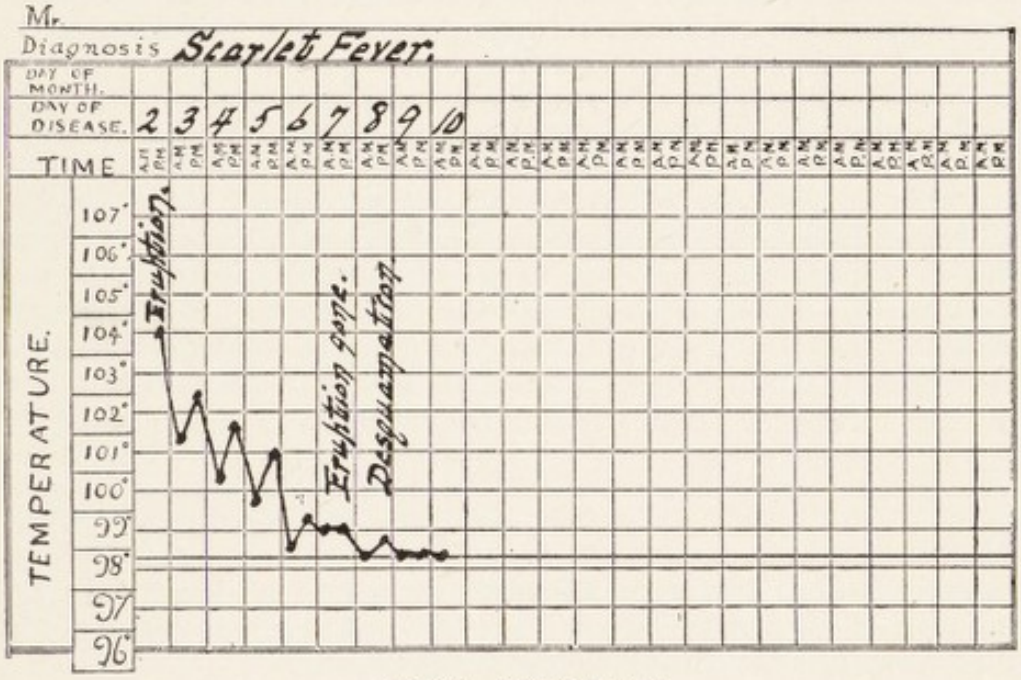

FIG. 97.—Scarlet fever.

days. It may be, however, very fleeting, while in some instances may last as long as ten or eleven days. It is accompanied by itching, which may be intense. In some cases there is swelling of the skin, usually most marked in the hands and face. It is never as marked, however, as in measles.

When the eruption is slight, it is usually most marked in the folds of the groins, axillae and elbows. In light cases the face is never involved. In such cases the rash is usually most marked on the chest and buttocks. In very mild cases it may be so fleeting that it is missed. In malignant cases death may occur before the appearance of the eruption. Occasionally the eruption is patchy instead of uniform. Sometimes there are many fine vesicles. I have never felt certain whether they were a part of the scarlet fever eruption or merely sudamina on top of it.

Other Symptoms.—The inflammation of the throat usually becomes more marked at the time of the eruption. In most instances there is membrane on the tonsils. The tonsils and fauces are much swollen and in severe cases the inflammation may become gangrenous. The inflammatory process usually extends to the nasopharynx and sometimes to the nose. It not infrequently also extends up through the Eustachian tubes

to the ears. The cervical lymph nodes are almost always enlarged. In severe cases the enlargement may be very marked and the inflammation may go on to suppuration.

The temperature remains high or increases somewhat until the eruption has fully developed. In uncomplicated cases it gradually falls, reaching normal, on the average, at about the seventh day. The rate of the pulse and respiration correspond to the height of the temperature. The tongue is at first coated and the papillae show through at the tip and edges as bright red points. Later the tongue is not coated, but bright red and a little swollen. The papillae are enlarged and distinct. It is the bright red, swollen tongue with enlarged papillae, not the coated tongue with the papillae showing through, to which the term "strawberry tongue" should properly be applied.

Urine.—The urine almost always contains albumin. In most instances it shows the evidences of acute degenerative nephritis, occasionally those of acute exudative nephritis.

Blood.—The blood shows a rapidly developing secondary anemia, the degree depending upon the severity of the infection. It persists well into convalescence. There is a polynuclear neutrophilic leukocytosis in all cases. This appears early and reaches its maximum about the fourth day. It is often not very marked in the mild cases. In cases of moderate severity it usually runs between twenty thousand and thirty thousand. In severe cases it may be as high as seventy thousand. It persists during the period of eruption and gradually diminishes during convalescence. The percentage of polynuclear neutrophiles may be as high as ninety or ninety-five. The eosinophiles disappear entirely, or are greatly diminished, at the onset and remain so until the temperature begins to fall. An eosinophilia then develops which often lasts as long as three weeks. In the most severe cases, when the organism is overwhelmed with the toxemia, there may be no leukocytosis.

Heart.—Cardiac murmurs are not uncommon at the height of the disease. They are almost always "febrile," but in severe cases are due to myocarditis and relative insufficiency at the orifices.

In very severe cases the rash may develop very quickly or its appearance may be delayed. It sometimes is very scanty and may be present for only a few hours. Death may occur before the appearance of the eruption. In these cases the symptoms of toxemia are very marked. The temperature is 105° F. or even higher, the pulse is rapid, the heart feeble and nervous symptoms marked. Hemorrhages from the mucous membranes are not uncommon. Death usually occurs in three or four days. In other severe cases the most marked symptoms are in the throat. There is marked swelling of the tissues about the fauces and pharynx. Usually there is much membrane. The inflammation extends upward into the pharynx and nose, causing interference with respiration and a profuse purulent discharge. The ears are usually involved. There is almost always marked swelling of the cervical lymph nodes. The temperature is usually not as high as in the other severe types. The evidences of toxic absorption are very marked. The urine contains much albumin. The heart shows evidences of myocarditis. The patient is either mildly delirious or apathetic. Death usually occurs in one or two weeks, but may be delayed longer. Recovery occasionally takes place, but only after a long convalescence.

Desquamation.—Desquamation usually begins soon after the rash has faded, that is, on about the eighth day. It usually appears first on the

neck and chest and gradually extends over the body and extremities. It is usually in the form of fine flakes, but may be in larger pieces. It lasts longest on the hands and feet, especially on the palms and soles, where the epidermis is thickest. Desquamation is usually complete, except on the hands and feet, within three weeks, but it may be six or seven weeks before they are entirely free. The appearance of the fingers and toes is often quite characteristic, as the skin usually comes off first at the tips, leaving the smooth, soft, new skin, while the edge of the old skin is quite distinctly marked. I have always read and heard about the skin coming off of the hands in large pieces, like the finger of a glove, or even of a whole glove. I have never, however, seen anything of this sort. In fact, when children are greased as they should be, practically no desquamation is ever seen, as it is washed off during the daily baths.

Complications and Sequelæ.—It is very difficult, in many instances, to know whether to consider special symptoms as unusually severe

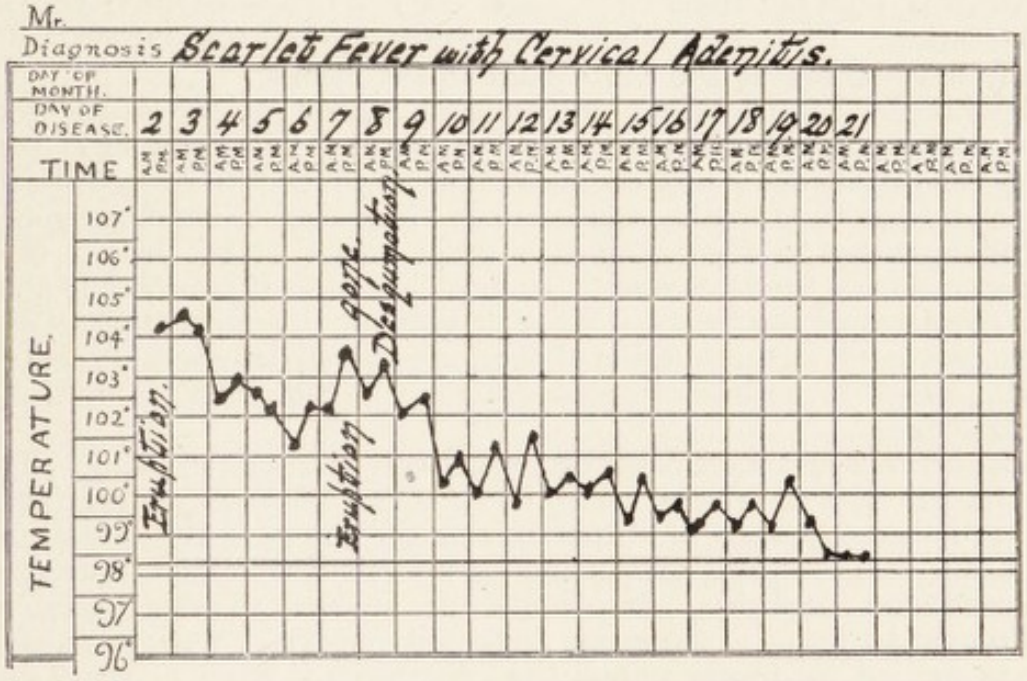

Fig. 98.—Scarlet fever with cervical adenitis.

manifestations of the disease or as real complications. It is, for example, difficult to say whether marked lesions in the throat and glands are complications or simply very severe manifestations. The inflammation in the throat may be gangrenous and accompanied by all the symptoms of a gangrenous inflammation. If the child lives long enough, there may be destruction and sloughing of the tonsils, soft palate and other tissues of the throat. Blood-vessels may be involved and serious or even fatal hemorrhages result. These severe lesions are often thought to be due to the diphtheria bacillus. In the vast majority of cases they are not, but are due to the causative streptococcus. The diagnosis can only be made by bacteriologic examination. Enlargement of the cervical lymph nodes is often very marked and may go on to suppuration. This usually does not occur until after the eruptive stage. In cases in which there is severe inflammation of the throat, there may be an extension to the soft tissues with the development of cellulitis. This may go on to suppuration and sloughing.

Otitis media is so common in scarlet fever that it is difficult to know whether it is a symptom or a complication. It is present in all fatal cases. It is caused by the streptococcus. Its development is usually not accompanied by much pain. The inflammation is likely to extend to the mastoid cells and not infrequently involves the sinuses and meninges.

Acute nephritis is a not uncommon complication. It almost always develops during convalescence, usually in the third week, and may occur in mild, as well as in severe cases. It is more common, however, in the severe cases. It may be either of the hemorrhagic or exudative type, more often, perhaps, mixed. The onset is almost always gradual. When it is apparently sudden, it is probably because the earlier symptoms have been overlooked. The development of postscarlatinal nephritis, at any rate of a severe type, can almost invariably be prevented by proper care of the patient during the acute stage and convalescence.

Cardiac complications are not uncommon. There is more or less myocarditis in all but the mildest cases. When this is marked, there may be dilatation. Endocarditis and pericarditis are not uncommon. They usually develop during the acute stage of the disease.

Pulmonary complications are unusual, except in the severe septic cases, with marked inflammation in the throat. The pathologic condition is almost always a bronchopneumonia secondary to bronchitis from extension from the throat. Occasionally there may be an empyema.

Inflammation in the joints may occur either at the height of the disease or during convalescence. In most instances it is probably due to toxic irritation of the joints. When it goes on to suppuration, it is, of course, the result of an invasion of the joints by streptococci through the blood. There is nothing characteristic about the joint complications of scarlet fever.

The nervous system is never directly involved in scarlet fever. There may be convulsions at the onset and various nervous manifestations, such as delirium, apathy or stupor, during the course of the disease as the result of toxemia. Convulsions may, of course, also be due to uremia in postscarlatinal nephritis. Meningitis or abscess of the brain may develop as the result of extension from otitis media. Various paralyses may be caused by septic emboli from the heart. None of these manifestations are, however, due directly to the scarlet fever, but are the results of the complications of scarlet fever.

Diagnosis.—The sudden onset with high temperature, sore throat and an eruption on the hard palate together with the character of the eruption on the skin are so characteristic that the diagnosis of scarlet fever is easy in the average case. In the first few hours, however, it may be impossible to distinguish scarlet fever from any of the other acute diseases which have a sudden onset, such as tonsilitis, pneumonia and acute indigestion. The great difficulty in diagnosis is in those cases in which the appearance of the eruption is delayed and in those in which the eruption is slight, fleeting or atypical. The diagnosis between German measles and scarlet fever is discussed in the diagnosis of German measles. There should be no difficulty in distinguishing between measles and scarlet fever. The onset in measles is less acute, catarrhal symptoms in the upper respiratory tract are marked, Koplik's sign is present and the rash does not appear for several days, while in scarlet fever the onset is sudden, the throat is sore, there are no definite catarrhal symptoms, there is a bright red eruption on the hard palate, Koplik's

sign is absent and the rash usually appears within twenty-four hours. In some cases in which the throat manifestations are very marked and the rash slight, it may be difficult to distinguish between diphtheria and scarlet fever. This can always be done, however, by bacteriologic examination of the throat. There are a number of drugs which cause a rash very similar to that of scarlet fever. Belladonna does it more often than other drugs. Among the other drugs which sometimes cause an erythema are quinine, antipyrin, and the salicylates. These drug eruptions, however, are not accompanied by the other symptoms of scarlet fever. There is much difference of opinion as to whether the erythematous rashes which occur in connection with wounds and burns are scarlet fever or not. My own feeling is that they are not. It is possible, however, that the toxins formed by the streptococcus hemolyticus in wounds may cause the same symptoms, except those in the throat, that they do in real scarlet fever. Great care must always be taken in making a diagnosis of scarlet fever simply on the appearance of the eruption. The diagnosis is justified only when there are other manifestations in the throat and mouth.

The *extinction test*, developed by Schultz and Charlton, is very valuable in the diagnosis of doubtful cases. They, and others, found that the serum of people convalescent from scarlet fever, and of those who had had scarlet fever in the past, when injected into the skin of patients with scarlet fever, produced a blanching of the skin which persisted. They used 0.1 c.cm. of convalescent serum. The skin in this area did not desquamate. The blanching appeared in from five to six hours after the injection and was from one to two inches in diameter. Convalescent serum had no such effect on other rashes or in erysipelas. The explanation is that the antitoxin in the serum neutralizes the toxin in the skin.

Dochez has succeeded in producing an antitoxic (immune) serum in horses by the injection of the streptococcus of scarlet fever. This serum causes an extinction of the rash in the same way as does convalescent serum. The amount injected is from 0.02 c.cm. to 0.5 c.cm. The area of blanching varies from two to six centimeters in diameter, according to the amount of serum used.

The question often arises when a child is desquamating, whether it has had scarlet fever or not. Desquamation is so common after serious febrile diseases in early life that its presence does not necessarily indicate, as so many apparently believe, that a child has had scarlet fever. No reliance can be placed upon it in diagnosis, unless the characteristic appearance at the tips of the fingers and toes is present. The question can now be settled by the use of the Dick test.

Prognosis.—It is impossible to give a definite prognosis early in the course of scarlet fever, except in the malignant type, in which it is evident that the child will not recover. It is impossible to foresee how severe the inflammatory condition in the throat may be or what complications may arise. In general, the prognosis improves progressively with age. In my experience the prognosis is better than that given in most text-books. Bad signs are a continuously high temperature, marked inflammation in the throat and great enlargement of the cervical lymph nodes. The most serious complications are those connected with the heart and ear.

Treatment.—*Prophylactic.*—It is now possible, by the use of the *Dick test*, to determine whether a child is susceptible to scarlet fever or not. This test is performed by injecting intracutaneously 0.1 c.cm. of a 1:1000

dilution of a filtrate of a culture of the streptococci which cause scarlet fever, that is, with a diluted toxin of the organism. If the child has not had scarlet fever, a reaction similar to that obtained in the Schick test appears in about six hours, reaches its maximum in from twelve to thirty-six hours and is gone in forty-eight hours. A yellowish stain remains for several days and desquamation occurs. This test is negative in those who have had scarlet fever. It is also negative during the first six months in babies whose mothers have had scarlet fever, antitoxin immunity having been transmitted through the placenta.

It is probably possible to prevent the development of scarlet fever after exposure, in a considerable proportion of cases, by the use of convalescent serum. The dose ordinarily used varies from 15 c.cm. to 30 c.cm. It is probable that the smaller dose is sufficient. Neff has had the most experience in the use of convalescent serum as a preventive after exposure. He used it in twenty-five cases and in none did the disease develop. He does not state how far along the patients from whom he got the serum were in convalescence.

The Dicks recommend the skin test in case of exposure. They take cultures of the throat on blood-agar plates at the time that the skin test is performed. If the skin test is negative, nothing is done. If the skin test is positive and the plates show no hemolytic streptococci, active immunization is carried out with three doses of toxin. If the throat culture shows hemolytic streptococci, passive immunization is accomplished by the injection of convalescent scarlet fever serum. Ten children with positive skin tests and hemolytic streptococci in their throats were treated in this way and did not have the disease. The Dicks were able, by the injection of suitable amounts of their filtrate, to produce what was apparently a mild scarlet fever. After having had this slight illness, the patients no longer showed a positive reaction to injections of the filtrate. Since then, by beginning with a smaller dose of toxin standardized on human beings, they have immunized adults by giving three injections of toxin at five day intervals. They begin with a dose equivalent to five hundred skin test doses and increase it to fifteen hundred skin test doses. An active immunization is produced in this way. Two hundred and six susceptible nurses, whom they immunized in this way before exposure, not only gave negative skin tests, but did not contract scarlet fever, although they worked in the scarlet fever ward. Four hundred and five other susceptible persons, whom they immunized, also escaped scarlet fever, although exposed to it. They emphasize the fact that sufficiently large doses must be given and that immunity is not present until the skin test is negative. Immunity is developed within two weeks and lasts at least a year and a half. Others, however, are doubtful as to the immunity produced in this way lasting more than a few weeks.

It seems probable, therefore, that in the not distant future it will be possible to protect children against scarlet fever by the production of an active immunity in the same way that it is now possible to protect them against diphtheria. If they have not been thus protected, it is possible to prevent the development of scarlet fever after exposure in a large proportion of the uncomplicated cases by the use of convalescent serum. This cannot be generally done, however, because of the difficulty in procuring a sufficient amount of serum. It seems reasonable to believe, however, that antitoxic sera, like those of Dochez and the Dicks, may confer a temporary passive immunity in the same way as convalescent

serum. The Dicks state that an immediate immunity is produced by their serum and that it lasts from four to eight weeks. From 5000 to 10,000 skin test doses should be given. One hundred thousand skin test doses of the serum of Dochez are advised by those who use this serum.

Children with scarlet fever should be isolated until all discharges from the mouth, nasopharynx and its adnexa have ceased and all lesions upon the surface of the body have healed, no matter how long this period may be. It is customary to establish a minimum period of quarantine of from four to six weeks, even if all discharges have ceased. If, as is now believed, the desquamation does not carry the contagion, it seems rather foolish to keep up the quarantine after the discharges from the mucous membranes, which are believed to carry the contagion, have ceased. The discharges from the nose and throat and their adnexa must be destroyed and all objects contaminated by them disinfected or destroyed. The greatest care must be taken by everyone who is with the patient and whose body or clothing may be contaminated by the discharges not to carry any of them out of the sick-room. The measures to be taken to prevent this are obvious.

After recovery the patient should be thoroughly washed before coming out of quarantine and should not wear any of the clothes which have been in the sick-room. Everything in the sick-room should be either destroyed or thoroughly disinfected.

If the patient is properly isolated, other children in the family, who have had the disease, may go to school. Those who have not had the disease should not return to school until eight days after the last exposure.

Care and Nursing.—Children with scarlet fever, no matter how light it is, should be put to bed and kept there until desquamation has ceased, except on the hands and feet. If children are kept in bed in this way, not nearly as many complications develop.

The diet should be regulated in the same way as in acute nephritis, that is, the proteins should be limited to the amount necessary to cover the minimum protein need, the necessary calories being provided by fats and carbohydrates. The child should be made to drink considerable amounts of water. If these precautions are taken, acute nephritis almost never develops. If it does, it is almost always of a mild type and yields promptly to treatment.

Children with scarlet fever should be bathed and cared for in the same way as children ill with other acute febrile diseases. If the temperature is high and causing symptoms, it should be treated in the same way as in typhoid fever. It is much better to use cold externally than antipyretics internally.

The itching of the skin can usually be relieved by sponging with equal parts of alcohol and water or with a solution of one teaspoonful of bicarbonate of soda in a quart of water. Any of the simple dusting powders may be used. Sometimes greasing the skin with sweet oil or vaseline works better. As soon as desquamation begins the skin should be thoroughly greased daily with sweet oil or vaseline.

The treatment of the inflammatory conditions in the nose, throat and ears is the same as when these conditions are not a part of scarlet fever. The ears should be examined daily so that, if otitis develops, it may be treated early. The treatment of cervical adenitis in scarlet fever is the same as when it is due to other causes. The treatment of acute nephritis complicating scarlet fever is discussed in the treatment of acute nephritis. The treatment of the cardiac complications is discussed under heart

disease. When stimulation is required, it should be along the same lines as in other acute diseases. Whisky is not a stimulant and can not be given in sufficient amounts to be of any use as a food without disturbing the digestion or "doping" the patient. I can see no justification for the use of digitalis in scarlet fever. The cardiac weakness is due to acute degeneration of the myocardium. Digitalis can not act on an acutely degenerated muscle and, therefore, can do no good. It is, moreover, contraindicated in the acute stage of endocarditis. The most satisfactory stimulant, in my experience, is strychnine. The beginning dose for a child of four years is $\frac{1}{120}$ of a grain. This dose may be increased as necessary. If a quick stimulant is needed, citrate of caffeine, in doses of from $\frac{1}{2}$ to 1 grain, may be given by the mouth, or slightly smaller doses of caffeine-sodium benzoate or salicylate may be given subcutaneously. A grain of camphor in oil may also be given subcutaneously. It must be remembered that these drugs have only a fleeting action and should be given only in emergencies. If a continued action is desired, it is necessary to give them at least as often as every hour.

Medicinal.—There are no drugs which have any effect on the course of scarlet fever. There are no drugs which, given internally, prevent any of the complications. It is useless to give hexamethylenamine with the idea of preventing complications, because this drug only acts after it has been broken up and it is broken up only in an acid medium. All the fluids of the body, except the urine, are alkaline. Even if it is broken up in the urine, it can have no effect in preventing nephritis, because the urine is not in the kidney.

Serum Treatment.—Very good results are obtained by the use of convalescent serum or whole blood intramuscularly. The action is undoubtedly antitoxic. There is usually a prompt fall in the temperature and marked improvement in the general condition. The blood is usually taken during the fourth or early in the fifth week of the disease. It cannot be kept long, as it begins to lose its effectiveness within a few weeks after it is taken. The dose of serum varies from sixty c.cm. to ninety c.cm. The earlier it is used, the better are the results.

Blake, Trask and Lynch have used Dochez' antiscarlatinal serum, prepared by the immunization of a horse with the scarlet fever streptococcus, in the treatment of a considerable series of cases. They conclude that "Scarlatinal antitoxin in proper amounts is a specific and prompt cure for uncomplicated scarlet fever. It indirectly benefits septic complications during the acute stage. It has no therapeutic value in post-scarlatinal sepsis after the rash has faded."

They advise the intramuscular injection of from thirty c.cm. to one hundred and twenty c.cm. of a serum which neutralizes 10,000 skin test doses of toxin per c. cm., depending upon the size of the patient and the severity of the disease.

The Dicks have succeeded in producing an antitoxic serum in horses by the use of sterile scarlet fever toxin. They have used it in twenty-nine cases with a mortality of 3.4%, compared with one of 20% in fifteen control cases. Complications were much less common than in the controls. They recommend a dose of serum which contains sufficient antitoxin to neutralize 20,000 skin test doses of toxin. One dose is usually all that is given, but two may occasionally be necessary. (Journal Amer. Med. Ass., 1925. Vol. 84, page 803.)

It seems almost certain from the reports of the Dicks, Blake and Trask, and others that the antitoxic sera are effective in the treatment of scarlet

fever. It seems equally evident, however, that much more must be learned as to the best type of antitoxic serum to use and as to its standardization before any positive statements can be made as to the dosage to be used, not only in treatment, but also in immunization. The Massachusetts State Board of Health recommends a minimum therapeutic dose of 300,000 skin test doses, that is, fifteen c.cm. of serum containing 20,000 skin test doses to the c.cm.

MEASLES

Measles, like whooping-cough, is a far more serious disease than is usually realized. Roughly, there are ten thousand deaths annually in the United States from measles. Ninety per cent. of these deaths are in children under five years of age and the vast majority of them in babies under two years. These figures do not include the large number of deaths from complicating bronchopneumonia.

Measles may occasionally occur in utero. When the mother has had measles previously, however, immunity is transferred to the baby from the mother through the placenta. No immune bodies are transferred through the milk. Measles almost never occurs in the first two months of life and very seldom in the first five months. The susceptibility rapidly increases from this time on, however, so that at one year at least 90% are susceptible and after two years it is probable that at least 99% of all individuals are susceptible. The susceptibility lasts throughout life. Permanent immunity almost always follows an attack. It is safe to conclude that, if a person is said to have had several attacks of measles, only one of them was really measles.

Etiology.—Measles is undoubtedly caused by some microorganism. The chances are that this organism is some form of filterable virus rather than the diplococcus of Tunnicliff and Moody. Experiments on animals show that the microorganism which causes the disease is present in the blood for at least one or two days before and for three days after the appearance of the eruption. It is not present in the blood during the first four days after exposure and apparently is not present longer than three days after the appearance of the eruption. The virus is also present in the buccal and nasopharyngeal secretions at least twenty-four hours before the appearance of the rash and for two days longer. There are no data as to how long it may continue to be present in these secretions, but it is certain that there is a reduction in, if not a total loss of the infectivity of these secretions as convalescence approaches. The data as to whether or not the virus is present in the skin during the eruptive stage are inconclusive.

Nothing is known as to the habitat of the virus of measles outside of the human body. It is certain, however, that the viability of the virus outside of the body is very slight.

Contagiousness.—In spite of the experimental data as to the apparently short duration of the period in which the virus is present in the blood and secretions of the nasopharynx, clinically measles is contagious from the first appearance of the symptoms, whether these are fever or catarrhal. It is probably contagious for about five days after the appearance of the eruption. The desquamation is not contagious. It is possible that measles may be contagious as long as a catarrhal condition of the nasopharynx and its adnexa persists. It is probable, however, that this is not the case.

The virus leaves the body in the secretions of the respiratory tract and its adnexa. It enters the body through the respiratory tract. Contagion occurs as the result of direct contact with people ill with the disease. The contagion may be air-borne, but only for a short distance. Only a slight exposure is necessary. Indirect contagion through utensils and clothing is possible, but the exposure must occur quickly, as in whooping-cough, the duration of the viability of the virus outside of the body being very short. There are no carriers.

Period of Incubation.—The usual period of incubation, that is, the time between exposure and the development of fever or catarrhal symptoms, is ten or eleven days. In rare instances it may be as short as seven days, and occasionally as long as fifteen days.

Pathology.—There is congestion of the skin accompanied by an exudation of round cells about the small blood-vessels. There is also an exudation of round cells about the sweat and sebaceous glands. As the result of this exudation and of edema there is swelling of the skin. This swelling is most marked in the face.

There is also a catarrhal inflammation of the mucous membranes of the nasopharynx and its adnexa, as well as of the larynx, trachea and large bronchi. This not infrequently extends to the small bronchi. The conjunctivae are also involved. The mucous membrane of the intestines is likewise involved in some cases. Complicating bronchopneumonia and otitis media are due to secondary infection.

Symptomatology.—The *prodromal stage*, or, as it is now the fashion to call it, the *stage of invasion*, lasts from three to five days, the average

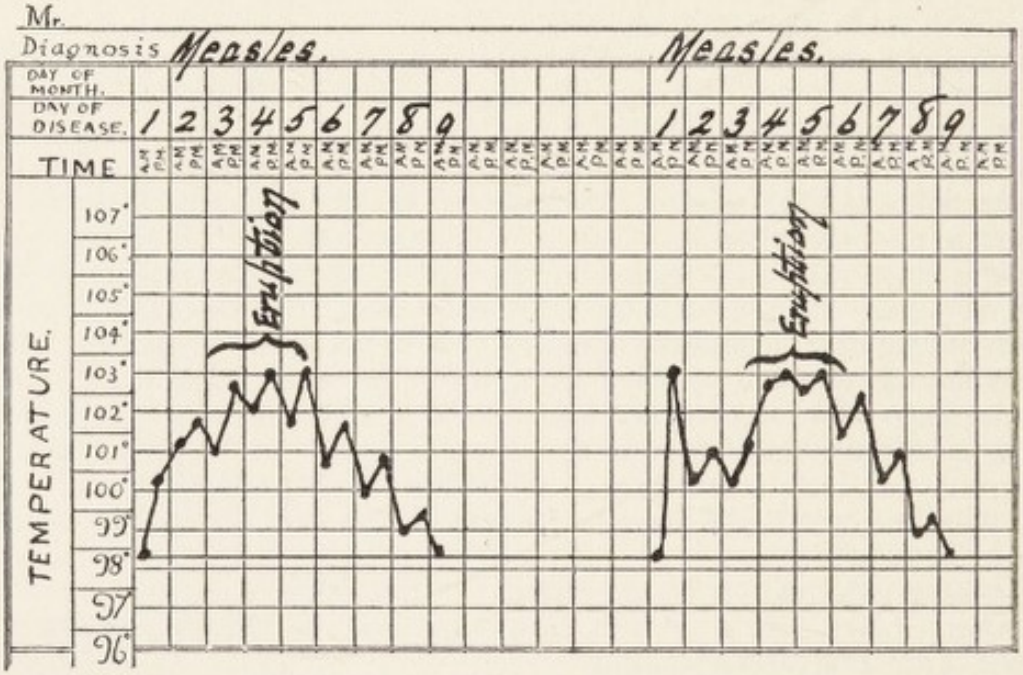

Fig. 99.—Characteristic charts of measles.

being four days. The first symptoms are usually simply those of a catarrhal inflammation of the upper respiratory tract. There is nothing about them in the beginning to distinguish them from those of any other catarrhal inflammation of this tract. These symptoms may sometimes be preceded for twenty-four hours by fever and malaise. As a rule, however, the fever first appears with the catarrhal symptoms. The temperature usually rises slowly during the stage of invasion, ordinarily not

going higher than 102° F. or perhaps 103° F. It is not uncommon, however, for the temperature to rise fairly quickly during the first twenty-four or thirty-six hours and then to fall to perhaps 100° F. to rise again just before the appearance of the eruption. In occasional instances the temperature may rise very quickly to 104° F. or even higher at the onset. In some instances the stage of invasion is very short. Toward the end of the stage of invasion there is likely to be a catarrhal conjunctivitis and photophobia. There is also likely to be more puffiness about the eyes than is usual with an ordinary cold. Cough is frequent, soft and short. It is very difficult to describe how it differs from the ordinary cough, but it is characteristic enough so that one who has heard it a few times always recognizes it and suspects measles at once. Children in this stage are likely to be somewhat dopy and sleepy. The old ladies used to describe the condition as "sleeping for the measles." There may or may not be evidences of bronchitis at this stage. In rare instances the onset is accompanied by vomiting and convulsions. In the cases in which the fever is high at the onset, the symptoms of bronchitis are likely to be quite marked. When the temperature drops after an initial rise, the symptoms often abate temporarily at the same time.

Koplik's Spots.—Usually about the second day after the appearance of symptoms, Koplik's spots appear in the mouth. They are most common on the inside of the cheeks opposite the molar teeth, next, on the inside of the lower lip. There may be only a few of these spots or the whole mouth may be peppered with them. The typical Koplik's spot is a pearly-white spot about the size of the shaft of a pin, surrounded by a rose-red areola somewhat larger than the head of a pin. When the enanthem is profuse, there are very many of the pearly-white spots but the rose-red areola is not distinct. In some instances the pearly-white spots are so numerous that they are practically confluent. It is impossible to see these spots except with a strong light directed into the mouth. Sunlight is far more satisfactory than artificial light. These spots are present in almost every case. They do not occur in any other condition and are, therefore, pathognomonic of measles. They usually disappear before the appearance of the exanthem, but may persist a day or two after its appearance.

A day or so before the appearance of the exanthem, dark red macules develop on the hard and soft palates. They average about one eighth of an inch in diameter. They are not the same as Koplik's spots and, while very characteristic of measles, are not pathognomonic, as similar appearances are sometimes seen in other conditions.

Eruption.—The eruption usually appears on the fourth day, but sometimes a little earlier or a little later. It appears first on the face and neck, most often about the ears or on the forehead at the root of the hair. Occasionally, however, it appears first on the trunk, more often on the back than on the front. It spreads over the face and neck on to the back, over the trunk, then over the upper arms and thighs and, finally, over the forearms and legs. The rash appears first as small rose-red macules about an eighth of an inch in diameter. These increase in size until they are a quarter of an inch or even more in diameter. As they develop in size they become somewhat elevated but, to my mind, are very seldom really papules. The color is rose-red or somewhat dark red, not at all like the bright red of scarlet fever. The individual spots usually have an irregular outline. I have seldom been able to make out either a crescentic shape of an individual spot or a crescentic arrangement of a

collection of spots, although these are described as characteristic of the disease. The spots may be so numerous that they coalesce. The rash is usually fully developed in from thirty-six to forty-eight hours, but the full development is sometimes delayed. In occasional instances the rash is much slower in appearing, and it is sometimes very scanty. It fades rapidly in two or three days, the fading occurring in the order of the appearance of the eruption. By the end of four or five days there is usually left only a brownish stain. This may last for a week or two longer.

The temperature usually continues high until the eruption has entirely appeared. In uncomplicated cases it then comes down to normal by a rapid lysis in the course of from two to five days. The rate of the pulse and respiration varies with the temperature.

The catarrhal symptoms are usually more marked until the eruption has fully developed. They then, in uncomplicated cases, quickly diminish with the fall of the temperature and the disappearance of the rash. The face is considerably swollen, especially about the eyes, at the height of the eruption. The conjunctivitis and photophobia are usually more marked. Children are usually very dopy and sleepy until the eruption begins to fade. There is, of course, lacrimation, nasal discharge and cough. The tongue is coated and the papillae are sometimes enlarged. There is, however, nothing characteristic about its appearance. The cervical lymph nodes are often enlarged. In some cases there is a coincident diarrhea, presumably due to a catarrhal condition of the intestines.

Desquamation.—Desquamation begins at the time of or soon after the subsidence of the rash. It begins on the face and neck, where the eruption first appeared, and follows the same course over the body and extremities that the eruption did. The desquamation is composed of fine, branny scales. It is often slight. It is usually most marked in the cases in which the eruption was most intense. In most cases it lasts from five to ten days, but it may continue as long as two weeks.

During this stage the temperature is normal and the child feels well. The catarrhal symptoms rapidly clear up, unless there are complications.

Urine.—The urine usually shows the evidences of acute degenerative nephritis, although in mild cases it may be normal.

Blood.—During the stage of incubation there is a relative diminution in the proportion of lymphocytes and an increase in the relative proportion of polynuclear neutrophiles. By the time of the appearance of symptoms, there is usually also a leukopenia. The leukopenia is usually most marked at the time of the height of the eruption, when the decrease in the proportion of mononuclear cells is also most marked. The eosinophiles diminish during the period of incubation and are almost entirely absent during the stage of eruption. As the temperature falls, the mononuclear cells increase in number and the eosinophiles reappear. If there is a leukocytosis, there is always some complication.

Atypical Measles.—In some cases the eruption is scanty. In others the spots are small and light-colored, so that it resembles a papular erythema. Sometimes the erythematous eruption is more or less confluent. Not very infrequently the eruption appears first on the body or extremities instead of on the face and neck. It may also sometimes be fleeting.

There is usually some enlargement of the postcervical glands. In some cases this is marked enough so that it may cause confusion with German measles.

Hemorrhagic, or *malignant*, measles is very rare and almost always fatal. In this type not only is the rash hemorrhagic, but there are hemorrhages from the mucous membranes and sometimes from the kidneys and bowels. It is very possible that in these cases the hemorrhagic symptoms are due to a concurrent streptococcus infection.

Complications and Sequelæ.—The most common complications are bronchopneumonia and otitis media. The younger the patient, the more likely are these complications to develop. There is nothing characteristic about the bronchopneumonia complicating measles. Otitis media is, perhaps, less likely to be accompanied by pain when it is a complication of measles than when it is secondary to other conditions.

Acute nephritis is a rather common complication. It usually develops toward the end of the eruptive stage.

Severe inflammation of the larynx is an occasional complication. When it occurs early, it is usually due to streptococci; when it occurs late, to diphtheria bacilli. When caused by streptococci, it may go on to necrosis and destruction.

Lobar pneumonia is an occasional complication. It may be accompanied or followed by its complications, pleurisy and empyema.

Slight or moderate diarrhea is common enough to be considered one of the usual manifestations of the disease. Occasionally, however, it is severe, and sometimes dysenteric in type.

Noma is one of the most terrible complications of measles. It seldom, if ever, occurs, except in feeble and debilitated babies.

In neglected cases, the inflammation of the conjunctiva may extend to the cornea and cause ulcerations. In rare instances, iritis may also develop.

Other occasional complications are infections of the skin, severe cervical adenitis with abscess formation and acute thyroiditis. The last is a trivial condition, which disappears in a few days and never goes on to suppuration.

I have never seen any of the complications in the nervous system which are spoken of, although I have occasionally seen an incidental tubercular meningitis or infantile paralysis with the measles.

Cardiac complications are very unusual, although in severe cases, as in other infections, there may be a myocarditis.

Measles does not cause infection with tuberculosis. If tuberculosis develops with or immediately after measles, the true explanation is that the measles simply lighted up an existing focus of tuberculosis. This is usually located in the tracheobronchial lymph nodes. Incidentally, these are almost invariably enlarged in measles, because of the catarrhal condition in the bronchi.

Diagnosis.—It is impossible to make the diagnosis of measles in the beginning on the symptomatology. It may be suspected, however, if there is an epidemic at the time or there has been a known exposure to measles. The most suggestive points in the early symptoms are drowsiness and puffiness of the eyelids. The blood shows a rather characteristic picture, that is, a leukopenia with a marked diminution in the relative proportion of the mononuclear cells and a diminution in the number or even absence of eosinophiles. Koplik's sign is very valuable in diagnosis. It occurs before the appearance of the eruption, is present in almost every case of measles and does not occur in any other condition. The only thing with which it is likely to be confused is stomatitis herpetica. In stomatitis, however, the red spots are larger and the centres yellow and depressed. The enanthem on the hard and soft palates is also of impor-

tance and appears before the eruption on the skin. It does not have the value of Koplik's sign, however, as similar appearances are sometimes seen in other conditions.

The diagnosis between measles and German measles is usually not difficult and is taken up in discussing the diagnosis of German measles. The diagnosis between measles and scarlet fever is also seldom difficult. The onset of scarlet fever is more acute, there are no Koplik's spots, the enanthem is diffuse and made up of small, bright red points, not of rose-red or dark red blotches. The exanthem appears much earlier. It appears first on the neck and upper chest instead of on the face and about the hair. It is bright red and made up of fine points instead of rose-red or dark red and made up of larger spots and blotches. It fades on pressure much more than does the measles rash. The tongue is red and the papillae enlarged in scarlet fever. It is coated in measles. The catarrhal symptoms are lacking in scarlet fever although, of course, there is often much inflammation in the throat. There is a marked polynuclear leukocytosis in scarlet fever instead of the leukopenia with diminution in the mononuclear cells and eosinophiles in measles.

Antitoxin rashes and rashes due to indigestion are often mistaken for measles. They should not be, however, because they are not preceded and accompanied by catarrhal symptoms in the upper respiratory tract. They do not appear first on the face and usually are widely distributed from the beginning. Antitoxin rashes are likely to be accompanied by pain and to be multiform in character. Furthermore, there is always the history of the administration of antitoxin shortly before. Indigestion rashes are almost invariably accompanied by other symptoms of indigestion and, in most instances, there is a history of the ingestion of some improper food. Toxic erythemas from drugs are also occasionally mistaken for measles. They should not be, because they are not preceded by catarrhal symptoms, are widely distributed from the beginning and are usually not accompanied by fever. There is also a history of the ingestion of some drug. The syphilitic roseola is also sometimes mistaken for measles. This ought never to happen, because it is not preceded by catarrhal symptoms and is not accompanied by fever. The distribution of the rash is different, as is also its color, which is light red.

During the period of desquamation it is sometimes necessary to decide whether the child has had scarlet fever or measles. This is easy, if there is a history. If there is no history, it may be harder. The desquamation in measles is fine and branny; in scarlet fever it is in large scales or flakes. The characteristic appearance of the fingers and toes in scarlet fever is never seen in measles. The desquamative period in measles seldom lasts more than ten days, while in scarlet fever it often lasts six weeks. The mistake is also often made of thinking that a child has had measles because it is desquamating a little. Unless there is a definite history of measles, however, it is usually safe to conclude that the desquamation is not due to measles, because desquamation of the skin is very common after all severe illnesses in children.

Prognosis.—Uncomplicated measles is seldom fatal, even in infancy, except in feeble and debilitated babies. Unfortunately, however, complications, especially in the lungs, are very common in the first few years of life and cause a large mortality. Bad signs in measles are a very high temperature, a scanty eruption in a manifestly sick child, late appearance of the eruption and a hemorrhagic eruption. Death is almost invariable in the hemorrhagic type.

Treatment.—*Prophylaxis.*—Measles is so serious a disease in infancy, because of its complications, that every precaution possible should be taken to prevent babies from having it. While it is not as important to prevent children from two to five years from having the measles, it is much better for them to avoid it, if possible, until five years have passed. After this, measles, while an uncomfortable and disagreeable disease, is seldom dangerous. Unfortunately, it is very difficult to guard babies and young children against infection with the measles, because of its contagiousness in the catarrhal stage before characteristic symptoms have developed. As it is impossible to distinguish the early stage of measles from a common cold, children with colds should be kept away from babies. Children with measles should be isolated from the time the disease is recognized until at least five days after the appearance of the rash. All articles contaminated by the secretions of the nose and mouth should be disinfected or destroyed. Formal disinfection is not necessary. Thorough airing and cleaning of the room is sufficient. There is no reason why other children in the family who have had the measles should not go to school. Those who have not had it should be kept out of school until sixteen days after the last exposure. It goes without saying that it is just as important to keep children who have been exposed away from places of public congregation and other children as it is to keep them out of school.

Active Immunity.—Herrman, of New York, believes that it is possible to develop an active immunity against measles and to confer protection for at least two years, that is, during the time when the disease is most dangerous. His theory is that infants under five months of age are relatively immune and that, if infected, they acquire a mild form of the disease. He seeks to utilize this period of relative immunity as the time to inoculate all children and get a mild form of the disease, which will render them immune. He applies the nasal washings, mixed with saline solution, taken from twenty-four to forty-eight hours before the appearance of the rash, to the nasal mucosa of the infants. The majority show no distinct reaction. In some there is a slight rise of temperature to from 100° F. to 101° F., which appears from the eighth to the fourteenth day. Occasionally there are a small number of spots on the face and body, which appear from the fourteenth to the eighteenth day. There are no other symptoms.

Regan believes that it is better to get modified measles by giving convalescent serum late in the incubation period. Measles modified in this way also confers active immunity. He believes that this is better than to give convalescent serum early and get a passive immunity of short duration. He finds that when convalescent serum is injected late in the period of incubation, modified measles results. The disease is altered as follows: The incubation shows a definite tendency to be longer. The period of invasion is shortened. Coryza is either absent or mild in degree. Koplik's spots are usually absent or are atypical in evolution. The enanthem is lacking. Prodromal rashes occur with greater frequency. The eruption is altered by being either scanty or atypical and, in most instances, by becoming ecchymotic as it fades. The temperature curve is distinctly lower, the constitutional reaction is decidedly less pronounced, complications are practically lacking.

Passive Immunity.—Passive immunity can be produced in many instances, but not in all, by the injection of blood serum or blood from persons convalescent from measles. The best time for bleeding convales-

cent donors is from the sixth to the tenth day after the temperature has reached normal. The blood serum from persons who have had the measles several months, or even years previously, also has a protective value. The dose, however, should be larger. The Wassermann test should, of course, always be done. Three different serums should be pooled, as it is possible that a convalescent donor may be a poor anti-body producer.

The doses of serum recommended by Degkwitz are 2.5 c.cm. for children who have been exposed not more than four days previously, 5 c.cm. for children exposed five or six days previously and 7.5 c.cm. for children exposed seven days previously to infection with the measles virus. Injection on the eighth day, or even later, after exposure apparently, in some instances, protects against the disease. In others it modifies it and in others has no effect. The serum should be given intramuscularly. The doses recommended are for children of three years. For older children they should be increased in proportion to the age and weight of the child.

Zingher recommends the use of whole blood, as being easier and equally satisfactory. The dose is about twice that of the serum. In order to obviate haste, he recommends putting 1 or 2 c.cm. of a 5% solution of sodium citrate in the syringe before taking 10 to 20 c.cm. of blood into the syringe. The blood should be injected intramuscularly.

The passive immunity produced by convalescent serum or blood injected during the first few days after exposure does not last more than four or five weeks. When the serum is injected on the sixth day or later, a mixed type of immunity is produced, which lasts from two to three months. If a modified attack of measles develops, the immunity produced is probably permanent.

Convalescent Serum.—Convalescent serum given during the period of invasion apparently has very little effect on the course of the disease or in modifying its severity. It does not influence any of the characteristic symptoms.

Medicinal.—There is no drug which affects in any way the development of the symptoms, the course or the prognosis of measles. Treatment must be directed, therefore, to making the patient comfortable, relieving the symptoms and preventing complications.

Children with measles should be put to bed and kept there until they are well, that is, until all catarrhal symptoms have disappeared and desquamation has ceased. They should wear their usual nightclothes. It is not necessary to put on heavy clothes or to have an unusual number of bedclothes. The temperature of the room should be equable. It is not necessary to have it as high even as from 68° F. to 70° F., as is usually recommended. It certainly should never be above 70° F. when the child is in bed. 64° F. is the optimum temperature. The room must be well ventilated. Fresh, pure air does more than anything else to prevent the development of complications in the respiratory tract. Nothing is so likely to cause them as close, hot, bad air. Sunlight does not cause the conjunctivitis of measles. It does cause discomfort, if conjunctivitis has developed. The light in the room should be regulated, therefore, for the comfort of the patient. It is not necessary to exclude the light, if it does not cause discomfort. When the child is too young to tell how it feels, those in charge of it should use what common sense they have as to the amount of light allowed.

There are no special indications as to the diet in measles. When the appetite is poor, it is inadvisable, of course, to give soups and broths,

which contain no nourishment. Milk should form the basis of the diet. There is no objection to soft solids. Water should be pushed. The bowels should be kept open. If there is diarrhea, it should be treated in the same way as diarrheas from other causes. Bathing will not hurt a child with the measles. It is impossible to drive the rash in. A child with the measles should have the usual cleansing baths morning and night that other ill people have. After the baths it is advisable to grease the skin with sweet oil or some simple ointment, like cold cream or boracic acid ointment, to diminish the itching. In some cases, thoroughly powdering the skin affords more relief than greasing. Any simple dusting powder is suitable. Stearate of zinc powder is better than most of them, because it sticks better.

If the temperature is high and causing symptoms, it should be treated by the use of cold externally, as in other diseases. The methods to be used are described in the treatment of typhoid fever. There need be no fear of doing any harm or driving the rash in. While it is impossible to drive the rash in, it is sometimes possible, however, to bring it out, when it is slow in appearing, by the application of heat externally, best in the form of a hot bath. An icebag should be applied to the head, if the temperature is high and there are marked nervous symptoms.

The treatment of the catarrhal condition in the nose and throat, of the bronchitis and of the cough which accompanies them is the same as in nasopharyngitis and bronchitis from other causes.

When there is conjunctivitis, a 4% solution of boracic acid should be put in the eyes every one, two or three hours, according to the severity of the condition. It may be put in with a medicine dropper or with absorbent cotton wet with it. Another useful eye wash is ten grains of borate of soda in one ounce of camphor water. In severer cases a 10% aqueous solution of argyrol may be used several times daily. If there is much secretion, vaseline, cold cream or boracic acid ointment should be applied to the edges of the lids after each cleansing to prevent them from sticking together. If there is much swelling, cold compresses may be used over the eyes.

The ears should be examined daily. The treatment of otitis media complicating measles is the same as when it is due to other causes.

The bromides, paregoric, codeine, aspirin, phenacetine and salol may be used for discomfort in measles as in other acute, febrile diseases. Their use has been taken up in discussing nasopharyngitis, tonsilitis and bronchitis. Stimulation should be used as in other conditions, if needed. It is seldom needed, however, unless bronchopneumonia has developed.

The susceptibility of the mucous membranes of the respiratory tract after measles should be kept in mind during convalescence and for some months afterward, and the children protected, as far as possible, against exposure and infection. Most of the troubles which develop after measles and are attributed to measles are due to lack of care during the convalescence rather than to the measles.

GERMAN MEASLES

There is now no doubt that German measles is a disease *sui generis*, entirely distinct from measles and scarlet fever. Nothing whatever is known as to the nature of the causative microorganism or as to how it enters or leaves the body. It is generally believed at present that it is contagious only during the period of eruption. It is apparently

disseminated chiefly by direct contact and rarely, if ever, indirectly. There are no carriers. Infants under six months of age are almost immune. The susceptibility is general after this age.

Period of Incubation.—The usual period of incubation is between fourteen and twenty-one days, the average being seventeen days. It is possible that the lower limit may be ten days.

Symptomatology.—The *prodromal stage,* or *stage of invasion,* is very short. It is seldom longer than one day. In many instances there are no prodromal symptoms, the rash being the first evidence of the disease. When there are prodromal symptoms they are usually insignificant, slight fever and malaise. Sometimes there are slight catarrhal symptoms in the upper respiratory tract. In rare instances there may be headache, chills and vomiting. I have never seen convulsions, nose-bleed or delirium, although they are mentioned by various authors.

Eruption.—The eruption usually appears first on the face. It spreads rapidly over the body and extremities and generally reaches its full development within twenty-four hours. Occasionally the eruption appears first on the trunk and sometimes it appears over the whole body simultaneously. In other instances the rash is slight. Sometimes it is limited to the face.

As a rule, the rash resembles quite closely that of measles. It is, however, almost always lighter in color than that of measles and somewhat less elevated. Occasionally the eruption is much darker in color, and sometimes it is elevated more than is usual in measles. In other instances the rash is profuse and almost confluent. This is especially likely to happen on the face. In still other instances there is a nearly uniform red blush, which resembles closely the eruption of scarlet fever. The color is seldom, however, as bright as that in scarlet fever and there are almost always places where it is macular in character. The eruption quickly fades, the duration usually being three days. It may, however, be as short as one day or as long as four days. It fades in the order of its appearance. The pigmentation of the skin is much less than in measles and may be entirely wanting.

Desquamation.—The desquamation is usually very slight, and may be entirely absent. It is in the form of fine scales.

Other Symptoms.—The temperature usually reaches its maximum at the height of the eruption. It is seldom over 101° F. Often it is not as high as this. Occasionally it may be as high as 103° F. or 104° F. It seldom lasts more than forty-eight hours. It may fall to normal by crisis or by a rapid lysis. It usually does not last as long as the eruption. The malaise and discomfort correspond to the height of the temperature. There are occasionally catarrhal symptoms in the nasopharynx and sometimes slight sore throat. The postcervical glands are almost always enlarged, but usually not very much. The inflammation of the glands never goes on to suppuration. The urine is normal. There is a slight leukopenia with a slight relative lymphocytosis both for a few days before and at the time of the eruption.

An enanthem appears on the uvula and soft palate at the same time as the exanthem. It rarely extends on to the hard palate. The spots are about the size of a pinhead or a little larger, and are rose-red in color. During the process of involution yellowish or yellowish-brown spots or streaks are sometimes seen.

Diagnosis.—Because of the similarity of the eruptions, German measles and measles may be confused. In many instances it is impossible

to make a positive diagnosis on the characteristics of the eruption alone. The eruption in measles is, however, preceded for some days by fever and catarrhal symptoms. Koplik's sign is present in almost every case and the enanthem precedes the exanthem. In German measles the eruption is preceded by other symptoms for less than twenty-four hours or the eruption is the first symptom. Catarrhal symptoms are absent or very slight. There is no Koplik's sign and the enanthem, if present, appears at the same time as the exanthem. The enlargement of the postcervical glands in German measles, to which so much importance is often attached, is of no value, because the postcervical glands are also almost always enlarged in measles.

The leukopenia and the diminution in mononuclear cells are less marked in German measles than in measles. These differences are only in degree, however, and, therefore, of no importance in the differential diagnosis. The absence of eosinophiles in the blood in measles is, however, pathognomonic of that disease.

When the eruption in German measles is diffuse and red, it may be mistaken for that of scarlet fever. It ought not to be, however, because the eruption is not as bright red as in scarlet fever, is not made up of a large number of very fine points and does not fade as much on pressure. Furthermore, it does not appear first on the face and neck as in scarlet fever and is not more marked in the folds of the elbows and groins as it is in scarlet fever. The eruption in scarlet fever is usually preceded, moreover, by a high temperature, marked constitutional symptoms and a sore throat. There is a marked leukocytosis in scarlet fever, none in German measles.

Antitoxin rashes, as well as rashes from indigestion and from drugs, may also be confused with German measles. The principles of the differential diagnosis between these rashes and German measles are the same as between them and measles.

Prognosis.—German measles is a very mild disease. I have never known a child with it to be seriously sick. There are almost never any complications or sequelae.

Treatment.—The treatment is entirely symptomatic and for comfort. As a precaution, children with German measles should be kept in bed and on a light diet as long as the temperature remains elevated and until the rash has entirely disappeared. They should be isolated until the disappearance of the eruption. Formal disinfection is not necessary. Other children in the family, who have had the disease, should be allowed to go to school. Other children in the family, who have not had the disease, should be kept away from school and other children for twenty-two days from the last exposure. They are, of course, not dangerous to other children during the first ten days after the first exposure.

INFANTILE ROSEOLA

This syndrome, also called *exanthem subitum*, is, without much question, a clinical entity. It is not the condition to which the term was applied fifty or one hundred years ago, but, as the present generation of physicians knows nothing about the former use of the term, it seems allowable to adopt it for this symptom complex.

Etiology.—Nothing whatever is known as to the etiology of this condition. It has all the earmarks of an acute infection. It is, however, not contagious. It is also evidently not connected with any form of food or with disturbance of the digestion. It may occur at any time

of the year, but apparently is more common in the winter and spring. It almost never occurs after two years of age.

Symptomatology.—The onset is always acute. The temperature rises suddenly, usually to about 104° F. It usually remains high for four days and then drops quickly to normal by crisis. The duration of the fever in some cases is three days and in others five days. It is seldom either shorter or longer. During the febrile period the chief symptoms are irritability and loss of appetite. Sometimes the babies are drowsy. The pharynx and fauces are often somewhat reddened and occasionally there are symptoms of a slight inflammation of the nasopharynx.

Coincident with the drop in the temperature or immediately after it, the eruption appears. It resembles very closely the rash of German measles. The eruption usually appears first on the trunk. It spreads rapidly over the body and extremities and upward on to the neck and face, reaching its maximum in about twelve hours. It is usually less marked on the face than elsewhere. It fades out quickly and is gone in from twenty-four to forty-eight hours after it has reached its height. There is usually no desquamation. If there is, it is very slight. As soon as the temperature drops and the rash appears the baby acts perfectly well.

There is no enlargement of the postcervical lymph nodes. The urine is normal. The blood shows a fairly marked leukopenia with a considerable relative lymphocytosis.

Diagnosis.—It is impossible to make a positive diagnosis of this condition before the appearance of the eruption. The combination of acute onset, high temperature, lack of physical signs, normal urine, leukopenia and relative lymphocytosis is very unusual, however, and should suggest this condition as its explanation.

The diagnosis is easy after the appearance of the eruption. It is true that the eruption is similar to that in German measles and in mild cases of measles. In German measles, however, the temperature is seldom as high and the eruption appears within twelve hours of the beginning of the symptoms. In this condition the temperature falls before the appearance of the eruption. In measles, while there is fever for several days before the appearance of the eruption, it continues during the eruptive period. Furthermore, marked catarrhal symptoms and Koplik's sign are present for several days before the appearance of the eruption. The absence of postcervical adenitis is also of some importance, as these glands are enlarged in both German measles and measles.

Eruptions of this sort sometimes occur from indigestion or anaphylaxis. When they are due to indigestion, there are always other symptoms of indigestion and the symptoms do not disappear with the appearance of the eruption. When they are due to anaphylaxis, the temperature and other symptoms are coincident with the eruption.

Treatment.—The treatment before the eruption appears must necessarily be entirely symptomatic. After the appearance of the eruption the baby is well and no treatment is necessary.

CHICKEN-POX

(VARICELLA)

Etiology.—The causative microorganism of chicken-pox has not been discovered. It probably belongs in the class of the filterable viruses. It is present in the fluid in the vesicles. This has been proved by various successful vaccination experiments and by the establishment of protection

by intravenous injections of the contents of the vesicles. It is probable, however, that contagion does not occur through the skin. It is somewhat doubtful if the scabs are contagious, because the disease is most contagious before the vesicles and scabs have formed. It seems probable that the virus enters through the respiratory tract. How it leaves the body is unknown. It is usually stated that the disease may be transmitted indirectly. So far as I know, however, there is no proof that this happens. Nothing is known as to the habitat or duration of life of the virus outside of the human body.

Chicken-pox is less common during the first three months of life than during the rest of childhood, perhaps because the exposure is less. Almost all children are susceptible to chicken-pox. The susceptibility rapidly diminishes, however, after childhood. It is a rare disease in adults. An attack almost invariably confers lasting immunity.

It is probably contagious for a short time before the appearance of the eruption. It is most contagious at the height of the eruption, before the development of scabs. It is usually considered to be contagious as long as there are any scabs left. If this is true, the contagiousness at this time must be very slight.

Period of Incubation.—The period of incubation varies between ten and twenty-one days, the average being seventeen days.

Symptomatology.—In most instances, the first symptom noted is the appearance of the eruption. This may be preceded, however, for twenty-four hours or so, by slight fever and malaise. In rare instances the onset is acute, with high fever, vomiting and convulsions. This happens most often in infancy. The eruption usually appears first on the face, scalp or trunk. It is characteristic of chicken-pox that the eruption appears in successive crops for four or five days or even a week. The eruption appears first as small red papules. These papules gradually increase in size and are surrounded by a reddened areola. This is seldom more than a quarter of an inch in diameter, but sometimes may be as much as one half inch. After a time the papule becomes veiscular. The vesicle is slightly flattened on top and, as it begins to dry, becomes slightly umbilicated. It is unilocular. The fluid is clear. Sometimes it becomes cloudy, but it is never purulent unless the vesicle is scratched and infected. The vesicle dries up in a few days, forming a scab, which drops off in from one to three weeks. In other instances the initial lesion is a vesicle. When this happens, there is no areola about the vesicle. All stages of the eruption, from the initial papule to the scab, may be seen at the same time on the same patient. Not all the lesions, however, go through all the stages. In most instances no scar is left, unless the lesion has been scratched and secondary inflammation has occurred. The eruption is usually most marked on the face and body. There may not be more than a dozen spots altogether. On the other hand, they may be so profuse as to be confluent.

The eruption also not infrequently occurs in the mouth and throat. It is said that it also develops in the larynx. When it appears in the mouth and throat, the initial lesion is a vesicle. The top quickly comes off, however, leaving a shallow ulcer.

In feeble and poorly nourished babies and young children the eruption may become gangrenous—*varicella gangrenosa.* When this occurs, recovery is very exceptional.

The temperature rarely goes higher than 102° F. and seldom lasts more than three or four days. In many instances there is no fever

at all. In other instances, however, when the eruption is profuse, the temperature may reach as high as 104° F. or even 105° F. The constitutional symptoms are, as a rule, very slight. In cases with high temperature, however, they may be quite marked. Itching is almost always troublesome. Enlargement of the peripheral lymph nodes is not uncommon.

Complications.—The complications of chicken-pox are, in most instances, due to secondary infection. Among them are erysipelas, local abscesses and suppuration of the lymph nodes. Acute nephritis occasionally develops, usually toward the end of the disease. I have never seen any complications in the nervous system, although several varieties have been described.

Diagnosis.—The diagnosis of chicken-pox is usually easy. When seen before the vesicles have developed, it is often very difficult, however, to distinguish the papules from any other pimples. Sometimes scattered lesions of impetigo contagiosa may resemble chicken-pox after the crusts have formed. The history and the distribution of the lesions usually make diagnosis possible. Chicken-pox ought not to be confused with herpes, because herpes always occurs in the distribution of one of the peripheral nerves, while the lesions of chicken-pox are generally distributed. The diagnosis between a severe case of chicken-pox and a mild case of smallpox is sometimes difficult except for an expert, and has, at some time or other, given much worry to every physician. The incubation period of chicken-pox is between two and three weeks, that of smallpox between one and two weeks. The prodromal symptoms in chicken-pox are absent or slight, while they are severe and last for three or four days in smallpox. There are no initial rashes in chicken-pox. They are the rule in smallpox. The eruption quickly becomes vesicular in chicken-pox and never pustular, while the development of the vesicle is slow in smallpox and it always becomes a pustule. The vesicle is unilocular in chicken-pox and multilocular in smallpox. The papules are hard and shotty in smallpox, soft in chicken-pox. The constitutional symptoms are always much more marked in smallpox than in chicken-pox, even when chicken-pox is severe. The temperature is irregular in chicken-pox. In smallpox it rises suddenly, remains high until the papules develop, then falls, to rise again when the pustules form.

Prognosis.—The prognosis in chicken-pox is good, except in the gangrenous form and when there are serious complications from secondary infection.

Treatment.—It has been shown that temporary immunity to chicken-pox can be conferred in a considerable proportion of cases by vaccination on the skin with the contents of the vesicles and by intravenous injections of the contents of the vesicles. It has also been shown that the injection of the blood serum of children convalescing from chicken-pox during the first few days after exposure protects against chicken-pox in the majority of cases and in the others diminishes the severity of the disease. So little work has been done, however, that it is impossible to say, at present, how early or how late the convalescent serum may be taken, or how early it has to be given after exposure to prevent the development of the disease. The dose used is 5 c.cm. Chicken-pox is such a mild disease, however, that it hardly seems necessary to resort to the use of convalescent serum or to the injection of the contents of the vesicles intravenously, except in institutions or when children are very feeble or ill with some other disease, so that it is dangerous for them to have chicken-pox.

Children with chicken-pox should be isolated from the first appearance of symptoms until the scabs have all come off. This period is probably longer than is necessary. The scabs which form after the original scabs have been scratched off, as they often are, are not contagious. Formal disinfection is not necessary. There is no reason why other children in the family, who have had the disease, should be kept out of school. Children who have been exposed to chicken-pox are not dangerous to others for ten days. They are then dangerous until twenty-one days have elapsed from the time of exposure.

Children with chicken-pox should be put to bed and kept there until the temperature is normal and the constitutional symptoms have disappeared. It is perhaps wiser to keep them in bed until crusts have formed on all the lesions. The diet should be regulated according to the severity of the constitutional symptoms, bearing in mind that the child, even if not in bed, is not getting its usual amount of exercise.

The child must be kept from scratching in order to prevent secondary infection and also the formation of pockmarks. In most instances a simple ointment, like zinc oxide or boracic acid ointment, is sufficient to relieve the itching. If the itching is more severe, bathing with equal parts of alcohol and water or a solution of bicarbonate of soda may help. Another useful application is "white wash." The formula for "white wash" is: carbolic acid 0.5 gram, powdered zinc oxide 20 grams, lime water 100 c.cm. If children can not be prevented from scratching in any other way, they must be confined so that they can not. After the scabs have formed, it is wiser not to put any ointment on them. A scab should never be picked off, but allowed to fall off.

The urine should be examined, not only during the course of the disease but also during convalescence.

VACCINIA-VACCINATION

Before the introduction of vaccination, smallpox caused one tenth of all the deaths in the human race. Between 30% and 40% of all the deaths in children under ten years of age were due to smallpox. Eighty per cent. of all the cases of smallpox occurred in children under five years of age. Everyone, with rare exceptions, that survived the disease, was pockmarked and disfigured for life. Two thirds of the inmates of the blind asylums had lost their sight from smallpox.

Vaccinia, or cowpox, is a mild febrile disease of short duration, which is never fatal, has no complications, and leaves no after effects, except one scar on the arm or leg. Vaccinia can be given to human beings by inoculation, that is, by what is commonly known as vaccination. Jenner's statement that vaccination, "duly and efficiently performed, will protect the constitution from subsequent attacks of smallpox as much as that disease itself will" is not now believed to be strictly true. A recent successful vaccination will confer protection, however, against smallpox in at least ninety-nine out of one hundred individuals. After a lapse of five or ten years the susceptibility to smallpox may, in part, return and the individual may require vaccination to reëstablish the immunity. An attack of smallpox does not always, however, protect against a second attack of the disease. Even a single vaccination, however, if it does not always protect against smallpox, so modifies the course of the disease that it is seldom serious and almost never fatal. After a second successful vaccination the individual is, in the vast majority of instances, protected against smallpox for life.

Virus.—The use of bovine virus eliminates the possibility of the transmission of syphilis by vaccination, as cattle do not have syphilis. The killing and examination of the calves after the virus is taken and before it is used excludes the transmission of tuberculosis or other diseases from the calves. The glycerinization of the virus destroys saprophytic bacteria, so that the only organisms present in the virus are those of vaccinia. The preservation and distribution of the virus in sealed capillary tubes prevents the contamination of the virus after it is prepared. There is no basis, therefore, for the statements that vaccination is a dangerous procedure and that it causes other diseases.

Time for Vaccination.—In spite of the fact that, owing to vaccination, smallpox is a rare disease, there are, nevertheless, enough unprotected individuals, who may contract the disease and either intentionally or unintentionally travel about with it and expose others to infection, to make it necessary to protect even babies against it. It is true that the chance that a baby who does not travel in public conveyances will catch smallpox is very small. Nevertheless, there is a chance. This chance is greater, if it does travel in public conveyances. All babies should be vaccinated, therefore, before they are six months old. As the period of protection may not last more than five years, children should be vaccinated again when they are about five years old. It is advisable to repeat the vaccination in the early teens. If there is smallpox about and children have not been vaccinated within five years, they should be vaccinated. If they are immune there is no reaction; if the vaccination "takes," it shows that they are not immune and that the vaccination was necessary.

Unsuccessful Vaccinations.—If a primary vaccination does not "take," it is almost always because the virus is not active or there has been some error in the technic. At least three trials should be made. A good, active vaccine virus will almost always "take" after weaker lymphs have failed. Occasionally, even after repeated trials with active virus, a successful vaccination does not result. These persons, however, sometimes contract severe and even fatal smallpox. The explanation is that such people have a low susceptibility to vaccinia but not necessarily to smallpox.

Method of Vaccination.—Many different methods of vaccination are recommended, some of which, to my mind, are unnecessarily severe. I vaccinate boys at the insertion of the left deltoid and girls on the outer side of the left calf. It does not seem fair to me to make a disfiguring scar on a girl's arm where it will always show, when it is unnecessary. The skin should be first cleaned with soap and water. It should be then washed with alcohol and allowed to dry thoroughly. The superficial layers of the skin should then be scraped off with a sterile knife over an area about one-quarter of an inch in diameter until there is oozing. There should not be any bleeding. It is not necessary to make more than one spot. The vaccine should then be squirted on and rubbed in with the sterile knife. It should be allowed to dry thoroughly. This is very important, as the virus may be rubbed off unless it has thoroughly dried. A sterile gauze dressing should then be applied and fixed with two narrow strips of adhesive plaster. A bandage should be applied over this dressing. If the bandage becomes loosened, it should be reapplied as often as necessary. The dressing must not be allowed to get wet. It should be removed and the vaccination looked at at the end of a week. If it has taken, the dressing should be reapplied. If it has not taken, the

vaccination should be repeated. A shield should never be used under any conditions. It causes congestion about the vaccination area and this predisposes to infection. It does not prevent the access of bacteria to the wound. Care must be taken not to apply the dressing too tightly. The vaccinated area should be kept covered until the scab comes off.

If vaccination is done in this way and the wound kept covered and protected, there are no complications. Complications, such as suppuration, cellulitis and erysipelas, are always due to secondary infection. This almost invariably occurs after the vaccination has been done and is due to lack of proper care and protection. The vaccinated spot, like any other open wound, is liable to infection. If treated like an open wound, infection does not take place.

Course of Vaccination.—A small, reddish blood-crust forms at the site of the abrasion immediately after vaccination is performed. About the fourth day the inoculated area becomes red and slightly raised. About the sixth day a pearl-colored vesicle forms. This contains a clear fluid. The vaccination reaches its height on the eighth or ninth day. There is a flat vesicle with a dark central depression. Around the vesicle there is a zone of redness with some swelling. At about this time the contents of the vesicle become creamy or purulent. The glands in the axilla or groin are somewhat enlarged and tender. The vesicle gradually becomes darker and dryer, until it is converted into a hard, mahogany-colored scab. This usually does not come off before three weeks and often not for five or six weeks. Great care should be taken to prevent it from being knocked off, as it is the best protection there is for the vaccination site.

At times a smooth or slightly elevated red area, having an areola, develops instead of the vesicle. This is known as the "raspberry excrescence." It is not accompanied by constitutional symptoms. It usually persists for two or three weeks and then gradually disappears, leaving no scar. It is presumably the result of a feeble virus and gives little or no protection. A similar appearance is seen not infrequently in secondary vaccinations.

There are usually some constitutional symptoms for three or four days at the height of the vaccination, that is, at about the end of the first week after the vaccination is done. The temperature usually does not go over 101° F. There is often also, in addition to tenderness in the axilla or groin from inflammation in the glands, some itching or discomfort in the arm or leg. The constitutional symptoms are usually less marked in young babies. They may occasionally be more marked, however, in some instances in childhood. The temperature may reach as high as 104° F. or even 105° F. There may be considerable swelling and redness about the site of the vaccination. In very rare instances a generalized eruption of vaccine vesicles may occur. I have seen several cases of this sort. This condition should not be confused with that which results when the child transfers the infection from the site of the vaccination to other areas with its fingers. Erythematous or urticarial eruptions occasionally appear on other parts of the body. None of the diseases or accidents which occur during the next ten years after vaccination are due to the vaccination. It is not responsible for any criminal or bovine characteristics which may develop in the child later.

SECTION IX

DISEASES OF THE UPPER RESPIRATORY TRACT AND ITS ADNEXA

ADENOIDS

There is normally at birth and during infancy and early childhood lymphoid tissue in the nasopharynx. This tissue is often spoken of as Luschka's tonsil and differs from the faucial tonsils in that it does not contain follicles. When there is an overgrowth of this tissue it is commonly spoken of as an adenoid or adenoids. If the overgrowth is chiefly of the lymphoid elements, as it usually is in infancy, the growth is soft and spongy. If there is considerable increase in the connective tissue the growth is hard, firm and well-defined. The natural course of adenoids is to increase to a certain size and then to remain stationary without further growth. During late childhood, and not infrequently much earlier, atrophy of the lymphoid tissue takes place with consequent shrinkage of the mass. Even if there is but little diminution in the size of the adenoids, the symptoms which they produce are likely to diminish as the child grows older, because of the increase in the size of the nasopharynx with growth.

There is probably enough overgrowth of this normal lymphoid tissue in the nasopharynx to justify the designation of adenoids in 90% of the infants and young children living in the eastern part of this country. This fact does not imply, however, that the adenoids should be removed from all of these children. They should be removed only when they cause symptoms. They may be large enough at birth to cause marked symptoms or begin to cause them at any time thereafter. If, however, they have not caused any trouble before the child is four years old, they seldom do later. They are equally common in all classes of society. Heredity is not of much importance in their etiology. The damp, changeable climate of the eastern coast is probably the chief cause of their frequency in this part of the country.

Symptomatology.—The symptomatology of adenoids is somewhat different in infancy from what it is in childhood. This is because of the anatomical peculiarities of the nasopharynx and its adnexa at this age. The nasopharynx is very low at birth, but is relatively long from before backward, the distance from the back of the hard palate to the soft parts of the back of the pharynx being nearly as great at birth as in the adult. The nasopharynx at birth is, therefore, merely a narrow passage running obliquely backward and downward from the constricted opening of the posterior nares. The nasal cavity is relatively long and shallow at birth and the respiratory portion is very narrow. The whole opening of the posterior nares on either side is just large enough to admit the end of a medium-sized male catheter. The nasal cavity, and with it the height of the nasopharynx, begins to increase in height directly after birth, increasing rather rapidly during the first six months, but very slowly during the rest of infancy. The adult relations are usually attained at about seven years.

On account of these peculiar anatomical conditions, a moderate amount of adenoids at birth and in the first few months of life may cause so much interference with sucking and respiration as to produce very marked disturbance of nutrition or even death. Obstruction to nasal respiration is a very serious matter in infancy because it necessitates oral respiration, which the young infant performs very imperfectly, especially when asleep. This interference with respiration results in the constant deprivation of a sufficient supply of oxygen, which, in turn, produces a disturbance of nutrition which is uninfluenced by any method of feeding or mode of life. Another serious result of nasal obstruction is the interference with sucking and sometimes with swallowing. The effort of sucking is so great that these babies take only enough food to satisfy the acute pangs of hunger. Lack of food, therefore, also interferes with their nutrition and development. Adenoids in infancy undoubtedly predispose to the development of rickets. If babies have rickets, adenoids tend to increase the deformities of the chest as the result of the interference with the entrance of air which they cause, the soft rhachitic bones yielding in various ways to atmospheric pressure and the pull of the muscles. The most common of these deformities is a retraction at the insertion of the diaphragm. Pigeon breast is also common and retraction of the sternum not at all unusual.

FIG. 100.—Adenoids and depression of sternum.

Adenoids are the most common cause of frequent "colds in the head" in infancy and also of chronic snuffles. In fact, repeated colds in the head and chronic snuffles are very uncommon in infancy unless there are adenoids. Adenoids are often the cause of an irritating cough, especially at night, as the result of mucus running down into the pharynx. They are one of the common causes of sleeplessness and restlessness at night in infancy, even when there are no marked symptoms, like mouth breathing, snuffles or cough. They are often, as in childhood, responsible for enlargement of the cervical glands.

In infancy the Eustachian tubes are nearly horizontal and the opening is at or a little above the level of the hard palate. The tube is not only relatively, but absolutely, wider at its narrowest point during infancy than in the adult. These facts explain the ease with which catarrhal processes travel in infancy from the nasopharynx to the middle ear. Furthermore, a small amount of adenoids, especially if situated laterally, will block the Eustachian tubes. On account of these anatomical peculiarities, adenoids either directly or through the colds which they produce, are undoubtedly the most common cause of otitis media in infancy. In fact, repeated attacks of otitis media are very unusual in infancy unless there are adenoids.

The symptoms of adenoids are somewhat different after infancy as the anatomy of the parts gradually approaches that of adults. Adenoids continue to be, however, the most common cause of frequent colds, chronic snuffles and otitis media. The colds often "go down" and cause laryngitis and bronchitis. Chronic nasal catarrh often develops. As the result of frequent colds and a chronic catarrhal condition of the nasopharynx, the Eustachian tubes become more or less involved and

obstructed, especially if the adenoids are situated laterally, and deafness develops. Irritation of the nasopharynx and the dropping of mucus cause constant or recurrent cough. If the adenoids are so situated as to obstruct nasal respiration, the mouth is kept open. This causes noisy respiration and snoring at night, especially if the child lies on its back. Children with adenoids are often restless at night and have attacks of dyspnoea and night terrors. The voice becomes nasal and the speech thick. They are unable to blow the nose properly. The interference with nasal respiration often caues headache. Nose-bleed is not uncommon. Further evidence of nasal obstruction is the characteristic dull and stupid facies. The bridge of the nose is widened and rounded. The nostrils are small, the naso-labial folds deepened and the lower jaw drops. There is also often a prominent vein at the bridge of the nose, but this may also be due to other conditions. The hard palate is highly arched and the upper jaw narrowed, the front teeth often being pushed forward. The arching of the palate buckles up the vomer, which still further obstructs the nasal passages. This deformity of the upper jaw often prevents proper occlusion of the teeth which, in turn, interferes with the proper mastication of food and causes indigestion.

The constant lack of proper aeration of the lungs and the increased labor in breathing causes disturbance of nutrition and anaemia. The catarrhal condition in the nose and nasopharynx disturbs the appetite and digestion, which further affects the nutrition. Disturbance of the nutrition makes the children dull and stupid. They are backward physically and mentally. The mental dullness is, however, often due, in part, to deafness. One of the signs of imperfect aeration and disturbed nutrition is nocturnal enuresis.

Adenoids almost always increase the local symptoms in the nose and throat in scarlet fever, diphtheria and measles, make the chances of infection of the ears, heart and kidneys greater, render the treatment more difficult and the prognosis more serious. In some instances adenoids seem to be the cause of asthma; at any rate, the attacks of asthma cease after their removal. The probable connection is that with them is removed the focus of bacterial protein sensitization. Chronically infected adenoids are also one of the causes of continued fever in infancy and childhood. Adenoids are also a frequent cause of cervical adenitis in childhood. The glands involved are somewhat below and behind those ordinarily involved in infections of the faucial tonsils. The infection of the glands from adenoids may be tubercular.

Diagnosis.—The diagnosis of adenoids is usually easy from the symptoms and characteristic facies. In all but the most obvious cases, however, and in all cases in which it is possible that adenoids may be the cause of obscure symptoms, their presence or absence should be demonstrated. It is useless for anyone but an expert, and almost useless for him, to attempt to see adenoids with a mirror before children are five or six years old. In most instances, therefore, the diagnosis must be made with the finger.

The baby or child should sit sidewise on a chair or in its mother's lap with its back to the examiner. The mouth is held open with the thumb of the left hand or a gag while the forefinger of the right hand is introduced into the mouth. It is most important that the finger-nail is cut short, that the finger is thoroughly clean, that the finger is carefully introduced and that no violence or injury is caused. There should be no bleeding, if the examination is carefully done.

Treatment.—In general, when adenoids are large enough or so placed that they cause symptoms they should be removed, whatever the age of the patient or the time of year. It is true that if adenoids are removed in infancy they are liable to recur later. In the majority of cases, however, they do not recur. If they do and a second operation is required, the infant is spared in the meantime all the inconveniences and dangers of adenoids and the serious conditions which they bring about. It seems to me that a second operation for adenoids is a trivial matter compared with the disturbances of nutrition, the deformities of the chest, the repeated colds and attacks of bronchitis and bronchopneumonia and the repeated attacks of otitis media with the ever-present menace of mastoiditis, which adenoids in infancy cause. I have had adenoids removed a number of times in babies less than a week old and many times in babies

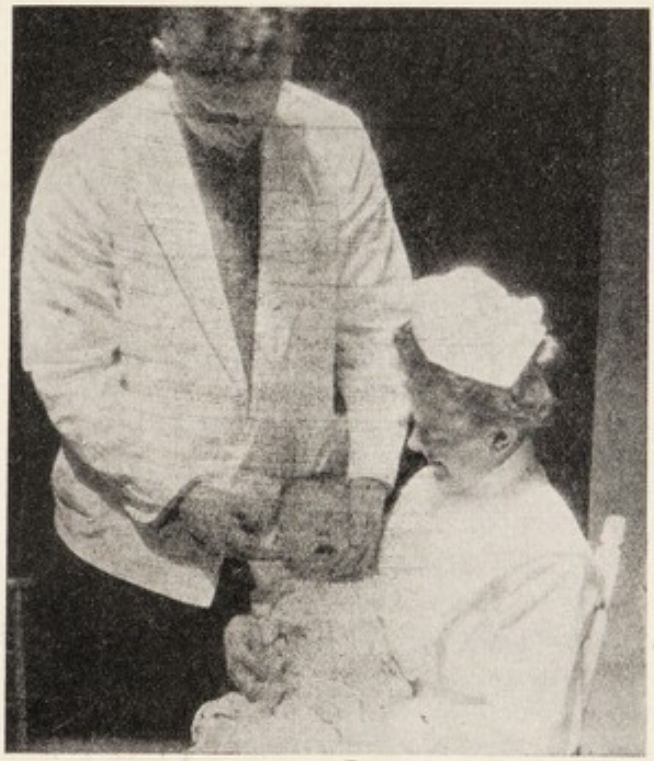

Fig. 101.—Method of examination for adenoids.

a few months old, with the result, I believe, of saving these babies' lives. In general, of course, it is preferable to wait until warm weather before removing adenoids. If the symptoms are at all marked, however, they should be removed at once. In obstinate cases of otitis media it is often advisable to remove the adenoids without waiting for the ears to clear up. In fact, in many instances, they never will clear up unless the adenoids are removed. The operation should not be delayed because the child is running a continued slight fever, because infected adenoids are often the cause of the fever, which will not go down until the adenoids are removed.

I do not think that it is ever justifiable, even in a small baby, to attempt to scrape out the adenoids with the finger-nail. They are never completely removed in this way and the dangers of infection are immensely increased. Furthermore, I do not believe that adenoids should ever be removed without an anaesthetic. The best anaesthetic is ether, which may be, if desired, preceded by nitrous oxide gas or gas-oxygen.

There is ordinarily no danger in the operation for adenoids. It must not be forgotten, however, that there is sometimes a severe secondary hemorrhage and that with a careless operation portions of the adenoids or blood may be inhaled and cause abscess of the lung. It is not at all uncommon, moreover, to have acute otitis media set up as the result of the operation, especially in children that are subject to otitis. Cervical adenitis also sometimes develops. It must not be forgotten, moreover, that the removal of adenoids does not remove the deformities of the hard palate, nose, face and chest which they have caused. In many instances in older children there will be little or no improvement in the breathing and in the facies after the adenoids are removed. In fact, there never will be any improvement, unless the jaws are widened, the hard palate brought into position, and the septum of the nose straightened. Furthermore, it must also be remembered that children will continue to have colds after their adenoids are removed. As a general rule, however, they will not have as many colds, the colds will not be as severe and there will be fewer complications in connection with them. In most instances the improvement in the general condition after the removal of adenoids is most striking and the relief of symptoms almost immediate. It must not be forgotten that when adenoids are removed a raw surface is left in the nasopharynx and that on this account the susceptibility to infection is temporarily increased. It is advisable, therefore, to guard children against exposure and contagion for several days or a week after the operation.

If the adenoids are small and not causing much trouble, it is often advisable, if the patient is a baby or small child to delay operation for a time. In such cases from 3 to 5 drops of the following preparation in each nostril 2 or 3 times daily sometimes seems to reduce the size of the adenoids, or, at any rate, to diminish the symptoms.

Iodine	gr. $\frac{1}{8}$–gr. $\frac{1}{4}$
Camphor	gr. $\frac{1}{4}$–gr. $\frac{1}{2}$
Menthol	gr. $\frac{1}{4}$–gr. $\frac{1}{2}$
Benzoinol or liquid albolene	ʒi

ACUTE NASOPHARYNGITIS

Nasopharyngitis is synonymous with the common "cold in the head." The pharynx, nasopharynx and nose are usually all more or less involved, although the stress of the disease may fall on any one. In infancy and early childhood, however, the most severe infections are in the nasopharynx. Acute nasopharyngitis in childhood, as in adult life, is usually merely a disagreeable incident, but in infancy it may be serious or even fatal. The complications of acute nasopharyngitis are serious at any age.

Etiology.—The inflammation of the mucous membranes is due primarily to microörganisms, staphylococci, streptococci, the pneumococcus, the influenza bacillus, the micrococcus catarrhalis, and perhaps to some as yet unknown specific organism. The contagiousness of colds and their occurrence in epidemics is strong evidence in favor of their being due to microörganisms. There is, however, almost always some reason why these microörganisms, some or all of which are constantly present in the respiratory passages, are able to gain a foothold. Many children and families are especially susceptible to colds. This susceptibility may, perhaps, be due to heredity, but is more often due to the mode of life, which lowers the resistance of the mucous membranes to infection.

Overheated houses, dryness of the air and lack of fresh, outdoor air, both by day and at night, predispose to infection. So does an excess of clothing, which makes the children perspire easily and consequently makes it easy for them to get chilled. In other instances children do not wear clothing enough. Parents often do not realize that it is necessary to regulate the clothing which children wear to their surroundings. The resistance of the mucous membranes is also undoubtedly lowered by living in damp, changeable climates. In many instances a direct connection seems evident between wetting the feet, sitting in drafts and exposure to cold, especially if it is windy and dusty. Presumably, in one way or another, these things change the blood supply to the mucous membranes or irritate them so that their resistance to microörganisms is temporarily much diminished.

Pathology.—In the beginning of a cold the mucous membranes are dry and swollen. After a few hours or one or two days the swelling becomes more pronounced and there is a profuse, watery secretion. As time goes on, the secretion contains more mucous, then becomes mucopurulent, and finally purulent. The swelling of the mucous membranes usually diminishes as the discharge becomes thicker. The usual duration of the pathologic process is about a week, but it is not uncommon for the swelling and purulent discharge to last for several weeks.

Symptomatology and Complications.—The nose in infancy is relatively small and the respiratory portion very small. The height of the posterior nares at birth is from 6 to 7 mm. and the breadth between the pterygoid processes at the hard palate 9 mm. The nasal cavity consists of an upper olfactory region occupying the ethmoidal portion of the cavity, and a lower respiratory region occupying the maxillary part. The nasal cavity at birth is relatively long and shallow and the respiratory portion is very narrow. The whole opening of the posterior nares on either side is just large enough to admit the end of a medium-sized male catheter. The nasal cavity begins to increase in height directly after birth, increasing quite rapidly during the first six months, but very slowly during the rest of infancy. The size of the posterior opening doubles in six months, after which it remains stationary until the end of the second year. At the end of the seventh month the nasal cavity begins to approach the adult shape, though it is still relatively broad. The nasopharynx is very low at birth, but is relatively long from before backward. The height of the nasopharynx increases in the same way as that of the posterior nares. The Eustachian tube is nearly horizontal at birth and opens at the level of the hard palate. It is not only relatively, but absolutely, wider at its narrowest point at birth and during infancy than it is in the adult. The connective tissue between the pharynx and the spine is very lax. The nasopharynx is extremely vascular and there is an abundant supply of lymphatic glands and vessels, especially in the posterior wall.

Owing to the anatomical peculiarities just mentioned, a comparatively slight swelling of the nasal mucous membrane in infancy completely closes the nose and entirely prevents nasal respiration. This, of course, necessitates oral respiration, which the young infant performs very imperfectly, especially when asleep. Sleep is so broken in many instances, that the baby gets little, if any, rest and, on this account, loses strength very rapidly. The occlusion of the nares also prevents proper sucking and interferes with swallowing, even when the food is given with a spoon or dropper. Consequently, the baby takes but little food or refuses it

entirely, even if very hungry. The temperature is always elevated, usually irregular and often very high. Loss of weight and strength is very rapid as the result of the insufficient supply of fresh air, lack of sleep and deprivation of food, and in feeble babies may be so great as to cause death.

On account of the anatomical peculiarities of the nasopharynx, *otitis media* is a very common complication of nasopharyngitis in infancy. Extension to the ethmoid cells is also common and in many instances accounts for the long persistence of colds in babies. The antra are also often involved, but on account of their small size this extension is not usually important. The frontal sinuses not being present at this age are, of course, not involved. There is also a marked tendency for nasopharyngitis in infancy to extend downward, causing laryngitis, bronchitis and bronchopneumonia. Cervical adenitis is common and retropharyngeal abscess not very unusual.

The anatomical relations in the nose and nasopharynx gradually change as the baby grows older, so that by the time the child is seven years old the relations are essentially the same as in the adult. The symptomatology of acute nasopharyngitis also gradually changes during early childhood, so that by middle childhood it is the same as in adults. It hardly seems necessary to describe it.

As the child grows older, it is progressively less likely to have otitis media and ethmoiditis as complications. On the other hand, infections of the antra and, after ten years, of the frontal sinuses are more common. The young child is just as likely to have cervical adenitis as the baby, but this tendency diminishes after ten years. Retropharyngeal abscess is very uncommon after three years. Endocarditis and nephritis become progressively more common as the child grows older. Nasopharyngitis is at all ages the most common cause of laryngitis and bronchitis.

Diagnosis.—It is easy enough to know that a baby or child has acute nasopharyngitis. It must not be forgotten, however, that an acute nasopharyngitis may be the prodromal stage of either measles or whooping-cough. Nasal diphtheria may also sometimes come on acutely with all the symptoms of an ordinary cold. It also should, therefore, always be borne in mind. It is most important not to be satisfied with a diagnosis of acute nasopharyngitis, because complications are liable to occur at any time, especially in infancy. Constant watch should, therefore, be kept for complications, especially in the ears and ethmoid cells.

Treatment.—The most important part of the treatment of common colds is the preventive. This consists in careful regulation of the life of the child. The house must be kept at an even temperature, neither too hot nor too cold; 68° F. to 70° F. is about right. The air must also in some way be kept moist. Both babies and children should have fresh air, both by day and at night. No one ever caught cold by exposure to the night air. When babies and young children have a susceptible mucous membrane, however, it is usually wise to keep them indoors on very windy, dusty and foggy days. Cold sponging of the chest or of the whole body is said to prevent colds. I doubt very much if it has any such action. In any event, children should not be sponged with cold water, unless the bathroom is hot and they always react well. The clothing must be regulated to the temperature of the house in which the child lives. If the house is kept hot, the child should wear thin clothes indoors and put on heavy clothes when it goes out. If it lives in a cold house, it should wear heavy clothes indoors and needs but few extra ones when it goes out.

The clothing should be so regulated that the child does not have to perspire every time it exerts itself and also so that it is not cold. Children must be protected against contagion by separation from other members of the family who have colds. Adenoids should be removed, especially if colds are frequent. Change to a more suitable climate will usually prevent colds, but is, of course, not ordinarily feasible or necessary.

A baby will, of course, be in bed anyway. A young child should be put to bed as soon as it develops a cold. This may seem unnecessary, but in most instances it will be found that time is gained and complications avoided by putting the child to bed for a day or two in the beginning. It will usually lose less outdoor air and less school in this way than if it is allowed to be up and about. Children with colds should be separated from the rest of the family. They ought not, in any event, to be allowed to go to school, to public places or in public conveyances. The room should be kept at an even temperature of about 64° F. if they are in bed, and of from 68° F. to 70° F., if they are up and about. The air should be kept fresh.

A baby that is on the breast, should be kept on it. If it is on an artificial food, the food should be weakened. Many babies are unable to nurse and have to be fed with a spoon, dropper or through a stomach tube. The child should at first be given liquids and soft solids. Later, it may have a more general diet, depending on its temperature and appetite.

The old custom of giving a cathartic at the beginning of a cold has nothing to justify it. By the time a cold is recognizable it is too late to expect to diminish the congestion of the mucous membrane of the nose by catharsis. It is doubtful if catharsis would do any good, anyway, if it was known in advance that the child was to have a cold. It is advisable, however, on general principles, to see that the bowels are open.

There are no drugs which, taken internally, have any effect on the progress of a cold in the head. Certain drugs, like belladonna, may, however, by drying up the secretions, make the patient temporarily somewhat more comfortable. Certain of the symptoms of a cold, such as fever, aches and pains, can be relieved by drugs like aspirin and the combination of phenacetin and salol.

Vaccines are contraindicated in acute infections. If a vaccine is to be used, it should, of course, be an autogenous and not a stock vaccine. If a cold persists or a child has repeated colds, it is possible that in some cases autogenous vaccines may be helpful.

Local treatment is more important. The only way in which applications can be made to the mucous membrane of the nose and nasopharynx in babies is by putting the solution into the nose with a medicine dropper. The baby should be laid on its back and the solution squirted into the nose with a medicine dropper, so that it can run through the nose and into the nasopharynx. From three to five drops in each nostril is sufficient in babies. A 5% aqueous solution of argyrol, used every two or three hours, is helpful in the acute stage. It should not be used too long, however, as it keeps up the irritation. Liquid albolene may be used at intervals of one or more hours whenever the nares are occluded. Another preparation, which is especially useful after the very acute stage is passed, is the following:

Iodine	gr. $\frac{1}{8}$
Camphor	gr. $\frac{1}{4}$
Menthol	gr. $\frac{1}{4}$
Benzoinol	$\mathfrak{M}$ 1

This should not be used more than three times daily. If there is much obstruction to nasal respiration, a solution of epinephrine, of the strength of from 1–1000 to 1–5000, may be used as necessary. The oily preparations of epinephrine are better than the watery, because they stay longer in contact with the mucous membrane. A good one is Parke, Davis & Company's adrenalin inhalant, which is a 1–1000 solution of adrenalin chloride with 3% of chloretone. In some of the severest cases it may be necessary to put a small catheter in each nostril for the babies to breathe through. I have seen an occasional case saved in this way. The upper lip should be protected by vaseline or boracic acid ointment. It is inadvisable to use washes or douches of any of the saline solutions in either the nose or throat, because they are likely to cause trouble in the ears. It is important to blow the nose as well as is possible and to keep the anterior nares clear.

The local treatment in children is along the same lines as that in infants. A 10% aqueous solution of argyrol may be used, a combination of camphor and menthol, one half grain of each, with albolene, instead of plain albolene, or the iodine, camphor and menthol solution made twice as strong. The oily preparations should be used in an oil atomizer. Alkaline sprays, such as Seiler's solution or the liquor antisepticus alkalinus, may also be used in the nose and throat. No douches should be used and nothing should be snuffed up in the nose, because of the danger of causing complications in the ears. Children should be made to blow their noses and not be allowed to snuff and sniff.

Both babies and children may have a constant and useless cough from irritation of the pharynx. Cracked ice to suck, slippery elm lozenges or gelatin tablets will help with children. The steam from a dish of boiling water, to which compound tincture of benzoin has been added, if the water is kept boiling constantly in the room, will help this cough, both in infants and children. If these methods are ineffectual, the bromides or paregoric, in doses suitable for the age, will have to be used.

CHRONIC NASOPHARYNGITIS

Chronic nasopharyngitis is more often spoken of as chronic rhinitis. Chronic nasopharyngitis seems to be the better term, however, as the trouble in the nose is almost always secondary to adenoids and there is generally a chronic catarrhal condition as well. This condition is far more common in infancy and early childhood than later. There is a more or less continuous mucopurulent or purulent nasal discharge, the nares are often inflamed and crusted and the upper lip irritated. There is also more or less obstruction to nasal respiration with the resulting interference with respiration and sucking and the disturbances of nutrition secondary to them. There is quite likely to be a troublesome cough as the result of the irritation from mucus coming down the pharynx from above. A complicating otitis media or ethmoiditis is not uncommon.

Diagnosis.—There is, of course, no difficulty in recognizing that a baby or young child has chronic rhinitis. It must not be assumed, however, that this is necessarily a simple rhinitis, because it may be either diphtheritic or syphilitic. Chronic *diphtheritic rhinitis* is far more common than is generally supposed and is very often the source of epidemics and of apparently sporadic cases of faucial or laryngeal diphtheria. The symptoms are, as a rule, no more severe than those of simple rhinitis. The nasal discharge, however, is usually thin, watery and acrid. Con-

trary to the usual statements, it is seldom bloody. It frequently irritates the upper lip, causing crusts about the nares and on the lip. The discharge in simple rhinitis is thicker, more purulent and less irritating. Whenever the nasal discharge is thin and irritating, it should be looked on with suspicion and a culture taken. There is usually no visible membrane in nasal diphtheria. *Syphilitic rhinitis* begins in the first few weeks of life and persists with increasing severity. The nasal discharge is usually thick and purulent, often bloody, and very irritating to the skin. The nares and upper lip are much irritated and crusted. There are quite likely to be fissures of the lips and mucous patches on the lips or in the mouth. Other evidences of syphilis can almost always be found after a few weeks.

Prognosis and Treatment.—Chronic nasopharyngitis is quite likely to persist, in spite of treatment, until the usual cause, adenoids, is removed. It then ordinarily yields promptly to treatment. If adenoids are not the cause, the prognosis is not as good and recovery is slow. The treatment is primarily the removal of adenoids, if they are present. Otherwise, it is along the same lines as that of acute nasopharyngitis.

INFECTIONS OF THE ETHMOID CELLS, FRONTAL SINUSES AND MAXILLARY ANTRA

The ethmoid area is present at birth, but the development into distinct cells does not usually begin until the fourth year and their development is not well marked until some years later. Nevertheless, infection in the ethmoid region is not at all uncommon in infancy and early childhood in the course of nasopharyngitis. It is also responsible for many long-continued catarrhal conditions and recurrent infections in the nose. It should always be thought of when there is a long-continued nasal discharge, especially if it is unilateral. Ethmoiditis is also one of the causes of infection in and about the orbit.

The frontal sinuses are sometimes present as early as the fifth year, but do not usually amount to much before seven or eight years. They do not have to be taken into consideration, therefore, before middle childhood and even then they are not likely to become seriously involved.

The maxillary antra are present at birth, but are small, triangular in shape and somewhat flattened. They do not change much in shape or increase very much in size until the second dentition. Although small, they may become infected at any age and, like the ethmoid cells, may be a source of recurrent infections of the nose or of a long-continued unilateral discharge. An infection of the antrum may be at the bottom of a chronic infectious arthritis or perhaps, through protein sensitization, the cause of some cases of asthma.

Whenever infants and children have a long-continued discharge from the nose, recurrent attacks of acute nasopharyngitis, asthma or chronic infectious arthritis, disease of the nasal adnexa should always be borne in mind as the possible cause. The diagnosis can usually only be made by a specialist, who often needs the help of a radiologist. The treatment of infections of these sinuses should, if possible, be placed in the hands of a specialist.

RETROPHARYNGEAL ABSCESS

Retropharyngeal abscesses in infancy are usually secondary to nasopharyngitis. In childhood they are almost always due to disease of the vertebrae or to the direct extension of an abscess from some

neighboring part. The greatest number of cases occur between four months and one year.

The retropharyngeal lymph nodes form a chain on both sides of the median line of the pharynx, extending from its upper portion to its junction with the esophagus. They lie between the prevertebral aponeurosis and the muscles of the pharynx. They drain the cavities of the cranium, pharynx, nose and middle ear. They are said to grow smaller after the third year and in some instances to disappear entirely.

Enlargement of these lymph nodes is called retropharyngeal adenitis; suppuration, retropharyngeal abscess. In infancy both adenitis and abscess are secondary to some inflammatory process in one of the cavities drained by these nodes. Several nodes are usually involved in simple adenitis, but suppuration rarely takes place in more than one. Suppurative inflammation is accompanied by a cellulitis of the surrounding tissues. The causative organism is usually the streptococcus, but may be any of the pus-forming organisms or even the tubercle bacillus.

Symptomatology.—The symptoms of retropharyngeal adenitis are not characteristic, being very similar to those of nasopharyngitis. Inspection shows nothing definite in the throat, but the enlarged nodules can be easily felt by the finger. It is probable that these nodes frequently resolve, without breaking down. Suppuration, when it occurs, usually develops in from five to six days. Originating, as it does, in a single gland, situated to one side of the median line, the abscess is in the lateral wall rather than in the back of the pharynx. When suppuration begins the temperature usually rises and becomes more irregular. The first symptom directly due to the abscess which is usually noted is unwillingness to take food or difficulty in swallowing. In the beginning this is probably due to pain; later to the mechanical obstruction from the tumor. Modification of the voice and difficulty in respiration next develop. These vary according to the location of the tumor. If the abscess is in the upper portion of the pharynx, the cry is nasal and nasal respiration is chiefly disturbed. The mouth is kept open and the breathing is snoring and snuffling. If the abscess is low down, the respiration is stridulous, like that in laryngeal spasm or stenosis. The respiratory murmur is diminished and many moist râles are often heard. These may be due to a complicating bronchitis, but are usually the result of the interference with respiration. In acute cases the head is held in a most characteristic position from the first, the neck being extended and the head drawn to one side. In the subacute cases this may not appear for some time. Swelling of the lymph nodes at the angle of the jaw on the affected side soon develops and is often very marked.

Diagnosis.—When the abscess is high up, the symptoms are often mistaken for those of a simple nasopharyngitis, although diphtheritic paralysis is sometimes suspected. If the obstruction is low down, the symptoms are often ascribed to laryngitis, either catarrhal or diphtheritic. Sometimes the throat condition is entirely overlooked and, on account of the signs in the lungs, mistaken for bronchitis or bronchopneumonia. A retropharyngeal abscess may be overlooked if inspection of the throat is trusted to entirely. The tumor is visible in the majority of cases, but not infrequently, especially if low down, it cannot be seen. It can always be felt, although if it is situated low down, the finger must be introduced very deeply. In no case, therefore, in which the symptoms in any way suggest a retropharyngeal abscess, should palpation of the throat be omitted. It causes the infant only temporary discomfort and

may prevent very serious mistakes in diagnosis. The swelling is usually situated on the side of the pharynx, but may be at any level. It is either tense and elastic or fluctuant. The examination of the throat should be made with a tongue depressor and not with a gag, because the introduction of a gag and the consequent spreading of the jaws may cause sudden death, presumably as the result of pressure on the vagus.

The diagnosis is easier in childhood, because there is usually definite disease of the vertebrae or an abscess in the neck which calls attention to the condition in the throat. In childhood the diagnosis can usually be made by inspection, and palpation is unnecessary.

Prognosis.—In untreated cases the usual termination is death. This may occur slowly as the result of progressive weakness or asphyxia, but more often suddenly from laryngeal spasm or from some disturbance of the function of the pneumogastric nerve. The abscess seldom opens spontaneously. If it does, death may result from suffocation or from a secondary inspiration pneumonia. The pus may burrow in various directions. In some instances the carotid artery has been opened. In others the abscess has opened externally or has discharged through the middle ear.

When the abscess is opened as it should be, the mortality is only about 5%.

Treatment.—It is doubtful if either cold or hot applications to the neck have any effect on the progress of either retropharyngeal adenitis or abscess. There is no direct treatment for adenitis. An abscess should be opened as soon as the evidences of suppuration are definite.

The abscess may be opened by either an external or an internal incision. The external operation gives better drainage and seldom needs to be repeated. It is, however, a delicate operation and requires a skilful surgeon and assistants, while the internal incision can be done by any physician at any time without skilled assistance. Moreover, the external operation requires dressing and leaves a scar, while the internal calls for nothing but cleansing of the mouth and the occasional expression of the contents of the abscess with the finger. It is true that the internal incision affords less efficient drainage, does occasionally close up and offers more opportunity for secondary infection. Nevertheless, practically, drainage is reasonably efficient, it is not difficult to open the abscess a second time, if necessary, and secondary infection is most unusual.

The incision is best performed with the infant in the upright position. If it is tipped forward the instant that the incision is made, there is no danger of the pus entering the air passages. The knife should be guarded, except at the point. A gag should not be employed, because of the danger of sudden death from its use, but the mouth held open with the finger or a tongue depressor. The abscess should be squeezed once or twice a day with the finger in order to keep up drainage and to prevent the opening from closing. If drainage is not free, the abscess should be incised again. It is advisable to wash out the mouth from time to time with some mild alkaline solution.

OTITIS MEDIA

Acute inflammation of the middle ear is very common in infancy and early childhood, but becomes progressively less common after five or six years. In spite of the frequency of its occurrence, its symptomatology is not well understood and it is often overlooked by many practitioners.

Etiology.—Otitis media is, of course, caused by microörganisms, these being, as a rule, the ordinary pus cocci or the influenza bacillus. The infection is generally due in the beginning to a single organism, but after the drum has been perforated it is almost always multiple. Infection does not take place, however, unless there is a catarrhal process in the nasopharynx or the Eustachian tube becomes blocked. The peculiar anatomical conditions in infancy favor the entrance of bacteria into the middle ear, the Eustachian tube being nearly horizontal at birth, the opening at the level of the hard palate and the tube being not only relatively, but absolutely, wider at its narrowest point at birth and during infancy than in the adult. During the latter part of the first year the opening of the tube moves slightly upward and it becomes more slanting, but the adult relations are not attained until about six or seven years. Adenoids are the most common cause of repeated colds in infancy and childhood and, furthermore, keep up a chronic catarrhal condition of the nasopharynx. They are, therefore, the most common cause of otitis media.

Symptomatology.—The symptomatology of acute otitis media in infancy is so variable that it is very difficult to describe it. The one almost constant symptom is fever, but the temperature, while often high, may be but moderately or only slightly elevated. It is usually irregular. It may rise rapidly or slowly. Babies with otitis media seldom cry hard as if they had a severe pain. They are restless and sleepless, moan and act uncomfortable and frequently cry out sharply, but they do not often cry long and hard. Babies are unable to localize pain and almost never put their hands to their ears when they have otitis media. They are far more likely to rub their noses or put their fingers in their mouths. Moreover, babies sick from any cause are very likely to wave their arms about, grab at their ears and pull their hair. Tenderness over the mastoid is unusual in otitis media and it is almost impossible to determine in infancy. No reliance can, therefore, be placed upon it. Reflex nervous symptoms are, however, very common. Among the more common are rapid respiration, vomiting, and tenderness and rigidity of the neck. Convulsions are not very infrequent. On account of these reflex symptoms, almost every disease but the right one is suspected, among which may be mentioned meningitis, pneumonia, gastritis, worms and dentition. In many instances there are no symptoms pointing to the ears, the first sign of trouble being a discharge. Otitis should, therefore, be thought of and looked for whenever a baby that has a cold has a higher temperature or seems sicker than it would be expected to be, whenever in the course of any disease a baby has symptoms hard to explain and whenever a baby is sick and no satisfactory cause for its illness is apparent. In fact, it is always advisable to examine the ears when a baby is ill. If they are examined, many awkward mistakes will be avoided. Babies with acute otitis media are very likely to refuse to take food, presumably because it hurts to swallow.

As the child grows older pain becomes a more marked symptom and the child is better able to localize it. Many children cannot localize pain until they are four or five years old, however, and I have known a boy to say that he had a pain in the knee when he really had an earache. Fever continues to be an almost constant symptom. Tenderness over the mastoid is uncommon in uncomplicated cases. Reflex symptoms become less marked as children grow older and the symptoms more characteristic. It is not safe to omit the examination of the ears even in older

children, however, unless the findings elsewhere account for all the symptoms.

The course of otitis media in infancy and early childhood is very variable. If the exudation is serous and the Eustachian tube soon opens up again, the symptoms, which may be mild or very marked, according to the amount of the exudation, stop at once. They may or may not recur. If the exudation is serous and the drum is perforated, either from within or from without, the symptoms may cease at once and the drum heal quickly, provided the Eustachian tube opens and the exudation remains serous. On the other hand, the exudation may become purulent and persist for several weeks. In other cases the exudation is mucopurulent or purulent from the first. It is possible that, even then, if the Eustachian tube opens quickly enough, the drainage will be sufficient to prevent perforation of the drum. Usually, however, the drum is perforated from within, unless it is opened. The discharge in such cases continues purulent. It may persist for many weeks if there are adenoids. A very marked characteristic of otitis media in infancy and childhood is the speed with which the symptoms and the appearance of the drum membrane may vary. A condition which seems likely to require operation will clear up in a few hours and another which looks very mild will suddenly become acute and in an hour or two demand immediate operation. Polynuclear leucocytosis is a constant accompaniment of otitis. It is often quite marked. It has never seemed to me that it is of much importance in either diagnosis or prognosis, or as an indication for treatment.

Otitis media is found very constantly at autopsies on infants dead of chronic disturbances of nutrition and at those on infants and young children dead of acute illnesses. In the first instance the infection of the ears is always of a low grade, is due to the lowered resistance and has probably played no part in the symptomatology or the course of the disease. In the second instance the infection is terminal and unimportant.

Chronic otitis is almost invariably secondary to acute otitis and is kept up by adenoids or a chronic catarrhal condition of the nasopharynx from some other cause. It is sometimes kept up by a low-grade mastoiditis. In other instances it is tuberculous. The possibility that chronic otitis media may be tuberculous should never be overlooked.

Examination of the Ears.—The only way to determine whether there is inflammation of the middle ear or not is to examine the ears with a speculum. A smaller speculum than comes with most sets is necessary. A head mirror and speculum may be used or one of the electric otoscopes. The external auditory canal, especially in babies and young children, is often wholly or partially blocked with wax, which must be carefully picked out with forceps or removed with a probe or toothpick wrapped with cotton. The examination of infants' ears is not a simple matter and requires considerable experience. The results, however, are well worth the time required to master the technic. The external auditory canal at birth and during infancy runs inward and downward and the membrana tympani is almost horizontal. In introducing the aural speculum, the ear must be drawn forward and downward instead of upward and backward or the canal will be bent on itself. The course of the canal and the position of the drum gradually approach and finally reach those of the adult at from five to seven years, when the speculum is introduced in the same way as in the adult. Great care must be taken

in infancy not to mistake the posterior wall of the canal for the drum. Swelling of the canal or an abscess of the canal often interfere with the examination.

Treatment.—If the drum appears normal or there is merely a little injection along the handle of the malleolus or around the periphery, dry heat should be applied externally and the nasopharynx treated. If there is earache, a few drops of a 1–1000 aqueous solution of one of the preparations of epinephrine or of a mixture of from 5 to 10 minims of phenol in an ounce of glycerine should be put in the ear. It is not advisable to put laudanum or any oily preparations in the ear. The epinephrine solution may be repeated frequently; the phenol and glycerine mixture should not be used oftener than once in three or four hours. From five to twenty grains of one of the bromides, according to the age of the patient, should be given and repeated every one or more hours, as necessary. In some cases the constitutional symptoms may be marked and the temperature high for some days without any more, or even as marked, signs in the drum than these.

If the drum is generally reddened, but the landmarks are distinct, or even if there is a little fullness of Shrapnell's membrane, it is usually safe to temporize and to try local treatment. If, however, the temperature is high and the pain severe, it is wiser to operate. If there is much bulging of Shrapnell's membrane, if there is much bulging of the drum as a whole or if there is a localized nipple-like bulge, the drum should be incised. If the drum appears thickened and hazy or if it is yellowish, it should be incised, as with these appearances there is usually a thick mucous, mucopurulent or purulent exudation in the tympanic cavity. An acute inflammation of the outer surface of the drum—*myringitis*—sometimes results in the formation of a bulla, which gives an appearance much like that of a bulging drum. Care must also be taken not to mistake a swelling of the posterior wall of the external auditory canal from local inflammation for a bulging drum. If the swelling of the drum is only of Shrapnell's membrane or is sharply localized, it is sometimes allowable to make a small incision at the point of swelling. In general, however, a long incision should be made, beginning in the lower portion and curving upward and backward behind the malleolus. It is hard to make the incision too long, it is very easy to make it too short. It takes no longer for a long incision to heal than for a short. It gives much better drainage and there is much less chance that it will close too quickly. A paracentesis knife should always be used, never a needle. In my opinion it is wiser in babies and in children young enough so that they can be firmly held to open the ears without an anaesthetic. It is true that it is very painful to have the drum incised in this way, but the child is no more frightened than it is by taking an anaesthetic. The whole procedure takes much less time and it is all over at once, there being none of the discomfort incident to coming out of an anaesthetic. In older children, it is wiser to use an anaesthetic.

When the discharge is serous, the external auditory canal should be wiped dry with pledgets of sterile cotton, using either forceps or a probe or a toothpick, and a drain of sterile cotton put in the canal. These drains should be changed frequently and the canal cleaned and wiped *dry* every few hours, the frequency depending on the amount of the discharge. The external ear and the skin about it should be protected with boracic acid ointment. When the discharge is thick or purulent, the treatment immediately after opening is the same, but after this it is wiser to wash out the ears every two or more hours, according to the amount of the dis-

charge, with sterile water, sterile physiologic salt solution or a 2% solution of boracic acid at a temperature of from 100° F. to 110° F. Sterile water is as good as anything. A soft rubber ear syringe should be used and the washing continued until the wash water returns clear. No force should be used. The canal should then be *thoroughly dried* and a drain introduced. The external ear and the skin about it should be protected with boracic acid ointment. I am well aware that many otologists and pediatrists, both "wipers" and "washers," will disagree with the treatment just outlined. It seems to me, however, more rational than to treat all cases in the same way and to always "wipe" or always "wash."

Physicians are quite likely to expect and to promise too great and too rapid improvement after the drum is incised. It is true that in some instances, when the discharge is serous, the temperature will almost immediately drop to normal, the discharge cease in a day or two and the child will be well. This is especially likely to happen when the onset of the symptoms is acute, the temperature unusually high and the discharge at first very profuse. In most cases, however, the temperature does not come down to normal or, if it does, soon rises again and the discharge continues. If it was not purulent at first, it soon becomes so. The incision often becomes blocked and then there is a return of the pain and an exacerbation of the symptoms. It is usually from one to three weeks, even if there are no complications, before the temperature returns to normal and the discharge ceases.

If the discharge lasts more than three or four weeks the condition is essentially a chronic one. The same methods of treatment have to be kept up, however, although if the discharge is very thick, the ear may occasionally be cleaned up with a 1 to 2 peroxide solution or a few drops of a mixture of 10 grains of boracic acid in an ounce of 95% alcohol put into the ear after it has been well dried. In most instances the trouble is likely to continue until the adenoids are removed. If there is reason to suspect that there is disease of the ossicles or of the antrum, that granulations have formed in the tympanic cavity or that the process is tubercular, the patient should, if possible, be placed in the hands of a specialist.

Complications.—*Inflammation and Infection of the External Auditory Canal.*—Among the most common complications of otitis media is inflammation of the external auditory canal. Not infrequently small furuncles develop, which, if situated on the posterior wall, are mistaken for swelling due to mastoid inflammation. Inflammation and furunculosis are less likely to develop if the canal is thoroughly cleaned and dried when the ear is treated. If furuncles develop, they should be opened.

Adenitis.—There are several small glands just in front of and below the lobule and one just behind the ear over the tip of the mastoid which not infrequently become enlarged and sometimes break down. Enlargement of the gland over the mastoid always suggests inflammation of the mastoid, but does not necessarily mean it. These glands should be let alone unless they suppurate, when they should be opened.

Mastoiditis.—The mastoid process is small at birth as is also the antrum, which is about the size of a pea. The mastoid cells are not present at birth but develop during infancy, being numerous and of fair size at three years. The rate at which they develop is so variable that it is never safe to assume that even a young infant has no mastoid cells. Mastoid inflammation may develop as a complication of otitis media at any age, but is less common in infancy than in early childhood. Symptoms which suggest that mastoid inflammation has developed are a

higher temperature, more profuse discharge, longer continuance of the discharge and more marked leucocytosis than would be expected from the appearance of the middle ear. None of these symptoms are, however, more than suggestive. Fullness of the posterior wall of the canal and enlargement of the posterior auricular gland are more important, but may be due to other causes. Tenderness over the mastoid is strong evidence in favor of mastoid trouble in childhood, but is of little importance in infancy, because there is often no tenderness at this age and because it is almost impossible to determine, if there is tenderness, whether it is in the mastoid or in the middle ear. Swelling and redness over the mastoid are relatively late but quite positive symptoms. Displacement of the external ear outward and forward is positive evidence of mastoiditis. The condition should have been recognized and treated, however, before this occurs. In my experience little information can be gained from the white blood count. It is certain to be high, anyway, from the otitis, varies much during an otitis, may or may not rise with a mastoiditis or may even fall if the infection is very severe.

When the mastoid involvement is slight it requires great judgment to decide whether to simply continue with the treatment of the otitis media or to operate. If the involvement is at all marked operation should be performed at once as, in this way, recovery is hastened and the chances of the extension of the inflammation to the sinuses and meninges much diminished.

Thrombosis of the Sinuses.—Owing to the fact that the petrosquamosal suture is not closed until after the first year, it would seem as if extension of inflammatory processes in the ear to the sinuses and meninges would be more common in infancy than later. As a matter of fact, however, involvement of the sinuses is more common in childhood than in infancy. Inflammation and thrombosis of the sinuses seldom develops unless there has been previous trouble in the mastoid, but cannot be ruled out if there has not. The symptoms of thrombosis depend on the severity of the process, on how much the clot is walled off, whether there is pus in the sinus and on whether infection is entering the systemic circulation. In general, the child seems sicker than it ought to be and the temperature is higher and lasts longer than would be expected from the local appearances. Headache and tenderness behind and above the mastoid and marked irregularity of the temperature are suggestive. Chills are almost positive proof. On the other hand, it must never be forgotten that a child may have thrombosis of the lateral sinus and not seem much sick, the only symptom being a continued irregular temperature. A high white count is also suggestive of trouble in the sinuses, but not of great importance, because the conditions which are the cause of the thrombosis also raise the white count and, in general, not much stress can be laid on the degree of the leucocytosis in children. Operation is demanded whenever there is thrombosis of the sinuses.

Meningitis.—Meningitis is an infrequent complication of otitis media and is more common in late childhood than in infancy and early childhood. The symptoms are those of infection of the meninges from any cause. Whenever meningitis is suspected a lumbar puncture should be done for diagnosis. The meningitis being due to one of the pus organisms or the influenza bacillus, the fluid will contain the causative organism and an excess of polynuclear cells. A moderate increase in mononuclear cells without organisms or polynuclear cells in the cerebrospinal fluid does not necessarily mean meningitis. The prognosis of

meningitis is almost hopeless and treatment is of little avail. There is little to do but to treat the local conditions and to do repeated lumbar punctures.

Abscess of the Brain.—This is a very uncommon complication of otitis media in early life. The symptoms are a combination of those due to a collection of confined pus and a cerebral tumor. The latter vary, of course, according to the size and location of the abscess. The treatment is operative. If a child has the signs of a cerebral tumor and it has previously had otitis, there is a fair chance that the "tumor" is really an abscess.

Facial Paralysis.—The facial nerve is, on account of its situation, not infrequently involved in otitis media with bone disease. The whole trunk of the nerve being involved, all the branches are affected. Taste may or may not be lost and hearing made more acute or not, according to what part of the trunk is involved. It is almost impossible and fortunately unimportant, to determine these points in infancy and childhood. The outlook for complete recovery is not very good, although there is usually some improvement. Something can be done by massage and electricity to help the condition.

Cervical Adenitis.—The glands in the neck may be involved in otitis and mastoiditis. The glands involved are the same as those which are involved in nasopharyngitis and in chronic infections of adenoids. In fact, whenever they are involved, the infection probably usually comes from the nasopharynx rather than from the middle ear or mastoid. They should be treated in the same way as enlarged cervical glands from any cause.

FOREIGN BODIES IN THE NOSE AND EARS

Young children occasionally put foreign bodies in their own noses or in those of babies. These foreign bodies, although they interfere with nasal respiration, seldom cause much discomfort, probably because they are usually in but one nostril. They cause local irritation and often superficial ulceration of the nasal mucous membrane with a purulent and a not infrequently foul discharge, which is, of course, unilateral. If the foreign body is of such a nature that it can decompose, the discharge is rendered more foul. A continuous purulent discharge, especially if it is foul, from one nostril in an infant or young child almost always means that there is a foreign body in the nose.

Foreign bodies are usually not very far in and are ordinarily easy to remove with forceps or a hook. The discharge usually promptly ceases when the foreign body is removed. Recovery may, perhaps, be hastened by spraying the nose with one of the mild alkaline antiseptic solutions.

Children are much less likely to put things in the ears than in the nose. Foreign bodies in the external auditory canal usually do not cause much discomfort unless they impinge on the drum, and are much less likely to produce ulceration and discharge than when in the nose. Bugs and worms of various sorts occasionally fly or crawl into the ears of babies and young children and cause much discomfort.

Bugs and worms can easily be drowned by putting water or oil in the ear while the patient lies on the other side, after which they are easily picked out. The removal of foreign bodies is often very difficult and the greatest care must be taken not to push them down onto the drum or through it. It is advisable to have a specialist remove them. If one is not available, it is safer to anaesthetize the child before attempting to remove the foreign body.

NASAL POLYPI

Nasal polyps are quite uncommon in childhood, but may occur. The symptoms are a combination of those of nasal obstruction, usually unilateral, and chronic nasal catarrh. The possibility of their occurrence is usually entirely forgotten and the symptoms are attributed to adenoids or sinusitis. They should be removed at once, as there is no other treatment which does any good.

DEVIATION OF THE NASAL SEPTUM

This malformation is not uncommon even in early childhood, but is often overlooked or forgotten. It may be a congenital malformation, but is more often the result of a deformity of the upper jaw from adenoids. The narrowing of the upper jaw exaggerates the arch of the hard palate, which, in turn, buckles the septum. The symptoms are those of nasal obstruction and chronic nasal catarrh. It is inadvisable and usually unnecessary to operate on a deviated septum in childhood. Proper orthodontic treatment to widen the upper jaw will lower the arch and allow the septum to come back in place. Recovery is also aided by the changes incident to the growth of the parts.

ACUTE CATARRHAL AND ACUTE FOLLICULAR TONSILITIS

These conditions are grouped together because they are both manifestations of the same disease, the appearances differing according to whether the process begins in or is more marked on the surface or in the crypts of the tonsils.

Etiology.—Tonsilitis is primarily due to infection with microörganisms. The causative organisms are almost always staphylococci and streptococci, but occasionally pneumococci and other organisms. It is evident, however, that, as some or all of these organisms are constantly present in the throat, there must be some predisposing cause which weakens the resistance to infection. Such causes are fatigue, indigestion and chilling of the body. The latter cause presumably acts by changing the local blood supply. The occurrence of tonsilitis in epidemics shows, however, that infection may be due to an increase in the virulence of the organisms, even when the resistance is normal. Tonsilitis is unquestionably contagious, but, on the other hand, for infection to take place the virulence of the organism must be increased or the resistance of the individual must be diminished. Some children and families seem to be especially susceptible to infection. Tonsilitis is not a manifestation of rheumatism, which is itself merely a name used to describe a special type of infection with microörganisms of the coccus group, which microörganisms may or may not be specific. Tonsilitis is uncommon in infancy, most common in early and middle childhood and less common in late childhood.

Symptomatology.—The onset of tonsilitis is usually acute. A chill is relatively uncommon in childhood, while vomiting is not unusual. The temperature rises rapidly.

The throat is sore, swallowing is painful and the neck is tender and stiff. Any or all of the symptoms which an elevation of the temperature and toxic absorption produce, such as pains and aches, chilly sensations, loss of appetite, vomiting, and other disturbances of the digestion may develop. There is nothing characteristic about these general symptoms, which are those common to all febrile diseases. The temperature remains

high, usually between 103° F. and 104° F., for from three to five days, when it rather quickly drops in two or three days to normal. The other symptoms usually disappear at the same time as the fever.

The fauces are much reddened and the tonsils swollen. Both sides are usually involved. The tonsils may be so much swollen as to nearly meet in the median line. In acute catarrhal tonsilitis the tonsils are simply reddened. In follicular tonsilitis there are numerous whitish or yellowish spots, the size of a head of a pin or a little larger, at the openings of the crypts. These spots may become larger and sometimes coalesce. The larger areas are easily scraped off and do not leave a raw

FIG. 102.—Method for examination of throat in infancy.

FIG. 103.—Method of holding child for examination of the throat.

or bleeding surface. The smaller spots can only be removed by pressing out the contents of the crypts. The tonsillar glands are usually somewhat or moderately swollen and tender. Sometimes they are considerably enlarged. They seldom, however, break down.

Complications.—Peritonsillar abscess is an uncommon complication in young children, but becomes progressively more common as children grow older. Infection of the cervical lymph nodes with abscess formation is also an uncommon complication. In most instances, even if the cervical lymph nodes are considerably enlarged, they quickly disappear after the subsidence of the tonsilitis. Endocarditis is a not uncommon and very serious complication of acute tonsilitis. Pericarditis occurs much less frequently and, when it does, is almost always associated with endocarditis. Acute nephritis is another not very uncommon complication of acute tonsilitis. It must not be forgotten, however, that albuminuria does not necessarily mean acute nephritis, but may be simply a manifestation of the so-called acute degenerative nephritis or febrile albuminuria. Appendicitis is not a very unusual complication, especially in older children.

On account of the frequency and seriousness of endocarditis, pericarditis and acute nephritis as complications of acute tonsilitis, the heart should be examined daily and the urine repeatedly during the course of every case of acute tonsilitis. Furthermore, the urine should be examined a few days or a week after apparent recovery from tonsilitis, because nephritis may not appear until some days after the temperature has fallen. This rule should also be followed after every case of acute nasopharyngitis.

Diagnosis.—Acute tonsilitis may be confused with scarlet fever and diphtheria. The onset in scarlet fever and tonsilitis may be much the same. The appearances in the throat in the beginning are quite similar, especially if there are no spots and no exudation on the tonsils. Theoretically, the scarlet fever throat should be a brighter red and the redness should be due to many minute points instead of to a diffuse flush. Practically, it is very difficult to make out these points. The condition of the tongue is the most important point in diagnosis, there being nothing characteristic about the tongue in tonsilitis, while in scarlet fever it is reddened and the papillae are enlarged and distinct. If the disease is scarlet fever the rash will, of course, appear in from twenty-four to forty-eight hours.

The onset in diphtheria is usually less acute and the temperature lower than in tonsilitis. The swelling of the tonsils is usually not as great. The throat is dull red in diphtheria and a brighter red in tonsilitis. In diphtheria a thin, grayish film soon appears on the uvula, pillars or tonsils, while in tonsilitis yellowish or whitish spots appear at the mouths of the crypts. Sometimes, however, these follicular spots appear also in diphtheria. If there is an exudation on the tonsils, it will come off easily and leave a clean surface in tonsilitis, while it will come off with difficulty and leave a bleeding, raw surface in diphtheria. The glands in the neck are usually much more involved in diphtheria than in tonsilitis. Theoretically again, the diagnosis is very easy. Practically, it is often very difficult, and, therefore, in every case of sore throat, no matter how certain the diagnosis of tonsilitis seems to be, a culture should be taken.

Prognosis.—Acute tonsilitis in itself is not a serious disease. Its danger lies in the complications, the most serious of which are those in the heart and kidneys.

Treatment.—A very important part of the treatment of acute tonsilitis is the preventive. Children ill with tonsilitis should be isolated. If they are not, parents should see that their own children are kept away from them. Good hygiene and intelligent care to prevent exposure and chilling will prevent many attacks. If a child has had several attacks of tonsilitis, its tonsils should be removed, because in any attack the heart or kidneys may be involved.

There is no treatment which will reach the primary cause of the disease, namely, bacteria. The condition is too acute for vaccines to be of any service. Treatment must be entirely for comfort, the relief of symptoms and the prevention of complications. The tincture of the chloride of iron, which has been so much used in the past, is useless. It has no action on bacteria and is not required, at any rate in the beginning, for anemia.

Children with tonsilitis should be put to bed and kept there until well. The temperature of the room should be from 64° F. to 68° F. and the air should be kept fresh. The diet must necessarily be restricted

to liquids and soft solids, because of the soreness of the throat. Milk should form the basis of the diet and may be given plain or in the form of gruel, junket, blanc-mange or ice cream. Cereals, milk toast and baked custard are allowable. Broths are inadvisable, as in all illnesses, because they contain practically no nourishment. Water should be pushed, at least a quart being given daily, in order to diminish the chances of irritation of the kidneys. It is useless to give a cathartic after the disease has developed, because at this time it can do no good to withdraw blood from the throat, even if catharsis does this, which is doubtful. It is advisable, however, to give a laxative to keep the bowels open.

The discomfort due to the fever and the symptoms which it causes can be much relieved by proper treatment. There are many ways to do this. I am old-fashioned enough to believe that sweet spirits of nitre does reduce fever by increasing perspiration and in that way makes children more comfortable. From five to twenty drops in a tablespoonful of water, every hour, according to the age of the child, is the usual dose. Aconite is another drug which is not being used nowadays as much as it should be. When given in doses of from one quarter to one drop every fifteen minutes for an hour and then every hour afterwards, according to the age of the child, it does diminish fever and make children more comfortable. The combination of sweet spirits of nitre and aconite is a very good one. Another good mixture is a combination of phenacetin and salol, in doses of from one and one quarter to two and one half grains of each, given at intervals of from one to three hours, according to the age. The present fashion is to use aspirin in doses of from one and one fourth to five grains every two hours or more, according to the age. This also relieves the symptoms. It does not do so, however, because it is an antirheumatic, whatever that may be, but simply because it reduces the fever. The temperature in tonsilitis in children is seldom high or prolonged enough to require treatment by bathing. There is, of course, no objection to the use of cold externally in tonsilitis, but most children object to it so much that it is hardly worth while to use it unless it is necessary. An ice bag to the head will often relieve the headache and often tends to reduce the temperature.

Chlorate of potash, which has been and still is almost universally used in the treatment of acute tonsilitis and is considered by many to be a specific, has been conclusively proved to be entirely inert. It has no local action on the bacteria and is not eliminated through the tonsils and mucous membrane of the throat as used to be claimed. It is also an irritant to the kidneys and may possibly do harm in that way. Personally, however, I feel that the danger of injury to the kidneys from this drug has been exaggerated. It is useless to treat tonsilitis with gargles. In the first place, very few children will gargle; in the second place, if they do, the gargle does not reach back of the anterior pillars. Sprays may do some good because they really do reach the tonsils and the back of the throat. Most children resist them so much, however, that they amount to very little. Strong applications, such as iodine and glycerine, glycerite of tannin, peroxide of hydrogen and the silver preparations, do not kill the bacteria, make the patient uncomfortable and increase the local irritation. Cracked ice to suck often gives temporary relief. The only method of local treatment which really does any good or gives any real comfort is irrigation of the throat with hot saline solution. This is prepared by adding a level teaspoonful of salt to a pint of water as hot as can be borne in the throat. This temperature is usually between

115° F. and 120° F. The hot salt solution is placed in a fountain syringe about two feet above the patient's head. The patient may either sit up or lie down on the side. A basin or a pus basin should be held under the chin. A hard rubber nozzle, preferably with a hole in the end and without holes in the side, is then introduced into the throat and the hot salt solution allowed to run in and out. It goes without saying, of course, that the patient must stop breathing while this is going on. With repeated intermissions, the whole of two quarts should be allowed to run in and out.

Heat and cold externally probably have little effect on the course of the disease, but do often make the patient more comfortable. The only feasible method of applying cold is with an ice collar. It must be remembered that the continued application of an ice collar may freeze the skin unless one or two layers of flannel are placed between it and the skin. It is usually not advisable, moreover, to use ice continuously anywhere on a small child. The best form of poultice is the old-fashioned flaxseed poultice, which should be changed at least every two hours. Antiphlogistine, not being put on as thickly and having no medicinal properties, is not as useful. Neither is flannel, because it is not as thick and warm. There is no advantage in using poultices that smell badly, like those made of onions, or those that are nasty, like those made of pork.

MEMBRANOUS TONSILITIS

Synonyms for membranous tonsilitis are streptococcus angina, septic sore throat and croupous tonsilitis. Membranous tonsilitis is almost always caused by streptococci, frequently of the hemolytic variety, but sometimes by staphylococci, pneumococci or gonococci. If the disease is epidemic or milk-borne, the infecting organism is almost invariably the streptococcus hemolyticus. Membranous tonsilitis may be sporadic, epidemic or occur in the course of other diseases. If epidemic, the infection is usually carried in milk, the source being either in the udder of some cow or in the throat of some person who handles the milk. The disease of which it is most often a complication is scarlet fever, although it sometimes complicates measles.

Symptomatology.—The characteristic lesion is a membrane on the tonsils. The appearances of this membrane are much like those of the diphtheritic membrane, but it is usually more yellow and the edges are less sharply defined. When the membrane is detached, the surface which is left is not quite as raw and does not bleed as much as in diphtheria. It cannot be positively distinguished, however, even microscopically, from the diphtheritic membrane. In the sporadic cases it seldom spreads beyond the tonsils, but in the epidemic variety and in the cases complicating scarlet fever, it may spread over the hard palate and uvula, back into the pharynx, up into the nose and even downward to the larynx.

The onset in the sporadic and epidemic cases is almost always acute with a chill or vomiting, and a rapid rise of temperature. In the secondary cases it is obscured by the symptoms of the primary disease. The throat is, of course, sore and swallowing difficult and painful. The temperature is high, often irregular, and all the symptoms which accompany fever and toxic absorption develop. If the membrane extends to the soft palate and pharynx the breath is very foul. If it extends to the nasopharynx and nose, nasal respiration is disturbed, there is a foul, purulent nasal discharge and otitis media often develops. There may be

necrosis of the tonsils and surrounding tissues with sloughing of the tissues and sometimes severe or even fatal hemorrhage.

The cervical lymph nodes are almost invariably involved. In mild cases those which drain the tonsils are alone affected while in more severe cases those which drain the nasopharynx and ears are also involved. In the most severe cases all the glands in the neck may be affected. They may or may not break down. If they do, the pus not infrequently burrows deep into the neck. In some instances the retropharyngeal lymph nodes are involved and retropharyngeal abscesses result. Secondary bronchitis and bronchopneumonia are not uncommon. The secondary bronchitis is sometimes purulent.

Degenerative changes occur in all the parenchymatous organs as the result of toxic absorption. The most common of these degenerative changes are myocarditis, acute degenerative nephritis and enlargement of the liver and spleen. Acute nephritis is also not uncommon, but may be due in part to the direct action of bacteria on the kidneys.

Many inflammatory conditions may develop in various organs as the result of the entrance of bacteria into the circulation. Among these are endocarditis, pericarditis, appendicitis, purulent peritonitis, osteomyelitis or periosteitis, arthritis, meningitis and localized abscesses. In other instances there are no localized inflammatory processes, but all the symptoms of a general systemic bacterial infection develop.

Prognosis.—The prognosis is usually good in sporadic cases. The membrane seldom extends beyond the tonsils. Improvement ordinarily begins after two or three days and the membrane is gone in four or five days. The enlargement of the cervical lymph nodes is usually not extreme. The symptoms of toxemia are not very marked and there are usually no severe complications.

In epidemic cases the course is almost always more severe and usually longer. The symptoms of toxemia are more marked and complications from systemic infection, especially peritonitis, much more common. Many die. Peritonitis is almost always fatal. Complications are liable to occur at any time and, therefore, patients cannot be considered out of danger until they are entirely well.

In the secondary cases the prognosis depends, in part, on the primary disease. It necessarily varies with the severity of the local symptoms and the complications in the individual case. Peritonitis is less common in the secondary cases, while endocarditis, nephritis and general systemic infection are more common.

Diagnosis.—It is very difficult to distinguish sporadic and epidemic membranous tonsilitis from diphtheria. The appearances in the throat and its adnexa are very much the same in both, although the onset is usually more acute and the temperature higher in membranous tonsilitis. Cultures should, therefore, be taken in every case and, if there is much membrane, diphtheria antitoxin should be given without waiting to hear from the cultures. It does no harm to give antitoxin unnecessarily. It may do great harm to wait, if the disease proves to be diphtheria.

If the infection in the throat occurs early in the course of another disease, such as scarlet fever or measles, the chances are very great that it is not diphtheria. Cultures should be taken, but it is not necessary to give diphtheria antitoxin at once. If the infection in the throat occurs late in the course of another disease, the chances are about even between membranous tonsilitis and diphtheria. Cultures should be taken, but it is usually safe to wait for the bacteriologic report before giving antitoxin.

Treatment.—Whenever a child has a membrane in the throat it should be isolated, cultures taken and antitoxin given, unless it seems safe to wait. If there is an epidemic of septic sore throat, the milk should be boiled. There is no specific local treatment for membranous tonsilitis. The local treatment is the same as in acute catarrhal and follicular tonsilitis. If the nose and ears are involved the treatment is the same as when they are involved primarily.

There is, unfortunately, no specific general treatment for membranous tonsilitis. Vaccines can do no good in acute cases. It is possible that an autogenous vaccine may help some in the subacute cases. In general, it is along the same lines as the treatment of diphtheria. Complications in the kidneys may, perhaps, be avoided by giving large amounts of water.

VINCENT'S ANGINA

Vincent's angina is caused by Vincent's bacillus. This is a fusiform bacillus which also often appears in a degenerated form as a spirillum. Both forms are usually seen together, although the fusiform bacillus may be the only one present. The spirillum is never seen alone. It is very easy to miss this organism, however, unless the smear is taken from deep down in the ulcerated area. It is cultivated only with great difficulty.

Symptomatology.—Vincent's organism produces an ulceration on the surface of the tonsil. The pathologic process is usually limited to one tonsil, but it is not infrequently associated with *ulcerative stomatitis* or *gingivitis*, due to the same organism. The onset is always slow. The constitutional symptoms are relatively slight and the throat is not very sore. In fact, it is often discovered by accident. There is some enlargement of the tonsillar glands on the affected side. The breath is often very foul. The ulcerated area has an irregular outline and is covered with a dirty yellow or grayish membrane, which may be necrotic. When it is pulled off it leaves a raw and bleeding surface, which may be sunken as much as one-fourth of an inch. The tonsil is not much enlarged and there is relatively little inflammation about the ulcerated area.

Diagnosis.—This condition may be confused with diphtheria, membranous tonsilitis and syphilis. The slowness of the onset, the mildness of the constitutional symptoms and of subjective symptoms in the throat are quite different from the onset and constitutional symptoms in diphtheria and membranous tonsilitis. Vincent's angina is usually unilateral, while both tonsils are ordinarily involved in diphtheria and membranous tonsilitis and there is more swelling and inflammation of the tonsils as a whole. The membrane in diphtheria is grayer, smoother and the edges better defined, and, when it is taken off, the ulceration is not as deep. While they can usually be readily distinguished, it is always advisable to make a smear from the deeper parts of the membrane and also to take a culture. Vincent's angina is not likely to be mistaken for a syphilitic ulceration, if it is thought of as a possibility. The appearances of the two may be quite similar, but it is most unusual to have a syphilitic lesion on the tonsil of a child, and to have it the only lesion. If the lesion is syphilitic, other evidences of syphilis are almost always present. If there is any doubt, a smear will quickly dispel it.

Prognosis and Treatment.—Vincent's angina is not usually a serious disease and ordinarily yields promptly to proper treatment. The child should be put to bed and proper food forced. It may be advisable to

give nux vomica for the appetite and cod liver oil to improve the nutrition. Chlorate of potash has no action. The best and simplest method of local treatment is to paint the ulcerated area daily with tincture of iodine. A 5% or 10% aqueous solution of chromic acid is also useful, but must be used carefully.

PERITONSILLAR ABSCESS

In this disease, which was formerly called quinsy, there is an inflammation of the cellular tissue surrounding the tonsil. This inflammation almost always goes on to the formation of an abscess, but may sometimes quiet down. It is, of course, due to bacterial infection, usually with streptococci. It is generally secondary to catarrhal or membranous tonsilitis, but may apparently sometimes be primary. If so, there is probably a focus of infection in the tonsil. It is almost invariably unilateral, and very seldom follows or complicates diphtheria, scarlet fever or measles. Peritonsillar abscess is extremely uncommon in infancy, rare in early childhood and not at all common in late childhood.

Symptomatology.—The symptoms differ somewhat according to whether the disease follows tonsilitis or not. If it is secondary to tonsilitis, the temperature rises again, the throat becomes sorer, the neck stiffer, and swallowing more difficult and painful. Speech is difficult, torticollis not uncommon. Inspection shows an increase in the swelling, which is usually above and in front of the tonsil, but sometimes behind it. In some instances it is in the deep tissues lateral to the tonsil, when it cannot be seen but is easily felt externally. The tonsil is, of course, displaced into the throat, its position depending on the location of the inflammation about it.

If the abscess is not secondary to tonsilitis, the onset is very much the same as in acute catarrhal tonsilitis, except that there is little or no redness of the tonsil for several days. The soreness of the throat, the pain and difficulty in deglutition and the stiffness and tenderness of the neck are all out of proportion to what can be seen in the throat. After a few days, however, the appearances in the throat are the same as in the secondary cases. In both types the abscess forms in from five to seven days. If left to itself, it soon ruptures into the throat.

Prognosis.—It is conceivable that the sudden rush of pus into the throat, when an abscess ruptures spontaneously, may in a baby cause asphyxiation or lead to a septic inhalation pneumonia. This danger is very slight, however, as babies and young children almost never have the disease. Except for it, the outlook for recovery is good. Recurrences are not uncommon. Complications in the heart and kidneys occur at least as frequently as in catarrhal and membranous tonsilitis. Cervical adenitis is common and may result in an abscess.

Treatment.—In the beginning treatment can only be for comfort and the relief of symptoms. Aspirin or phenacetin and salol may be given for the fever and general discomfort (see Acute Catarrhal Tonsilitis). Dover's powder, in doses of from 1¼ to 5 grains, sometimes helps. There is no drug, which, taken internally, has any effect on the course of the disease and there are no local applications which limit its progress in any way. An ice collar often affords considerable relief. It must not be forgotten, however, that it is not safe to use an ice collar continuously and that it must be covered with several thicknesses of flannel. In other instances a hot flaxseed poultice or an electric heating pad gives more relief.

Cracked ice to suck sometimes helps. Gargles are useless in children. They seldom know how to gargle well and furthermore, it is almost impossible to gargle with a peritonsillar abscess. Irrigation of the throat with hot saline solution gives more relief than any other form of local treatment (see Acute Catarrhal Tonsilitis). As soon as the abscess has formed, it should be opened. It is a mistake to incise the swelling before pus has formed, because, if the abscess has not fully developed, incision does little good and the opening quickly closes. The proper time to open the abscess can usually be determined better by palpation than by inspection.

The child should be held sitting upright in someone's lap with its back against the person who is holding it. He should put one arm around the child's body, holding its arms, and should hold its head with the other arm. It is advisable for the person holding the child to confine its legs by crossing one of his legs in front of them. The child must be held so that the light shines into its throat or, if preferred, the operator may reflect the light into the throat with a mirror. The abscess should be opened with a knife guarded to within half an inch of its point. If it is so guarded it is impossible to cut too deeply. It is important to make the incision long enough so that it will not close easily. It should be between three fourths of an inch and an inch long. The child's head should be bent forward as soon as the abscess is opened so that the pus and blood can run out of its mouth and not be inhaled. There is often more blood than pus. The relief from the subjective symptoms is almost immediate and the temperture quickly begins to fall. The constitutional symptoms diminish with the temperature and are usually gone in two or three days. The incision should be kept open with a director or with the finger for several days in order to prevent it from closing up too quickly. The saline irrigations should be continued for several days. Any of the mild alkaline antiseptic mouth-washes may be used, if desired. The wound usually closes in four or five days.

The urine and the heart should be watched in the same way as in acute catarrhal and membranous tonsilitis. If a child has had more than one peritonsillar abscess, the tonsils should be removed.

CHRONIC HYPERTROPHY AND DISEASE OF THE TONSILS

The faucial tonsils are relatively large in early life. There is normally an increase in size at the time of the second dentition. No one knows whether they play any part in nutrition and growth during infancy and childhood or not. There is much difference of opinion as to whether they prevent or favor infection of the cervical lymph nodes and other organs. I do not know. I can say, however, that I have never seen any disturbances of nutrition, growth or development or any local or general infections develop after their removal, which I have thought were the result of their removal or would not have occurred, if they had not been removed.

Treatment.—It is very difficult to know where normal hypertrophy of the tonsils ends and hypertrophy from chronic irritation or recurrent acute inflammation begins. It is probably safe to say, however, that when the tonsils are large enough to mechanically interfere with respiration and deglutition, part, at least, of the enlargement is pathologic. Whether it is or not, the tonsils should be removed when they cause such interference. It is often very difficult to determine from the appearance of a large tonsil whether it is diseased or not. If it appears

normal, the tonsillar glands are not enlarged and the child does not have repeated attacks of sore throat, it is reasonable to assume that it is not diseased. Such tonsils should not be removed, as it is not justifiable to remove an organ, which may possibly be of some use, simply because it seems larger than usual. If, on the other hand, the tonsillar glands are enlarged, there are repeated attacks of sore throat or even repeated colds without sore throat, provided there are no adenoids to account for them, evidences of toxic absorption or of disturbance of nutrition without other obvious cause, such tonsils should be removed. In this connection it must be remembered that it is impossible to tell from a casual glance at the throat whether a tonsil appears normal or is enlarged or not. The tonsils should be pulled out from the pillars and carefully examined. It must also be remembered that it is impossible to tell from the appearance of the outside of a tonsil what may be inside of it, and that small and buried tonsils are just as likely to be diseased as those which are enlarged. If the tonsils appear diseased, they should be removed, whether there are any evidences of infection or toxic absorption from them or not.

The tonsils should be removed, no matter how they look, if a child has had rheumatism, endocarditis, pericarditis or acute nephritis, unless there was some other evident cause for these diseases. They should be removed if there is chronic cervical adenitis, whether it is or is not tuberculous, excluding, of course, adenitis due to leukemia, pseudoleukemia or obvious lesions of the mouth, skin and scalp.

It is idle to talk about any local treatment for tonsils which are causing trouble, whether it is mechanical or otherwise. If they are doing harm, they should come out. Clipping of the tonsils is an irrational procedure. They should be enucleated. Incidentally, serious accidents are not uncommon when tonsilectomy is performed by amateurs. It should not be done by those who are not properly trained. Furthermore, it should never be done without an anaesthetic or in the office, and the operator or someone else qualified to stop a secondary hemorrhage should be within reach for at least twenty-four hours. The tonsils should not be removed until at least two weeks have elapsed since recovery from any acute inflammatory process in them. The results of treatment with the Roentgen ray are unreliable and unsatisfactory. Those from radium are little, if any, better. Moreover, all treatments with the Roentgen ray and radium, especially with radium, carry elements of danger both at the time and for the future. No one knows what the ultimate effects of exposure to radium may be. There are no such dangers associated with enucleation.

The tendency is to look upon the operation of tonsilectomy too lightly and to let children up and about too quickly. It takes at least a week to entirely recover from the shock of the operation and the throat is especially susceptible to infection until the raw surfaces have healed. Children should, therefore, be carefully guarded against possible infection until the throat is entirely healed.

Prognosis.—Too much is often promised and expected from tonsilectomy. Children will continue to have colds and sore throats after the removal of their tonsils and may even develop inflammation of the heart and kidneys. The colds will almost certainly, however, be less frequent and less severe and cardiac and renal complications less common. Moreover, if they contract scarlet fever or diphtheria, the course of the disease will not be as severe.

CERVICAL ADENITIS

Cervical adenitis, unless it is a manifestation of leukemia, pseudoleukemia or malignant disease, is always due to infection through the lymphatics from some primary focus, which is most often in the fauces, nose or nasopharynx. When the primary focus is in the tonsils, the lymph nodes involved are those just behind and below the angle of the jaw and in front of the sternomastoid muscle. When it is in the nasopharynx the nodes involved are beneath or behind the sternomastoid or a little below the tonsillar glands. In many instances, however, both the tonsils and nasopharynx are infected and both groups are involved simultaneously or, if one group is involved at first, the infection spreads to the others. The preauricular nodes, situated just in front of the auditory canal and deeply just below the zygoma, are involved if there is an infection or eczema of the forehead, front of the scalp, the crease above the ear, or the anterior wall of the external auditory canal. The nodes which are situated about the facial artery and vein in front of the jaw or just below it are affected if there is inflammation of the cheek, the outer corner of the eye, the mouth, nostrils, anterior portion of the nose or upper jaw. The submaxillary glands are involved if there is inflammation of the lower lip, chin or lower jaw. The occipital nodes are enlarged if there is inflammation of the posterior portion of the scalp, while those about and over the mastoid process are involved if there is disease of the mastoid or inflammation of the posterior wall of the external auditory canal or of the crease behind the ear.

The cause of the enlargement of the lymph nodes, other than those about the sternomastoid, is usually obvious. The enlargement may be acute or chronic. It is almost never due to tuberculosis, except occasionally when the postauricular and preauricular nodes are involved. The swelling usually subsides when the cause is removed. An abscess seldom develops, but, if it does, it should be opened. Some of the nodes are so close to the surface of the lower jaw that, if an abscess forms, it is easy to mistake it for an abscess of the jaw itself or for malignant disease of the jaw.

Enlargement of the nodes about the sternomastoid, which is what is usually meant by the term "cervical adenitis," may be either acute or chronic. If the enlargement develops quickly in connection with an acute inflammation of the tonsils or nasopharynx, the chances are very much against the process being tuberculous. If the swelling does not disappear within a few weeks after the subsidence of the acute process in the throat, the chances are that the process is tuberculous. The longer it persists, the greater are the chances. If the swelling develops slowly without an acute inflammation of the throat, the chances are that the infection is tuberculous. In general, at least 80% of chronically enlarged cervical lymph nodes are tuberculous, and most of them are caused by the bovine bacillus. A negative tuberculin test is of great importance and practically proves that the nodes are not tuberculous. A positive test is strong evidence in favor of their being tuberculous, but does not prove that they are, because they may be non-tuberculous and the positive reaction due to some focus elsewhere in the body.

Diagnosis.—Acute enlargement of the cervical lymph nodes may occur in leukemia. If so, there is almost always enlargement of other superficial and deep lymph nodes, which is not the case when the infection is a local one. There is, moreover, no primary infection in the fauces or naso-

pharynx. The preauricular nodes are often involved and also the lymphoid tissue in the parotids. This swelling of the parotids is sometimes mistaken for *Mikulicz's disease*. An examination of the blood will, of course, settle the diagnosis.

Leukemia is such a rapid disease in childhood that there is seldom time for the enlargement of the cervical lymph nodes to be called chronic. Chronic enlargement may, however, occur in pseudoleukemia. In this disease the lymph nodes on both sides of the neck and in other parts of the body are usually, but not always, involved. The nodes are usually harder and show no evidences of breaking down. The changes in the blood do not justify a diagnosis, as in both chronic tuberculous adenitis and pseudoleukemia they are simply those of secondary anaemia with more or less polynuclear leucocytosis. The diagnosis can usually only be made by the removal and microscopic examination of one of the nodes.

Prognosis and Treatment.—It is impossible to tell in any given case of acute cervical adenitis whether the inflammation will quickly quiet down with the subsidence of the primary disease in the throat or nasopharynx, the enlargement persist for a number of weeks and then disappear, persist indefinitely or go on to suppuration. In the beginning an ice collar usually makes the patient more comfortable and may, perhaps, to some extent, hinder or prevent suppuration. Some children prefer a poultice. Poultices are said to favor and hasten suppuration. I am very skeptical as to whether either cold or hot applications have any effect either in causing or preventing it, and use whichever form of treatment makes the patient more comfortable. Furthermore, I do not believe that medicated clay or any of the preparations of iodine, whether liquid or solid, colored or colorless, have any effect on the inflammatory process in the nodes in either the acute or chronic stage. If suppuration develops, the abscess should be opened and drained just before it is ready to point. When opened at this time drainage is better and healing quicker than when it is opened earlier, and the scar is smaller than when it is allowed to open spontaneously. If the swelling persists for several weeks after the infection in the throat has subsided and there are no evidences of suppuration, the tonsils and adenoids should be removed. Syrup of the iodide of iron or some other preparation of iodine may be given in the meantime and afterward, if desired, but I have serious doubts whether they really hasten resolution at all. Plenty of good food, sunlight and outdoor air with regulation of the life to prevent overfatigue are much more useful. It is possible that the ultraviolet rays may also do some good. If the swelling persists for several months after the throat has been cleaned up and in spite of good food, hygiene and care, the nodes should be removed. In most instances it will be found that, in spite of the acute onset, the infection is tuberculous.

If the swelling of the nodes develops slowly without any initial acute inflammation of the throat, the infection is almost certainly tuberculous and the focus in the tonsils or adenoids, usually the tonsils. They should be removed, if present. If there are many nodes no larger than peas or beans, it is better not to touch them. If there are many larger ones, or even one good sized one, they should be removed. Moreover, they should be removed before they have had a chance to break down. All the nodes should be removed. If suppuration has already taken place, it is sometimes wiser to incise the abscess and do a radical operation later. Unless there is already suppuration, the tonsils and adenoids should always be removed first; if there is suppuration, they should be removed before a

radical operation is done. It is useless to remove the nodes and leave the original focus of infection untouched.

It seems to me that treatment with the Roentgen ray is irrational, its object being to stimulate the growth of the periglandular tissue about and into the node and thus to prevent it from breaking down and to favor calcification. It is true that tuberculous cervical adenitis is essentially a local process and that there is very little tendency to dissemination, even when it is untreated, and probably less after treatment with the Roentgen ray. Nevertheless, it seems wiser to me to remove a tuberculous focus, when it is possible, than to leave it *in situ*. The same statement can be made in relation to treatment with the ultraviolet rays. The Roentgen ray is, however, of considerable value in healing broken down nodes and in hastening the closure of sinuses, thus preparing the field for operation. Treatment with radium seems to me too dangerous. I have seen very little of the treatment of tuberculous cervical adenitis with tuberculin, but what I have seen has been most disappointing. It seems to me to be much inferior to proper surgical treatment. It should go without saying that it is most important for children with tuberculous cervical adenitis to have every attention paid to their diet, care and general hygiene. Unless this is done the results of even the best surgery are likely to be unsatisfactory.

ACUTE CATARRHAL LARYNGITIS

Catarrhal laryngitis is usually not a primary condition in early life, but almost always develops as a complication of nasopharyngitis or pharyngitis. The symptoms which show that the larynx is involved are a harsh, dry cough and a husky voice. Older children may complain of soreness and a sense of constriction in the neck.

When laryngitis develops, it is advisable, if the child is not already in bed, to put it there. The temperature of the room should be from 66° F., to 70° F. and the air should be kept moist. The addition of a tablespoonful of the compound tincture of benzoin from time to time to a dish of water, which is kept boiling constantly in the room, often makes the patient more comfortable. Small doses of the wine or syrup of ipecac also help the cough and hoarseness (see Acute Bronchitis).

Spasmodic Croup.—Young children who have acute catarrhal laryngitis, even if it is very slight, are quite likely to develop spasmodic croup. Infants and children over eight years old seldom have it. The usual story is that a young child, who in connection with a "cold in the head" has had a harsh, dry cough during the day or late afternoon, wakes up at about 9 P. M., after having been asleep a couple of hours, with an attack of croup. It may have simply a very hard, dry cough with a husky voice and slight dyspnoea or it may be unable to cough or speak and scarcely able to breathe. Inspiration is prolonged, difficult and noisy; expiration is relatively easy. There is retraction of the supraclavicular and intercostal spaces and of the epigastrium. The child is unable to lie down and becomes cyanotic. It looks as if it would certainly die, but when it becomes sufficiently asphyxiated, the spasm, which is the cause of the serious symptoms and a complication of the laryngitis, lets up and breathing becomes easy again. Unless something is done the spasms are likely to recur at intervals during the night. All the symptoms of spasm have usually disappeared by morning and during the day the

child seems as well as it was before, but the symptoms recur the next night. It is usually said that attacks of the croup recur for three nights. This is in general true, but it is not an absolute rule. The symptoms of obstruction sometimes persist to a greater or less degree during the day and may be quite severe. This is uncommon, however, and when there is interference with the breathing in the daytime, the chances are that there is something more the trouble than catarrhal laryngitis and spasmodic croup.

Diagnosis.—The only disease with which catarrhal laryngitis and spasmodic croup are likely to be confused is laryngeal diphtheria. This can only occur, of course, when the laryngeal diphtheria is primary and not secondary to diphtheria in the fauces or pharynx. Laryngeal diphtheria is not usually preceded by a "cold in the head" as is catarrhal laryngitis. The symptoms of mild diphtheritic laryngitis, without obstruction, cannot be distinguished from those due to catarrhal laryngitis. If, as is almost always the case, diphtheritic laryngitis causes obstruction, the symptoms of obstruction come on much more gradually than do those of spasmodic croup in acute catarrhal laryngitis. There is a progressive and usually uninterrupted increase in the obstruction instead of a sudden onset of marked obstruction when there has not been any, as is the case in spasmodic croup. The obstruction in diphtheria does not let up in the daytime, but increases steadily as time goes on. It is easy to distinguish typical spasmodic croup from diphtheritic laryngitis. It is sometimes difficult to distinguish catarrhal laryngitis, in which the symptoms of obstruction continue, although varying in severity, from diphtheritic laryngitis. The mistake which is usually made is to mistake diphteritic laryngitis for catarrhal laryngitis or croup. If there is any question as to whether the condition is diphtheritic or catarrhal, antitoxin should be given without waiting for a diagnosis from a culture. It does no harm to give an unnecessary dose of antitoxin; not to give it in a case of diphtheritic laryngitis may well mean the life of the patient. Cultures are unreliable, moreover, unless taken from the larynx, because cultures from the throat are not infrequently negative, even when there is a diphtheritic laryngitis.

Treatment.—If a child has an attack of spasmodic croup it should be given one or two teaspoonfuls of the wine or syrup of ipecac to make it vomit. It is important to give a large enough dose, because a small dose does not cause vomiting and, therefore, does not relieve the symptoms. The dose cannot be too large, because, when the child vomits, it vomits up the excess. After the attack is relieved by the vomiting, a flaxseed poultice should be applied to the larynx and doses of from 5 to 10 drops of the wine or syrup of ipecac given every one or two hours, as necessary, to keep up relaxation, but not to induce more vomiting. The temperature of the room should be kept at from 68° F. to 70° F. and water kept boiling in the room, preferably with the compound tincture of benzoin added to it. It is useless to try to use a tent or a funnel with steam for small children. They almost always struggle and do not get as much as they do when the steam is kept constantly in the room.

The child should be kept in bed until all the symptoms of laryngitis have disappeared. If the spasm lets up during the day, as it usually does, the child should be given from 5 to 10 drops of the wine or syrup of ipecac every hour, beginning at 3 or 4 P.M., until it goes to sleep, the dose being varied according to the age of the child and its individual susceptibility to ipecac. If it becomes at all croupy during the night the ipecac should

be continued. If ipecac is given in this way, the attacks are not likely to return.

I have tried, at different times, various other methods of treatment for catarrhal laryngitis and spasmodic croup, including those recommended by the homeopaths, and have not found any of them as satisfactory as the treatment outlined above, except that, in some instances, it has seemed to me that the homeopathic spongia prevented the onset of attacks of spasmodic croup better than ipecac.

SECTION X

DISEASES OF THE LUNGS AND PLEURA

THE CHEST AND ITS DEVELOPMENT

The circumference of the chest at the nipples is smaller at birth than that of the head. The chest grows so much faster than the head that their circumferences are the same at two years, while at fifteen years the circumference of the chest is to that of the head nearly as 3 is to 2.

The shape of the infant's chest is different from that of the adult's, being rounder and shorter. The relation of the antero-posterior to the lateral diameter of the interior of the thorax at birth is as 2 is to 3, while in the adult the relation is as 1 to 2½ or 1 to 3. The ribs bend backwards much less and are more horizontal. The angle between the twelfth rib and the spine is, therefore, much wider and the angle formed where the ribs join the sternum at the ensiform, also wider. The sternum is higher by a little more than the width of the body of one vertebra. The diaphragm is also higher. There is a gradual change in the shape of the chest toward that of the adult as the child grows older, the adult relations being attained at about seven years. The final shape of the individual's chest is reached at twelve or thirteen years, any changes thereafter being simply in size.

The sternum is narrow and almost entirely cartilaginous at birth. The ribs are much shorter than in adult life, the front of the chest being almost entirely made up of the costal cartilages. The chest of the infant and young child is, therefore, very compressible and elastic. In consequence, retraction of the lower chest in inspiration often develops when there is obstruction in the upper air passages and the entrance of air into the lung is often interfered with when a baby lies on its side. Deformities are much more easily produced in it than in the chest of the older child or adult as the result of the interference with the entrance of air into the chest or of the pull of the muscles in rickets. When the pressure inside the chest is increased, as when there is an effusion in the chest or pneumothorax, the chest wall yields as a whole instead of bulging out between the ribs. The chest wall during the first few years is made up mostly of fat tissue. The muscles are very thin and when the fat tissue disappears as the result of malnutrition the covering of the thorax is not much thicker than paper.

Respiration.—The respiration is predominantly diaphragmatic in type during the first few years of life, the thoracic element not being markedly developed until about seven years. The variation in the type of respiration depending on sex is not noticeable until about ten years. There are, however, marked individual variations in the time of the development of the different types of respiration. An inspiratory recession of the epigastrium is not abnormal during the early months of life.

The rhythm of the respiration is quite irregular during the first two years and is easily disturbed from slight causes. It is almost never perfectly regular, except when the baby is asleep. Even then it may be

irregular and intermittent. Babies normally often hold their breath for a long time without apparent cause or discomfort. Not only the rhythm but the depth of the respiration varies much. Sometimes the respiration is superficial and then again deep. Sometimes, moreover, a baby will breathe more with one lung than with the other.

The rate of the respiration is highest in little babies and gradually diminishes with age. It is very difficult to give absolute figures, because the rate varies according to whether the baby or child is awake or asleep, active or passive, excited or quiet. In general, the relation of one respiration to four beats of the pulse holds throughout infancy and childhood as in adult life.

TABLE XXV

RATE OF RESPIRATION

At birth	40 to 45 per minute.
During first two years	25 per minute.
At 6 years	20 per minute.
At 10 years	18 per minute.

THE LUNGS AND THEIR DEVELOPMENT

Trachea.—The trachea is relatively large at birth and during infancy and early childhood. The mucous membrane is delicate and rich in blood vessels. It is relatively dry, because of the imperfect development of the mucous glands. It is also compressible, because of the lack of development of the elastic tissue.

Bronchi.—The mucous membrane of the bronchi, like that of the trachea, is delicate and rich in blood vessels. The proportion of the lungs occupied by the bronchi is relatively much greater in comparison with that occupied by the alveoli at birth and through infancy than in adult life. The adult relations are probably reached at five years or a little later.

Lungs.—The interstitial tissue in the lungs is relatively large at birth and throughout infancy and early childhood. There is also an insufficient development of the elastic tissue. This gradually increases, however, and is well developed at seven years. The bronchi and bronchioles are relatively large. The alveolar walls are thicker than they are later and the air spaces are in consequence smaller. The capillary system is well developed, but the large vessels are relatively small. In consequence, the lungs are richer in blood than they are later.

There is a rapid change during the first year. The alveoli increase in number, their walls become thinner, their capillaries are more firmly bound down and the epithelium becomes flatter. The adult relation of the bronchioles to the alveoli and the adult structure of the alveoli are nearly attained at five years, but the changes are probably not complete until the end of childhood.

PHYSICAL EXAMINATION OF THE LUNGS

The ideas of many physicians as to the fundamental principles of auscultation and percussion being, apparently, somewhat hazy and the terminology somewhat confused, it seems worth while to discuss these fundamental principles and to explain the terminology to be used before taking up the physical signs found in the various diseases of the lungs and pleura.

Percussion

Finger-percussion should, of course, be the only form used. When a portion of the body which does not contain air is percussed, a sound is elicited which is called "*flat.*" When a portion of the body which contains air is percussed, a clear sound is produced. If the air is enclosed in a large number of small cavities, as it is in the lungs, the sound elicited is non-tympanitic or the so-called "*normal*" lung sound. This sound may be exaggerated if the lung is partially distended, and the note is then spoken of as being "*hyperresonant.*" If the air is in a large cavity the sound elicited is called "*tympanitic.*" The pitch and intensity of the tympanitic sound vary with the size of the cavity and the character and tension of its walls. They also vary according to whether the cavity has a communicating opening or not, and, if it has, with the size of the opening. Furthermore, when the elasticity of the lung is lost, as when it is distended or partially relaxed from compression, the note is tympanitic. All the modifications of the normal lung sound to the flat sound are known as "*dull,*" and are due to the combination of the sounds from

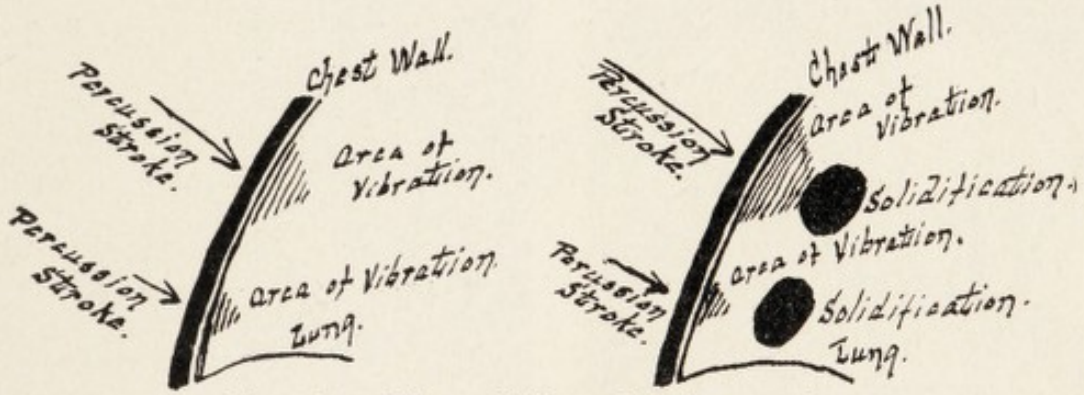

Fig. 104.—Effects of light and hard percussion.

normal lung tissue and bodies not containing air. It is evident that dullness may vary from slight to marked, according to the relative proportions of normal lung and solid bodies in the area reached by the percussion. It is also evident that various other combinations of the flat, tympanitic and normal lung sounds may be obtained.

When the chest is percussed, the intensity of the sound produced depends upon the amount of tissue reached by the percussion, that is, upon its strength. Roughly speaking, the area set in motion is an equilateral triangle, with its base at the surface, the size of the triangle depending on the strength of the percussion. Even with hard percussion, it is doubtful if the apex of the triangle is more than three inches from the surface. It is evident, therefore, that in determining the boundaries of organs the percussion stroke must be gentle, while if attempting to discover solid areas deep in, it must be hard. It is also evident that in babies and young children, in whom the parts are small, strong percussion will bring out mixed sounds from several organs and hence give unreliable results. Light percussion should almost always be used, therefore, in babies and young children.

Areas on the chest are usually described in terms of horizontal lines corresponding to the ribs and of certain arbitrary vertical lines. These lines are, in front, the sternal, which runs down the side of the sternum, the parasternal, which is midway between the sternal and the nipple lines, and the nipple, which runs through the nipple; laterally, the anterior

and posterior axillary lines, corresponding to the anterior and posterior folds of the axilla, and the middle axillary line, midway between the anterior and posterior. In the back, the only line of importance is the scapular, which runs through the angle of the scapula when the arms are hanging at the sides.

Percussion Boundaries of the Lungs.—On account of the higher position of the diaphragm the lower border of the lungs is somewhat higher in infancy than in late childhood and adult life, roughly, by about one rib. The lower border of the lung, therefore, beginning at the sternal line and running around the chest is at the fourth, fifth, sixth, seventh, eighth and tenth ribs. Owing to the relatively large size of the liver in infancy, the lower border of the lung may be a little higher than this line on the right and a little lower on the left. The lower

FIG. 105.—Lobes of the lungs. Front.

FIG. 106.—Lobes of the lungs. Back.

border gradually moves downward with the change in the shape of the chest and the position of the diaphragm, so that by seven years the adult relations are attained, namely, at the fifth, sixth, sixth, seventh, eighth, ninth and eleventh ribs. These boundaries are, of course, lines of flatness, not of dullness, which are about one rib higher and are determined by somewhat stronger percussion. As a matter of fact, it is almost impossible to locate them accurately in infancy. The lungs do not extend quite as far forward in infancy, not quite reaching the sternal border. There is, therefore, no pulmonary resonance under the sternum at this age. They also do not cover as much of the heart as they do later. The adult relations are reached at about six years. The lung-heart boundary is spoken of in the section on the heart.

Lobes of the Lungs.—The lobes of the lungs are fully developed at birth, and the relations of the lobes to the chest wall are essentially the same throughout infancy and childhood as in adult life. The divisions between the lobes run at an angle with the ribs, not horizontally, like layers of jelly cake or Washington pie, as so many physicians seem to think. The line between the upper and the lower lobes starts near the median line of the back at the level of the spines of the scapulae, runs through the fourth rib in the mid-axillary line and reaches the border of

the lung at the sixth rib in the nipple line on the left and the parasternal line on the right. The line between the upper and the middle lobes on the right side leaves the line between the upper and lower lobes at the outer border of the scapula, runs through the third rib in the mid-axillary line and reaches the border of the lung in front at the fourth costal cartilage. On the left side, therefore, the greater part of the back, almost all of the side and a very little of the front of the chest is lower lobe, while a small portion of the back and almost all of the front is upper lobe. On the right side the conditions in the back are the same. The upper part of the axilla is middle lobe, but most of it is lower lobe. Most of the front is upper lobe, with a small area of middle lobe and a still smaller area of lower lobe.

Position for Examination of the Lungs.—The easiest way to examine the front of the chest of an infant or small child is when it is lying on its

FIG. 107.—Method of holding baby for examination of back of chest.

back in someone's lap. It is less likely to be afraid than when it is on a bed or table. When the back of the chest is examined the baby should be held in someone's arms with its arms around her neck. When it is in this position the air can enter both sides of the chest freely and the baby feels safe and at ease. If the baby is too sick to be taken up, it may be turned over on its face for the examination of the back. The weight of the body on the compressible anterior wall of the chest interferes to a certain extent, however, with full respiration and makes the results less satisfactory. The back should not be examined while the baby is lying on its side, if it can possibly be avoided, because the weight of the body compresses the chest on the down side and interferes with the respiration on that side. This may lead to wrong conclusions unless due allowances are made. If, for any reason, the baby cannot be turned on its face,

the back may be examined, first, when it is lying on one side, and then when it is lying on the other. It is usually wiser to examine the back of the chest of infants and young children before the front, as they are less likely to be frightened. With care, it is often possible to examine the back without the child knowing what is being done.

The front of the chest of older children can be examined equally well when they are standing or sitting as when they are lying on the back. If they are unable to sit up, there is less chance of confusion from interference with respiration when they are lying on the face or side than there is in infants and young children. Great care must be taken, however, if they are on the side, to avoid false conclusions. Care must also be taken, when they are sitting or standing, that the parts of the chest are symmetrical, because the bunching up of muscles may modify the percussion note.

Percussion in Infancy and Childhood.—It is usually wiser to ausculti before percussing babies and young children, because they are less likely to be frightened by auscultation than by percussion. Another reason is that, in general, the results obtained in them by auscultation are more reliable than those obtained by percussion. Light percussion must always be used in babies because of the small size of the parts. The sides of the chest must be symmetrical, if possible, because if the baby is on one side the compression of that side will modify the percussion note. In older children, especially in muscular boys, the contraction of the muscles will modify the sound.

The percussion note is normally more resonant during infancy and childhood than later. Even under normal conditions, a tympanitic element is always added at the left base because of the position of the stomach, just below the lung, unless the stomach is full. When the abdomen is distended with gas, a tympanitic element may be present on both sides of the chest, especially on the left.

It is impossible to percuss the apices of the lungs in infancy and early childhood because of the small size of the parts and because the apices do not come up into the supraclavicular spaces as high as in later life. The percussion note is not at all dulled at the right apex in infancy and childhood as it is in adults. There is, instead, an area of slight dullness under the inner third of the left clavicle up to nine or ten years, although it is hard to recognize this area in infancy. It is due, in part, to the presence of the great vessels and the oesophagus on the left side and in part to the fact that the left lung does not extend as far forward as the right.

Sense of Resistance.—A sensation of more or less resistance is always felt in percussion. This depends on the presence or absence of air in and the density of the parts percussed. The skilled examiner often gets as much information from this feeling of resistance as he does from the sound which he elicits. This is not what I mean, however, when I speak of the *sense of resistance*. By this term I mean the resistance which is felt when the chest is tapped with the ends of the fingers, the two distal phalanges being flexed at a right angle on the proximal. A great deal can be learned in this way as to the conditions within the chest in early life because of the thinness of the chest walls. When the lung is normal, a sense of elastic resistance is felt. When there is solidification of the lung, the feeling of elasticity is lost and the resistance is increased. When there is an effusion in the pleural cavity, the sense of resistance is very marked, like that felt when the thigh is tapped.

AUSCULTATION

Stethoscope.—The stethoscope is, on account of the small size of the parts, far preferable to the naked ear in the examination of infants and young children. It is also important to use a stethoscope with a small bell. The diameter should not be more than three quarters of an inch. If a larger bell is used, it is impossible to get it all down on the surface of the chest of a thin baby or to pick out small enough areas. A large bell or a phonendoscope transmits the sounds from too large an area and makes it impossible to accurately locate their origin. Moreover, the bell of a full-sized phonendoscope is as large as a baby's heart and will almost cover a lobe of a lung. It is impossible with a phonendoscope, therefore, to examine a baby's heart accurately. A stethoscope with a metal bell should never be used because the end is almost always cold. This frightens the child or makes it uncomfortable.

Respiration.—The respiratory sound is made when the air passes through the larynx. This sound is known as "*bronchial.*" It is heard normally over the larynx and trachea in front and through the seventh cervical spine behind. It is not changed in character in its passage through the bronchi. When this sound is transmitted through solid tissues or liquids it is not changed in character, but may be more or less diminished in intensity. When it is transmitted through a tissue made up of many small spaces containing air, such as a lung, it is modified in character and is known as "*vesicular.*" The vesicular sound is not made by the air passing in and out of the alveoli. This can be proved by wrapping an expanded lung about a glass tube and blowing through the tube. No air enters the alveoli, but a vesicular sound is heard over the lung. If a solid tissue, such as a pneumonic lung or the liver, is wrapped about the tube bronchial respiration will be heard over the lungs or liver. Vesicular respiration is not modified in character by passing through solid tissues or liquids, but is diminished in intensity. All combinations of bronchial and vesicular respiration may be heard, according to the amount and relations of air-containing and solid tissues lying between the bronchi and the surface of the body. The term "*bronchovesicular*" is applied to all these modifications of the character of the respiration lying between the bronchial at one extreme and the vesicular at the other. It is evident that toward one end the bronchial element will predominate and toward the other, the vesicular. It is also evident that the character of the respiration and its intensity are independent of each other and due to different causes. All possible combinations of intensity and character may, therefore, be heard. If there is any question as to whether the sound which is heard is bronchial or not, it can easily be settled by comparing it with the sound heard over the larynx and trachea in front or above the seventh cervical spine behind. It goes without saying, of course, that, as bronchial respiration is heard only over solid lung, it cannot be heard over the whole of both lungs at the same time. Furthermore, the bronchial sound over the lung merely shows that there is solidification of the lung. It does not give any information as to the cause of this solidification. Another minor difference between bronchial and vesicular respiration is that expiration is relatively somewhat longer when compared with inspiration in the former.

The respiratory sound heard normally in infancy and childhood up to about twelve years is different from that heard in adults, being a modification of the vesicular toward the bronchial, that is, bronchovesicular, but near the vesicular end. It is known as "*puerile.*" This type of respira-

tion, especially if it is loud, often confuses physicians, who mistake it for bronchial. Puerile can always be distinguished from bronchial respiration by listening over the trachea. Bronchial respiration is heard normally over a wider area at the root of the lungs in the back in infancy and early childhood than in later childhood and adult life. It is almost always heard in the interscapular space and may even extend a little beyond the inner borders of the scapulae. The relative prolongation of expiration to inspiration in bronchial respiration is much less constant in infancy and early childhood and is of very little importance in comparison with the character of the sound. In adults the respiratory sound at the right apex has a little admixture of the bronchial element, and expiration is slightly prolonged. In infancy and up to late childhood the respiratory sound is the same at both apices, and the expiration is not prolonged at the right apex.

The respiratory sound is often so feeble in infancy, especially if there is disease of the lungs, that it is impossible during ordinary respiration to determine its character. When this is the case, the baby must be made in some way to cry and thus to take a long breath. If this is not done, a satisfactory examination is impossible. The respiratory sound is diminished on the down side in babies and young children when they lie on the side. Physicians often forget this and mistake the relatively loud puerile respiration on the up side for bronchial respiration.

If the bronchial sound is transmitted into a cavity it is changed in character, the change depending on the nature of the walls of the cavity. If the walls are soft, the sound is called "*cavernous;*" if they are hard, "*amphoric.*" If the cavernous or amphoric sound is transmitted to the surface of the body through normal lung tissue it is modified by the addition of a vesicular element. If the cavity is small and deep in, the cavernous or amphoric element will be obliterated by the vesicular. If the cavity is not connected with a bronchus, no air will enter it, and it will, of course, have no effect on the respiratory sound.

TABLE XXVI

TYPES OF RESPIRATION

- Respiration
  - Vesicular—Exaggerated.
  - Bronchovesicular.
  - Bronchial
    - Cavernous.
    - Amphoric.

Voice Sounds.—The voice sound is made in the larynx and, like the respiratory sound, transmitted downward unchanged through the trachea and bronchi. This sound is heard normally in the same situations as bronchial respiration and, like it, is known as the "*bronchial*" voice sound. When this sound is transmitted through solid tissues, it is not changed in character. When it is transmitted through normal lung tissue, it is changed in character and is called "*normal.*" A better term for it is "*vesicular.*" All the modifications between the bronchial voice sound and the normal voice sound are called "*bronchovesicular.*" When the bronchial voice sound is transmitted into a cavity, it is changed in character. When the wall of the cavity is hard, the sound produced is called "*amphoric;*" when the wall is soft, it is called "*cavernous.*" The intensity of the voice sound varies according to the same laws as that of the respiration. The whispered voice sometimes reveals changes which cannot be detected with the spoken voice.

Babies cannot and young children often will not speak. Reliance then has to be placed, of course, on the cry. In general, it is more diffi-

cult to recognize changes in the voice sounds than in the respiration. On the other hand, a change in the character of the voice sound can often be detected before there is any change in the respiratory sound, when there is beginning solidification in the lungs. In adults the voice sound at the right apex is changed a little from the normal sound heard at the left apex toward the bronchial. This is not so in infants or in children before adolescence.

Râles.—Râles are made in the trachea and bronchi. Similar sounds may be made in the nose and pharynx. These sounds may, in infants and young children, be transmitted downward and heard over the lungs. If they are made in the upper air passages they can be heard over the trachea, both in front and behind, and are the same on both sides of the chest, back and front. They can also be heard over the neck and sometimes over the cheeks. Râles made in the bronchi are not heard in these situations and are never exactly the same on the two sides of the chest, back and front.

There are two kinds of râles, dry and moist. *Dry râles* may be either sibilant or sonorous. *Sibilant râles* are made by the whistling of the air through small bronchi, whose lumen has been locally narrowed by swelling of the mucous membrane, or by the vibration of thin shreds of tough mucus stretched across bronchi or attached to their sides. *Sonorous râles* are made by the vibration of larger strings of tough mucus in the bronchi. They are, of course, made only in the trachea or larger bronchi. *Moist râles* are made by the bursting of bubbles or by the moving of bubbles of watery secretion over each other. They vary in size according to the size of the bubbles and are classified as fine, medium and coarse. Coarse, moist râles can, of course, only be made in the larger bronchi. Fine, moist râles can be made in bronchi of any size. In a general way, but only in a general way, the size of the râle corresponds to the size of the bronchus in which it is made. If moist râles are transmitted to the ear through solid tissue they often have a high-pitched or bronchial character. Râles may also be made in cavities. If the cavities have hard and stiff walls, they may have a metallic or ringing sound. Both dry and moist râles may be heard in both inspiration and expiration, but are usually more numerous in inspiration.

Very fine, moist râles are sometimes heard when there is edema of the lungs with fluid in the alveoli or when there is a little sticky secretion in the alveoli, as at the beginning or end of lobar pneumonia. These râles are probably made in the alveoli and are probably due to the separation of the alveolar walls with inspiration. They are, at any rate, heard only in inspiration. A similar sound, often heard in the lower backs when a deep inspiration is taken after a long period of quiet breathing, is probably due to the same cause. These râles are called crepitant or subcrepitant. "*Crepitant*" is the better term. They can be closely imitated by rubbing the hair between the fingers.

TABLE XXVII

RÂLES

- Râles
  - Dry
    - Sibilant
    - Sonorous
  - Moist
    - Fine
    - Medium
    - Coarse

  } Made in bronchi.
  - Crepitant—Made in alveoli.

Friction Sounds.—These are made by the rubbing together of the inflamed pleural surfaces during respiration and are usually much more marked in inspiration than in expiration. They may also be due in the precordial area to the movements of the heart. They vary from a soft, shuffling sound, heard with difficulty, to a loud, rough, leathery rub. Although infants and young children pathologically have inflammation of the pleura, especially with pneumonia, just as often as older children and adults, pleural friction sounds are hardly ever heard in them. Friction sounds are just as common in middle and late childhood as in adults.

Tactile Fremitus

By tactile fremitus is meant the sensation felt by the hand placed on the chest wall when the patient speaks or cries. The sensation is due to the vibrations of the glottis, which are transmitted down the trachea and bronchi and through the intervening tissues to the hand. Normally the tactile fremitus in children is essentially the same on the corresponding parts of the two sides, although it may sometimes be stronger on the right. The stronger the voice, the stronger the fremitus. The fremitus is more marked when transmitted through solidified than through normal lung tissue. It is diminished or abolished by fluid. "Forty-four" gives better results than any other numbers or words.

It is much more difficult to determine the tactile fremitus in infants and young children than in older children and adults. They refuse to speak or do not speak loudly enough. In such instances the fremitus must be determined from the cry. Sometimes the voice is so thin that no fremitus can be detected. The elasticity of the chest wall also modifies the fremitus and may lead to erroneous conclusions. In general, the tactile fremitus is of relatively little value in early childhood in comparison with auscultation, percussion and the sense of resistance. It is of more value in late childhood.

ACUTE TRACHEITIS

Tracheitis is almost always secondary to nasopharyngitis or laryngitis or a part of bronchitis. It may, however, be primary. It is caused by the same organisms as bronchitis.

Symptomatology and Prognosis.—The symptoms of tracheitis, outside of the general symptoms of fever and intoxication, are a sense of soreness or pain under the sternum, a feeling of constriction of the chest and a harsh, unproductive cough. Babies and young children being, of course, unable to describe their sensations, the only characteristic symptom in them is a dry cough. When tracheitis is a part of bronchitis the symptoms are obscured by those of the bronchitis. There are usually no physical signs, but sometimes an occasional sonorous or coarse, moist râle may be heard under the sternum. Tracheitis is usually not of serious import, but, if not a part of bronchitis, is likely to lead to it. In some instances, however, the inflammation may be very severe and the swelling of the mucous membrane so great as to cause obstruction to respiration and even death from asphyxiation. In these cases the causative organism is usually the streptococcus hemolyticus.

Treatment.—The treatment is the same as that of bronchitis. In the cases due to the streptococcus hemolyticus, in which there is so much swelling of the mucous membrane and obstruction, treatment is of little avail. Even tracheotomy does no good, as the obstruction extends further down than the tube can reach.

FOREIGN BODIES IN THE TRACHEA AND BRONCHI

About two thirds of the cases of foreign bodies in the air passages occur in children, presumably because they are so apt to put and keep all sorts of objects in their mouths. They are apt to forget, moreover, that they are there and to laugh and breathe deeply, thus inhaling them.

There is almost always an attack of strangling and coughing when a foreign body is inhaled. It is, however, seldom coughed up at this time. If it is small enough to pass through the larynx, it is usually small enough to pass through the trachea. If it does not, death occurs in a few minutes from suffocation. Sharp and irregularly shaped objects may, however, even if small, stick in the trachea. If the foreign body gets through the trachea it most often passes into the right primary bronchus, because that is larger than the left, being almost as large as the trachea, and deviates much less from the long axis of the trachea. The axis of the trachea falls, moreover, to the left of the division between the right and left bronchi. How far it goes depends upon its size and, if it is sharp or rough, where it happens to stick. It seldom, however, gets far beyond a secondary bronchus and seldom into the middle lobe. If the foreign body is light, it may possibly be soon coughed up; if it is sharp, coughing fixes it more firmly; if it is heavy, its weight prevents it from being moved; if it occludes a bronchus it cannot be coughed up, because the air cannot get behind it. If it is not coughed up soon, it almost never is later.

Even if the foreign body is not coughed up, the strangling and coughing soon stop, although a troublesome cough may continue. There are no abnormal physical signs unless a large bronchus is plugged, when there is diminished or absent respiration in one lobe. In such cases, the area which is cut off becomes solidified, partly from the retention of secretions and partly from atelectasis. It becomes dull or flat on percussion. The respiratory and voice sounds are absent or diminished because no air enters the lung. They may be either bronchial or vesicular, according to the source of the sound which is transmitted through the solidified area. Tactile fremitus is absent and there are no râles. An abscess is almost certain to develop with the appearance, of course, of the constitutional symptoms of confined pus, irregular temperature and leucocytosis.

If the foreign body does not occlude a bronchus there is usually a quiescent period, lasting days, weeks or months, ordinarily weeks, but sometimes many months. In rare instances, if the foreign body is a particle of food, it may be broken down and coughed up without doing any serious or permanent damage. If the foreign body is a pin or something sharp, which sticks into the bronchial wall and becomes fixed, the secretions are not much increased. If the foreign body is metallic, but not sharp, there is a moderate increase in the secretions. How much damage the increase in secretion does, depends on how much the foreign body interferes with the clearing of the bronchi. If the foreign body is not metallic, it usually causes a marked increase in the secretions. This is especially true if it is a piece of peanut shell, one of the objects most often inhaled. The increase in the secretions eventually leads to a secondary infection of the bronchial mucous membrane, which, in turn, may lead to peribronchitis and bronchiectasis or to atelectasis and bronchopneumonia with, eventually, the formation of an abscess. The foreign body may be coughed up at this time. Usually, however, it is not. It is conceivable that the foreign body may finally become encap-

sulated. This probably almost never occurs. The physical signs of bronchopneumonia, bronchiectasis and abscess of the lung from foreign bodies are the same as when these conditions are due to other causes. There is, moreover, nothing characteristic about the constitutional symptoms when these conditions are due to foreign bodies.

Diagnosis and Treatment.—The physician seldom sees the child during the initial period of coughing and strangling. If he does, he should not encourage coughing, but should take measures to restrain it. If there is any reason to think that a child may have inhaled a foreign body, the chest should be examined at once with the Roentgen ray, remembering that many of the foreign bodies which may be inhaled are not of sufficient density to be shown in this way. If the Roentgen ray shows that a foreign body has been inhaled, an attempt should be made at once to remove it. If possible, this attempt should be made by someone skilled in the use of the bronchoscope. If the Roentgen ray does not show any foreign body, but coughing continues or soon develops without any other apparent cause, bronchoscopy should be done and the foreign body removed, if present.

Whenever children develop peculiar pulmonary conditions and show physical signs in the lungs for which it is difficult to account, the possibility that they may be due to a foreign body, which was inhaled sometime in the past, must always be borne in mind. Careful inquiry should be made as to whether the child has been in the habit of putting things in its mouth or has had a severe attack of strangling and coughing in the past, as might be the case if it had inhaled a foreign body. Older children may remember such an attack, although sometimes they may have forgotten it or have thought that the object was swallowed. Young children will, of course, not understand. A slow development of the symptoms and the localization of the physical signs in one part of one lung are rather suggestive. A diminution in the respiratory sound out of proportion to the other physical signs is also suggestive of a foreign body. The pressure of an enlarged gland on the outside of a bronchus may give the same physical signs and symptoms. I have seen several cases of this sort. The onset may be sudden, however, and suggest that of pneumonia, as happened in one of my cases which was due to the inhalation of the metal tip of a top some weeks before. Whenever there is any possibility that the trouble in the lungs may be due to a foreign body inhaled in the past, the chest should be examined with the Roentgen ray. If this shows that there is a foreign body in a bronchus, it must, of course, be removed at once. If it does not show a foreign body, but the condition still suggests a foreign body as the cause, bronchoscopy should be done and the foreign body removed, if found. If an abscess or a large bronchiectatic cavity develops, the treatment is the same as if they were due to some other cause.

ACUTE BRONCHITIS

By this term is meant the ordinary acute catarrhal bronchitis. Acute suppurative bronchitis is a very uncommon modification of catarrhal bronchitis, in which, for some reason, the secretion in the bronchi is more purulent than usual. I have never seen a case of fibrinous bronchitis in childhood.

Etiology.—Acute bronchitis is due primarily, of course, to microörganisms. These are, in most instances, the various forms of staphylococci, streptococci and pneumococci, but may be the influenza bacillus,

the micrococcus catarrhalis or some other organism. Bronchitis is usually the result of a descending infection from the upper air passages. It is also a very constant complication of measles and whooping-cough as well as of typhoid fever. It may, however, apparently be primary. In such cases it usually follows exposure or chilling of the body or chest in some way. Such exposure or chilling presumably brings on bronchitis by causing some change in the circulation in the bronchial mucous membrane which weakens the resistance to infection by bacteria. The inhalation of dust or irritating gases may decrease the resistance and cause bronchitis in the same way.

Pathology, Symptomatology and Physical Signs.—When acute bronchitis is due to exposure, chilling or the inhalation of irritating gases, the onset may be quite acute with a chill, or more often vomiting, followed by a rapid rise in temperature. When it is secondary to a descending infection or a complication of some other disease, the onset is usually shown only by a further rise in the temperature and an increase in the cough and constitutional symptoms.

First Stage.—In the beginning of acute bronchitis the mucous membrane of the bronchi is red, swollen, hot and dry. The cough is, therefore, dry and unproductive. There is a sense of soreness or burning under the sternum and of constriction of the chest. Babies and young children are, of course, unable to describe these sensations. There being very little secretion, very few râles are heard, and, if any are heard, which are due to secretion, they are sibilant or sonorous, because the first secretion is sticky. Sibilant râles may also be heard if the swelling of the mucous membrane narrows the lumen of the smaller bronchi. When the bronchioles are generally involved, as they are in the so-called "*capillary bronchitis*" of infants, no râles are heard, but the respiratory murmur is much diminished and there is marked dyspnoea and cyanosis because the entrance of air into the alveoli is prevented. There is no reason, of course, for any change in the percussion sound or in the character of the respiration, voice sounds or tactile fremitus in the first stage of bronchitis.

Second Stage.—After from one to three days the mucous membrane of the bronchi becomes somewhat less reddened, somewhat less swollen and hot, and the glands begin to secrete freely. The sense of burning and heat, as well as the sense of constriction, consequently cease. Microscopically, the mucous membrane shows enlargement of the mucous glands and numerous leucocytes passing through it. The secretion, at this time, is watery. Numerous moist râles are heard, therefore, over the whole chest, back and front. These râles vary in size according to the size of the bronchi in which they are made. In the very beginning of this stage there may be a few dry râles, but they quickly disappear. Some of the bronchi may be occluded by secretion. If this happens in a fairly large bronchus, the respiration is locally diminished in intensity until the bronchus is cleared. Small areas of atelectasis often develop when the bronchi are plugged. They usually, however, do not give any physical signs, because a compensatory emphysema almost always develops at the same time, which prevents the atelectatic areas from causing any dullness. There are, therefore, in the second stage of bronchitis, as in the first, no changes in the percussion note or in the character of the respiration, voice sound or tactile fremitus. The secretion being watery, the cough becomes loose and productive. Babies and young children almost always swallow the sputum. If they spit it out, it is thin and watery with, of course, some mucus.

Third Stage.—After three or four more days the redness of the mucous membrane has almost disappeared and the swelling is much less. The secretion becomes thicker and more purulent. Microscopically, the mucous membrane shows less enlargement of the mucous glands and more leucocytes passing through it. There is often desquamation of the superficial epithelium. The cough is still loose, but less productive. The sputum is thick and purulent. Râles are still heard over the chest, both back and front, but they are less numerous and sonorous or sibilant. The other physical signs are the same as in the second stage.

Bronchitis may, of course, be of any grade of severity. It may be so mild that there is but little elevation of temperature, very few constitutional symptoms and very few physical signs. On the other hand, it may be very acute, very severe, with a high temperature, marked constitutional symptoms, marked physical signs and end in death. It is useless to attempt to describe all the different varieties. One special form, should however, be mentioned, that is, the so-called capillary bronchitis of infants. This is a very uncommon variety, but does occur. In it the bronchioles are chiefly affected. The mucous membrane of the bronchioles is so much swollen that air cannot get into the alveoli. Consequently, there is marked dyspnoea and cyanosis. Respiration is, of course, rapid because of the need for air. There are no physical signs, because the bronchioles are so blocked that no air can whistle through them and there is no secretion. Death is likely to occur in these cases in the first twenty-four or forty-eight hours. If they get by this period, there is a fair chance that they may recover. Another variety which is, unfortunately, not very uncommon in infancy and early childhood is that in which the secretion during the second stage is very profuse and the infant is unable to clear the bronchi by coughing. The respiration becomes very rapid, cyanosis develops, the heart weakens and death often results from asphyxiation.

The borderline between severe bronchitis and bronchopneumonia is very indefinite. When the smaller tubes are involved there is very likely to be a peribronchitis and an extension of the process to the alveoli. When this happens, the condition is, of course, bronchopneumonia. When the bronchopneumonic areas are small they have no effect on the physical signs. This is due in part to the fact that there is almost always more or less compensatory emphysema about the bronchopneumonic areas. There is usually, however, an increase in the severity of the symptoms.

Temperature, Pulse and Respiration.—There is nothing whatever characteristic about the temperature in bronchitis. It is always elevated, but the elevation may be slight or marked. It is quite likely to be irregular. It usually begins to fall with the beginning of the second stage, but often does not disappear entirely until convalescence is well established. The pulse rate usually corresponds to the degree of the rise of temperature. The rate of the respiration in mild cases is but little more increased in proportion than that of the pulse, whereas, in severe cases it is much increased.

The constitutional symptoms depend largely on the temperature and the amount of interference with respiration. Intoxication, as a rule, plays but little part in their production.

Prognosis.—The prognosis varies, of course, according to the severity in the individual instance and the age of the patient. In children it is usually not a serious disease, but may cause bronchopneumonia and be fatal. Bronchitis always has serious possibilities in infancy as it not

infrequently leads to bronchopneumonia, which is a very serious and fatal disease at this age. When the process is located in the bronchioles, as it sometimes is in babies, it is usually fatal. Babies and young children sometimes drown in their own secretions in the second stage in spite of anything that can be done to prevent it.

Treatment.—The most important part of the treatment of bronchitis, as of almost all diseases in infancy and childhood, is the preventive. If babies and children are cared for in the manner described in the treatment of nasopharyngitis and are put to bed whenever they have a "cold," they will seldom develop bronchitis, and, if they do, it will seldom be of a severe type.

When a child is apparently "coming down" with bronchitis it is possible that the further development of the disease may be prevented. It is also possible that, if it does not develop, it would not have, even if no treatment had been given. The child should be given a hot bath, put to bed and covered warmly, and given a drink of hot milk or soup, so that it will perspire freely. Further relaxation may be produced by giving sweet spirits of nitre, in doses of from five to twenty drops every hour, or of tincture of aconite, in doses of from one eighth of a drop to one drop every fifteen minutes for an hour and then every hour, according to the age of the child. One or two doses of Dover's powder of from 1¼ to 2½ grains, according to the age, will sometimes work better in older children. It is probably useless to give a cathartic, as it does not seem reasonable to suppose that congestion in the bronchial mucous membrane can be materially relieved in this way. It is, however, advisable to give a mild laxative.

Whenever a baby or child has the bronchitis, it should be put to bed and kept there until the temperature is normal and the inflammation of the bronchi has subsided. The air in the room should be kept fresh, but should not be cold. Cold air increases the discomfort and cough in the first stage of bronchitis. The temperature of the room should be kept at from 64° F. to 66° F. In the early stage of bronchitis the bronchial mucous membrane is dry. When the air is moistened, it makes the patient more comfortable in that it relieves, to a certain extent, the dryness of the mucous membrane. Water should, therefore, be kept boiling constantly in the room. It is advisable to add a tablespoonful of the compound tincture of benzoin to the boiling water from time to time, as the vapor of benzoin seems to allay irritation of the bronchial mucous membrane. It is useless to attempt to use tents or canopies with young children or to expect them to hold their heads over a pitcher of boiling water. The pain and soreness under the sternum can be helped by the application of an electric heating pad, a hot water bottle, flaxseed poultices or a mustard paste. It must never be forgotten, however, that the skin of a child is very sensitive and that it is very easy to burn a child with a hot water bottle or a poultice. A mustard paste is made by mixing one part of mustard and three parts of flour with warm water and the white of an egg. It should be spread one eighth of an inch thick on a piece of linen or cotton and covered with cheesecloth. Great care must be used not to leave it on too long. It should be removed as soon as the skin is definitely reddened. The skin should then be greased with vaseline or well powdered.

In the first stage of acute bronchitis the bronchial mucous membrane is swollen, hot and dry. There is very little secretion. The râles which are heard are dry. Drugs are needed which will diminish the congestion of and relax the mucous membrane and thus increase the secretion. Such drugs are the so-called "sedative" expectorants. These are ipecac, tar-

tar emetic, wine of antimony and apomorphine. The alkaline salts also have a somewhat similar action. Tartar emetic, wine of antimony and apomorphine are dangerous drugs and the line between the medicinal and the poisonous doses is very narrow. Ipecac, on the other hand, is not a dangerous drug even when given in large doses, and the line between the medicinal dose and the dose which will cause disagreeable symptoms is a very wide one. The alkaline salts are not as efficient as ipecac and considerable doses are required. Ipecac, therefore, is the only one of these sedative expectorants which it is safe and advisable to use with babies and children. The best preparations are the wine and the syrup, the dose of which is the same. Neither the wine nor the syrup of ipecac has a bad taste, in fact, the taste is rather pleasant. It should not, therefore, be put up in a mixture with syrups, but should be given plain in water. The dose of the wine or syrup of ipecac for babies and children is, roughly speaking, about one drop for each year of age. The individual susceptibility to ipecac is, however, very variable. Some children can take much larger doses and for others the dose must be diminished. The object for which the ipecac is given is to relax the bronchial mucous membrane and to increase the secretion. It should, therefore be given frequently enough to keep up a continuous action, that is, at least every two hours. As large a dose should be given as can be without causing nausea. Ipecac is not given for the symptom, cough.

If there is much cough, which is not relieved by moist air, compound tincture of benzoin and applications to the chest, it is advisable to give something to allay the cough. Bromides will sometimes do this. In general, however, it is necessary to use some preparation of opium. The best and only safe preparation to use in babies and young children is paregoric. It is practically impossible to do any harm with this preparation, unless it is used absolutely recklessly. The dose of paregoric is from 3 to 10 drops for a baby and from 10 drops to ½ a teaspoonful for a child, the dose varying, of course, according to the age. A teaspoonful of paregoric contains about one fourth of a grain of powdered opium. For older children codeine or the phosphate or sulphate of codeine, in doses of from $\frac{1}{16}$ of a grain to ½ a grain, may be used according to the age and individual susceptibility of the child. The hydrochloride of heroine may also be used in doses about one-half as large as those of codeine and its salts. Whenever heroine or codeine are used a small dose should be given first, because so many children are susceptible to opium. If that is not sufficient, it may be increased later. Paregoric and other preparations of opium should be used simply to relieve the symptom cough. They should not be given, therefore, when they are required for the cough, at any stated intervals, except that there should be a minimum interval at which they may be given.

It is, or should be, evident that, as ipecac and other expectorants are given in order to get a continued action on the bronchial mucous membrane, while the various preparations of opium are given to relieve the symptom cough, they should never be combined in the same prescription. It is always wrong to combine drugs which are given for different indications in one prescription, because, if this is done, too little of one or too much of the other is certain to be given. Any prescription which contains an expectorant and any preparation of opium shows that the person who wrote the prescription was either careless or thoughtless.

When the bronchial mucous membrane begins to relax and to secrete freely, as it does in the second stage, the sense of pain and soreness under

the sternum and of constriction of the chest ceases, the cough becomes productive, and moist, instead of dry, râles are heard over the chest. It is not necessary, therefore, to continue applications to the chest or to keep the air of the room moist or impregnated with the vapor of compound tincture of benzoin. The time for sedative expectorants has also passed. The mucous membrane is relaxed and secreting. Some drug is now needed which will stimulate the mucous membrane to further secretion and hasten its return to normalcy. Such drugs are the so-called "stimulant" expectorants. Among these may be mentioned senega, terebene and the salts of ammonium. The ammonium salts are much less irritating to the digestive tract than the other drugs and the chloride is less irritating than the carbonate of ammonium. Chloride of ammonium, therefore, is the best drug of this class for children. It tastes badly and many children object to it. The taste is disguised better by the fluid extract of licorice than by anything else. It should be prescribed, therefore, in the proportion of one grain of ammonium chloride to one minim of the fluid extract of licorice, in water. The dose is from ¼ of a grain for babies to 2 grains for older children. Being given for its action on the bronchial mucous membrane, it should be given continuously at intervals of no longer than two hours. The cough is usually less troublesome in the second stage. Furthermore, it is a productive cough. It is not wise, therefore, to limit it, unless it is excessive. In fact, it is possible to do much harm and even to cause death in babies and young children by stopping or diminishing the cough in the second stage of bronchitis, when the secretion is excessive. Paregoric and the other preparations of opium should, therefore, be used with the greatest caution in this stage.

When the second stage of the bronchitis is over and the third begins, which is shown by the change in the character of the râles from moist to dry, mostly sonorous, clinical experience shows that one of the so-called "alterative" expectorants, whatever "alterative" may mean, is more useful than the sedative or stimulant expectorants. The only drugs of this class which are suitable for babies and children are the salts of iodine and its combination with hydrogen, hydriodic acid. The iodide of potash is the salt most commonly used. It may be prescribed as a saturated solution in water, the required number of drops being given in water or milk, or in combination with the essence of pepsin. It is less likely to disturb the digestion when given with essence of pepsin. The dose is from one fourth to one half of a grain for babies and from one to five grains for children. As the iodides, although quickly absorbed, are slowly eliminated, it is not necessary to give frequent doses in order to get a continued action. A dose once in four hours or even three times a day is sufficient. Hydriodic acid is usually given in the form of the syrup of hydriodic acid. A teaspoonful represents about one grain of the iodide of potash. Most physicians are apparently not aware how little iodine it contains and hence give altogether too small doses to get any effect. If given in sufficient doses, the syrup with which it is made is likely to disturb the digestion.

None of the "cough syrups" which are so commonly used have any action whatever on the bronchial mucous membrane. Syrup of tar, syrup of wild cherry, syrup of tolu, and all the other syrups, including syrup of squills, are entirely inert and serve only to disturb the digestion. No one thinks of giving a well baby or child a teaspoonful of syrup every hour or two in order to upset its digestion. Why is it any more reasonable to do this when a child is ill? It certainly is not. It is simply add-

ing insult to injury. In fact, there is no reason why any syrup should ever be given to babies and children as a medicine. Most drugs do not taste badly, and, if they do, the syrup usually does not disguise the taste very well or even makes it worse. Children will take drugs, even if they have a bitter or an acid taste, if it is not suggested to them that the taste is bad and if they are properly brought up.

There are no applications which can be applied to the chest which influence in any way the pathologic process in the bronchi. Hot applications and poultices, as described in the treatment of the first stage of bronchitis, make the patient more comfortable, but do not hasten recovery. Camphorated oil probably does no harm and, when it is put on the chest, mothers, nurses and some physicians are comforted, because they are constantly reminded by the smell that something is being done. It is better than goose grease on this account and the odor is pleasanter than that of onions.

When the secretion in the bronchi is excessive and the baby is being drowned in its own secretions, active treatment must be instituted either to diminish the secretion or to aid the baby to clear out the bronchial tubes. Atropine or the sulphate of atropine given by mouth or subcutaneously, according to the urgency of the condition, sometimes diminishes the secretion materially, but in other instances does not help at all. The dose for little babies is from 1–1000 of a grain up and for older children from 1–500 of a grain up. The simplest way to make babies cry is to dip them alternately in water from 105° F. to 110° F. and from 65° F. to 75° F., as is done in resuscitating new-born infants. This procedure usually makes them cry, breathe deeply and cough and in this way get rid of the excessive secretion. If this method of treatment is not successful, they should be given one or two teaspoonfuls of the wine or syrup of ipecac to make them vomit and in this way clear out the bronchial tubes. The continuous administration of oxygen in this condition as long as there is cyanosis is of some benefit, in that the babies in this way get more oxygen in the little air which enters the alveoli.

Sometimes adrenalin chloride, or some other preparation of epinephrine, given subcutaneously, in doses of from 1 to 5 minims of the 1–1000 solution, diminishes the severity of the symptoms in the capillary bronchitis of infants, apparently by relieving a complicating spasm of the bronchioles. Oxygen should be given continuously in these cases also as long as there is cyanosis.

The temperature in bronchitis is seldom high enough or persistent enough to require treatment by cold applications externally. The general symptoms of discomfort from fever and intoxication may be relieved by aconite, phenacetin and salol, and aspirin, as described in the treatment of acute tonsillitis. Vaccines are inadvisable, because it is an acute disease and it is difficult to get cultures of the causative organisms. Treatment with stock vaccines is illogical and unscientific. If convalescence is slow, a change of climate will do far more good than vaccines or tonics. Stimulants, if necessary, should be used in the same way as in other diseases.

CHRONIC BRONCHITIS

Chronic bronchitis may be a secondary condition in chronic valvular disease of the heart, tuberculosis of the lungs and chronic interstitial pneumonia. It is usually, however, the result of repeated attacks of acute bronchitis. It is especially likely to develop in debilitated and

malnourished children who live in unhygienic surroundings. It is often a complication of rickets, presumably partly as the result of the deformity of the chest in this disease, partly as the result of the lowered nutrition and partly because of the recurrent attacks of acute bronchitis which are so common in rickets. Chronic bronchitis may be associated with asthma or bronchiectasis. On the whole, it is uncommon in childhood, and when it occurs, is usually not of a very severe type.

Pathology.—The pathologic changes are usually slight, seldom going beyond a mild catarrhal inflammation. In some instances, however, more marked changes, such as atrophy and ulceration, may develop as in adults. Chronic emphysema, so common in adults, seldom accompanies it in childhood, but, in rickets, when the chest is deformed, limited areas of emphysema are not uncommon. In infants and young children emphysema is not infrequent in the upper anterior portions of the lungs. Chronic enlargement of the tracheobronchial lymph nodes is not uncommon.

Symptomatology.—The chief symptom of chronic bronchitis is cough. This may be hardly noticeable, however, except during acute exacerbations. It is more troublesome at night and in the early morning and is worse in the winter than in the summer. There is, as a rule, nothing characteristic about it, but it sometimes occurs in paroxysms which resemble those of whooping-cough. There may be little or no sputum, while, in other instances, it is fairly profuse. Bronchorrhoea, is, however, most unusual in early life. The sputum may be almost entirely made up of mucus or be either mucopurulent or purulent. The expectoration is almost never foul, however, as it so often is in adults. If it is, it is almost always because there is an associated bronchiectasis.

The general health is often but little affected. The children are usually thin and somewhat pale, but do not, as a rule, look or act seriously ill. They are quite likely to be a little short of breath on exertion. The temperature is usually but little, if at all, elevated, except in acute exacerbations.

Physical Signs.—Babies are quite likely to show some enlargement of the upper portion of the chest in front, with some hyperresonance. Otherwise, there are no changes in appearance or on percussion. The character of the respiration is not changed, but in severe cases with emphysema it may be diminished in intensity. Not infrequently râles may be absent for a time, but, as a rule, some can be heard. They may be either moist or dry, according to the character of the secretion, and may be of any size. There are, of course, no changes in the vocal resonance or tactile fremitus.

Diagnosis.—Chronic bronchitis is often mistaken for pulmonary tuberculosis. In chronic bronchitis the physical signs are those of bronchitis, with, perhaps, those of mild emphysema. There are never any signs of solidification. In pulmonary tuberculosis there are always some signs of solidification and the signs of bronchitis are usually localized, not general. The temperature is more often elevated in tuberculosis and usually ranges higher than in bronchitis. The Roentgen ray shows signs of solidification in tuberculosis, but nothing or nothing more than peribronchitis in chronic bronchitis. Evidences of tracheobronchial adenitis may be seen in both. A negative tuberculin test is strong evidence against tuberculosis, but a positive test does not prove that the pulmonary symptoms are due to tuberculosis, because the positive reaction may be due to some small, localized tuberculous focus elsewhere in the body.

Prognosis.—Most of the mild and moderately severe cases of chronic bronchitis recover in time, if they can receive the proper treatment. The more severe cases may persist for many years before death occurs, the course being much like that in adults.

Treatment.—The first element of treatment, of course, is to remove the cause, that is, to cure the rickets, relieve the malnutrition and put the child in proper hygienic surroundings. Cod liver oil is probably of some benefit, especially in the cases which are secondary to rickets. Change of climate, preferably to one which is high and dry, does more good than anything and everything else. If that is impossible, the causes of acute bronchitis should be avoided. Great care should be taken about sending children out of doors when there is much dust or smoke, when the wind is cold, or when the air is cold and damp. The iodide of potash probably

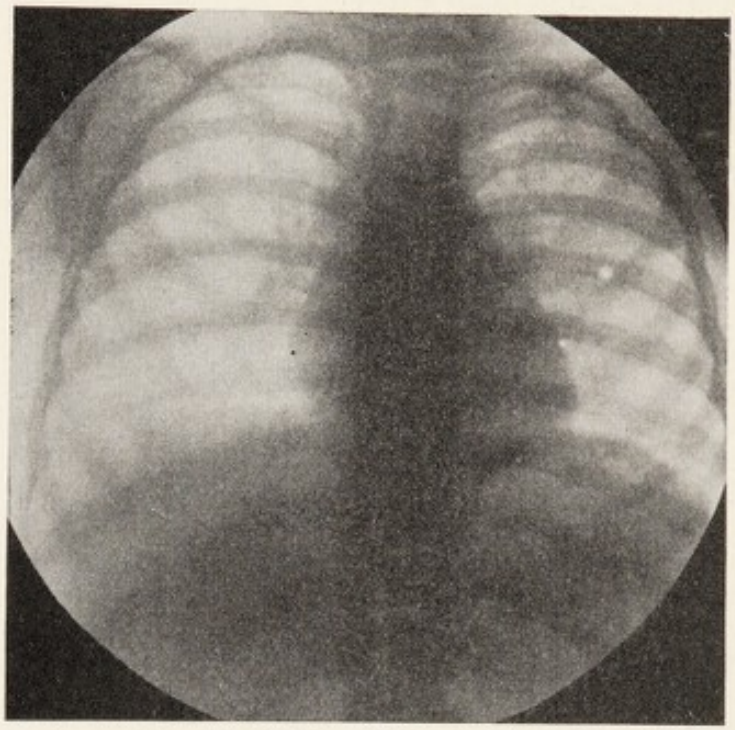

FIG. 108.—Tracheobronchial adenitis in bronchitis.

helps more than any other drug. It should be given in the same way as in the third stage of acute bronchitis. I have seen very little help from the various balsams or creosote, guiacol and their preparations, when given internally. It is very difficult to get young children to use these drugs properly by inhalation, but they may be used in the same way as in adults. It must never be forgotten that when there is much secretion in the bronchi the use of any preparation of opium is always attended with danger. This is especially true in young children. Vaccines, whether "stock" or autogenous, seldom do any good, but are worth trying in obstinate cases.

BRONCHIECTASIS

Congenital bronchiectasis is a pathologic curiosity. I have never seen a case. It is almost always limited to one lung or to one lobe of one lung and is, therefore, compatible with life, even for a long time. It often passes unrecognized for many years. The physical signs are those of other types of bronchiectasis. It should be easily recognizable with the Roentgen ray.

All the varieties of bronchiectasis, cylindrical, sacculated and spindle-shaped, may develop in children. The process may be localized or general.

Etiology.—Acute general cylindrical bronchiectasis usually develops as the result of long-continued, excessive cough, as, for example, in whooping-cough. There are usually no symptoms and the condition cannot be recognized on physical examination. Recovery is the rule after the cause has been removed. Bronchiectasis sometimes develops in childhood as the result of peribronchitis in bronchopneumonia but seldom results from peribronchitis in chronic bronchitis in childhood. Localized cylindrical or sacculated bronchiectasis may result from local obstruction in one of the larger bronchi as the result of partial obstruction from a foreign body or the pressure of an enlarged gland on the outside of the bronchus. The most common cause of bronchiectasis in childhood is chronic interstitial pneumonia. In such instances, of course, the process is limited to one lung. It may be either localized or general and either cylindrical or sacculated. It is more likely to be sacculated. Bronchiectasis also sometimes develops as a part of a tubercular process in the lungs.

Pathology.—There are practically no changes in either the bronchial wall or mucous membrane in acute general dilatation of the bronchi. In chronic bronchiectasis the changes are those found in chronic bronchitis. If the secretions are retained in the sacculated form there may be inflammation, ulceration and destruction of the mucous membrane or even of the bronchial wall as the result of secondary infection. In other cases there may be atrophy of both wall and mucous membrane.

Symptomatology.—There are no symptoms by which acute general dilatation of the bronchi can be recognized. In chronic cases, if of moderate severity, the symptoms are indistinguishable from those of the chronic bronchitis which has caused it or accompanies it. If there are large, sacculated bronchiectases, considerable amounts of sputum may be coughed up at intervals. The sputum in such cases may be foul, putrid or gangrenous. Cases of this sort are, however, much less common in childhood than in adult life.

Physical Signs.—The physical signs are the same as those of chronic bronchitis, except when there are large, sacculated bronchiectases. In such cases the signs are those of a cavity which at times is full and at others empty. Even in fairly marked cases the physical signs of bronchiectasis may be disguised by those of an associated emphysema.

Diagnosis.—The diagnosis of acute general cylindrical bronchiectasis is impossible. Bronchiectasis may be suspected when, in the course of chronic bronchitis, the sputum is more abundant and more foul than is ordinarily the case. It is almost certain when the signs of cavities develop in chronic interstitial pneumonia. It is often very difficult to distinguish between a large bronchiectasis and an abscess of the lung. In general, the onset of an abscess is more acute and develops quickly after an operation for the removal of tonsils or adenoids or an empyema. Either may develop after the inhalation of a foreign body. If, after pneumonia, the process in the lungs is localized, an abscess is the more probable; if the whole lung is involved, there is more likely to be a bronchiectasis. If the pneumonia was due to influenza, bronchiectasis is more common than abscess.

Prognosis and Treatment.—Recovery is usual in acute cylindrical dilatation of the bronchi. When bronchiectasis is the result of chronic

bronchitis, recovery is the rule, if the bronchitis can be cured. If the bronchiectasis is due to the inhalation of a foreign body, there is a fair chance for recovery if the foreign body is removed or is coughed up. If the bronchiectasis is due to chronic interstitial pneumonia, there is no hope for improvement. The treatment of bronchiectasis is essentially the same as that of chronic bronchitis, that is, by general measures, change of climate and the administration or inhalation of the balsams or of creosote and its preparations. When there is a large cavity which is near the surface of the lung, operation should be considered. Sometimes it is successful. I have never seen a metastatic brain abscess in childhood as the result of bronchiectasis.

BRONCHIAL ASTHMA

The term bronchial asthma should be applied only to a symptom-complex characterized by peculiar periodic attacks of dyspnoea. These attacks come on suddenly and last hours or days. In them the respiration is wheezy, expiration is relatively prolonged and numerous dry râles are heard in the chest, but the temperature is not elevated. The sputum is at first scanty and tenacious and contains Curschmann's spirals. As the attack lets up, the râles become moist, the sputum abundant and mucopurulent and the Charcot-Leyden crystals may be found in it. The blood shows a marked eosinophilia. These peculiar attacks are presumably due to spasm of the bronchi and bronchioles with more or less intense congestion and swelling of the bronchial mucous membrane. They are seldom seen in children under five years of age, but become progressively more common as the age increases. Infants and young children, however, very frequently develop in the course of bronchitis symptoms which are quite similar to those in the attacks described. There is more or less dyspnoea, expiration is prolonged, many dry râles are heard and there is but little sputum. The temperature, however, is elevated. The sputum is not characteristic and Curschmann's spirals and the Charcot-Leyden crystals are almost never found. There is, moreover, usually little or no eosinophilia. The peculiar characteristics of the respiration are presumably due to the same conditions in the bronchi, spasm and swelling of the mucous membrane. This condition is also usually called asthma, but probably really does not deserve this name, as its etiology is presumably not the same and its course and prognosis quite different. I shall, however, take it up under this name.

Bronchitis with Asthmatic Manifestations

Babies and young children sometimes, at or soon after the onset of attacks of acute bronchitis, have great difficulty in breathing. The respiration is superficial. There is difficulty in both inspiration and expiration, but more in expiration. The chest is partially expanded as the result of emphysema and is, of course, hyperresonant. There are usually a few dry râles, but sometimes none are heard. The voice and cry are clear. The temperature is elevated. The blood does not show eosinophilia. These symptoms have almost always been preceded for a few days by those of nasopharyngitis or catarrhal tonsillitis. Death may occur in an attack, but usually the dyspnoea ceases in a few hours or a day or two. In most instances the disease then pursues the course of an ordinary bronchitis, but sometimes this does not happen and the child is well in a short time. These symptoms are probably due more to

spasm of the bronchi than to swelling of the mucous membrane, but it is likely that both play a part. It is difficult to explain why they occur in one instance and not in another. Presumably, there is a constitutional neuropathic basis for them. They are almost certainly not due to anaphylaxis, as evidences of protein sensitization are almost never found in these cases.

Diagnosis.—These cases are sometimes mistaken for diphtheritic or catarrhal laryngitis or acid intoxication or are thought to be due to enlargement of the thymus. They are easily distinguishable from laryngitis, whether diphtheritic or catarrhal, because the voice is clear and the dyspnoea is more marked in expiration instead of being entirely in inspiration. In acid intoxication the respiration is deep, instead of superficial, there is no prolongation of the expiration and the chest is not hyperresonant. If the thymus is large enough to cause much pressure, it is enlarged to percussion and palpable in the suprasternal notch. The larynx does not move in respiration and expiration is not prolonged. A variety of this type, is which the dyspnoea is less marked, the duration longer and the râles more numerous, has been described as *acute sibilant bronchitis.*

Treatment.—The treatment is essentially the same as that of the first stage of acute bronchitis, but, on account of the severity of the symptoms, must necessarily be more active. Cold air definitely increases the severity of the symptoms. The air should be moist and the temperature of the room between 68° F. and 70° F. A hot bath often relieves the symptoms. A flaxseed poultice or mustard paste should be applied to the chest. Ipecac should be given in doses sufficient to cause vomiting, in the same way as in acute catarrhal laryngitis. If the symptoms are not relieved by these measures, from 1 to 5 minims of Benzyl Benzoate or from 5 to 20 minims of H. W. & D's Solution of Benzyl Benzoate, Miscible, according to age, may be given at hour intervals. If the symptoms persist, from 3 to 10 minims, according to age, of the 1–1000 solution of adrenalin chloride may be given hypodermically.

Other babies and young children develop asthmatic symptoms after a few days every time that they have an attack of bronchitis. These occur even when the bronchitis is very mild and are not infrequently more important than those of the bronchitis. They last as long as does the bronchitis, sometimes even for weeks. They are ordinarily not very severe, but may be. The usual story is that there is more difficulty in breathing than would be expected under the circumstances, the respiration wheezy, expiration somewhat prolonged and the cough unproductive. The blood may or may not show eosinophilia, but generally does not. If the symptoms persist for several weeks, slight evidences of emphysema may develop, especially in the upper part of the chest in front. There is presumably a constitutional neuropathic basis for these cases also. Tests show that they are not due to protein sensitization, except sometimes to bacterial proteins. Further evidence that they may be due to sensitization to bacterial proteins is that they are not infrequently benefited when possible niduses of bacterial intoxication, such as diseased tonsils and adenoids, are removed. Another possible explanation of the improvement after the removal of tonsils and adenoids is that the tendency to acute infections of the nasopharynx and, hence, to secondary bronchitis, is diminished with their removal. However this may be, it is certain that children with this type of asthmatic manifestation are more likely to be benefited by the removal of the tonsils and adenoids than any others. The tendency is for children to outgrow this type of asthma.

The first thing in the treatment of this type of case is to prevent, as far as possible, the development of bronchitis. The adenoids should always be removed, and the tonsils also, if there is any reason to suspect that they may be diseased or a source of infection or intoxication. The nose and accessory sinuses should be investigated and treated, if diseased. The children should spend the winters in a warm, dry climate, if possible, just as in chronic bronchitis. All the precautions described to prevent the development of nasopharyngitis and acute bronchitis should be taken.

If asthmatic symptoms develop, little more can be done in the way of treatment than has already been done for the bronchitis. It must be remembered, however, that cold air increases the symptoms. An occasional dose of Benzyl Benzoate may be helpful. It is usually not necessary to give epinephrine subcutaneously. It is inadvisable to use opium in any form.

In the cases in which the attacks of bronchitis are associated with rickets, cod liver oil should be given continuously until the rickets is cured. Cod liver oil is of very little use in other cases or after the cure of the rickets. I am not convinced that the continued use of the iodide of potash between the attacks is of any advantage. It is the prevention of the attacks of bronchitis which counts. Autogenous vaccines are sometimes useful in the prevention of the bronchitis which brings on these attacks. They should be made, if possible, from the sputum. If this cannot be obtained, they may be made from the material obtained by swabbing the nose or nasopharynx. All the forms of bacteria in the sputum should be isolated and separate vaccines prepared from pure cultures of each organism. Each vaccine should contain about one billion organisms to the cubic centimeter. The initial dose is one quarter of a cubic centimeter, or about two hundred and fifty millions, of each vaccine. The doses should be given at intervals of from five to seven days. The dose should not be increased more than $\frac{2}{10}$ of a cubic centimeter at a time and it is not advisable to give more than one and one half billion bacteria of one kind at a dose. It is useless to give more than ten doses.

Hay Fever and Asthma

Hay fever and the asthma which not infrequently accompanies it are manifestations of sensitization to the proteins of the pollens of various plants. Hay fever and hay fever asthma seldom occur in children under five years of age. They are, of course, seasonal in their manifestations, the season varying according to the time of year at which the plants which cause them pollenate.

There are, at least, fifty different pollens which may cause hay fever and asthma, all of which are members of three large families. The following table shows the pollens which are most often the cause of hay fever and the seasons at which they are active.

April-May	June-July	August-September
Elm	Timothy grass	Rag Weed
Willow	Orchard grass	Goldenrod
Oak	Red-top	Sunflower
Maple	Daisy	Daisy
Walnut	Rose	Corn
Birch	June grass	
Dandelion		

The sensitization is usually single, but may be multiple. In this type of asthma also there is probably a constitutional neuropathic basis, as is

shown by the facts that the symptoms sometimes cease in adults when they wear a string of amber beads about the neck or a paroxysm is brought on by the sight of a bunch of artificial roses. There is also no doubt that a diseased condition of the nasal mucous membrane predisposes to the development of hay fever and the asthma which accompanies it. This type of asthma is likely to persist or even to increase with age.

Diagnosis.—The seasonal occurrence and the marked symptoms of nasal irritation make it almost impossible not to recognize this type of asthma. It is also usually possible to make a fairly accurate guess as to the kind of pollen which causes the attacks from the time of year at which they occur. This guess should, however, be verified by skin tests for anaphylaxis to the various pollens.

Treatment.—The surest way to obtain relief from hay fever and hay fever asthma is to go, during the season at which they occur, to some part of the country where the plant whose pollen causes the symptoms does not grow. When it is not feasible or convenient to do this, an attempt at desensitization should be made.

In attempting to desensitize the patient, skin tests should be made with different dilutions of the pollen extract. The dilutions should be $\frac{1}{500}$, $\frac{1}{1000}$, $\frac{1}{5000}$ and $\frac{1}{10,000}$. The initial dose should be of the dilution which did not cause a reaction. It is wise to begin with 0.1 cc. of this solution, increasing the dose to 0.2 cc., 0.3 cc. and 0.4 cc. of this solution, then changing to 0.2 cc. of the next stronger solution, giving 0.3 cc. and 0.4 cc. of this, and so on. The injections should be given subcutaneously every five to seven days. About a dozen injections are usually necessary. This process is not always successful, but is, however, worthy of trial. It is also advisable to have the nose and throat put in the best possible condition during the winter. A certain amount of relief can be obtained by spraying the nose with mild alkaline solutions or with a 1–5000 solution of adrenalin chloride. The various inhalations are likely to increase rather than to diminish the asthmatic symptoms. Benzyl benzoate and adrenalin chloride are, however, sometimes of benefit.

Anaphylactic Asthma

This, the ordinary adult type of asthma, is an anaphylactic response to sensitization to the proteins of foods, animal emanations or bacteria. Hay fever asthma really belongs in this class, but its symptoms are so characteristic and its etiology so definite that is has been discussed as if it were a separate entity. Anaphylactic asthma is not common in infants and young children. The sensitization may be single or multiple; if to foods, it is quite often multiple; if to animal emanations, it is usually single. The sensitization to one thing is often not sufficient to cause attacks; but the sum of several sensitizations may bring on an attack; that is, each sensitization lowers the threshold, but not sufficiently to cause an attack when there is exposure to that protein alone. When there is an opportunity for several proteins to which the patient is sensitive to act at the same time, the threshold is passed and an attack develops. Bacterial sensitization is usually the result of some nidus of infection. This nidus is most often located in the nose and throat or their adnexa. The animal emanations, so-called, which are most often responsible are horse dander, cat hair, rabbit hair and feathers. Rabbit hair is used much more commonly than is ordinarily supposed. Many toys are covered with rabbit skin. Rabbit hair is used in the stuffing of pillows and mattresses and in

the manufacture of various kinds of hangings and clothes. It is also used in making many of the little caps which babies and children wear. The foods which are most often the cause of sensitization are eggs, milk, the grains, and sometimes meats and nuts. The fruits and vegetables are seldom sources of sensitization.

The tendency to recover from this type of asthma in childhood is not as great as in that which complicates bronchitis, but is greater in childhood than in adult life. There is the same tendency to develop chronic bronchitis and emphysema in childhood as in adult life.

Diagnosis.—It is not at all difficult to recognize that a child has asthma of the adult type. This diagnosis is, however, not sufficient. It is necessary to attempt to discover the cause of the asthma. This can sometimes be made out by a careful analysis of the life and the diet in relation to the onset of the attacks, by giving and stopping various foods, by having the child associate or not with various animals, or by changing the room or its furnishings. The best method of determining the cause, however, is by the skin tests for protein sensitization.

These tests are not difficult to make, if the various proteins are available. These can now be obtained in great variety from many dealers. A series of scratches, one-half inch in length and at least an inch apart, are made on the skin, preferably that of the back. They should not be deep enough to draw blood. A small amount of one of the protein powders is placed on each scratch. Incidentally, it is very important to note which protein is put on which scratch and to have several scratches as controls. A drop of a $\frac{n}{10}$ sodium hydrate solution is placed on each scratch and the powder and solution mixed together. Great care must be taken to cleanse the needle with which they are mixed between each drop. The drops should be let alone for twenty minutes, after which they are wiped off. If the patient is sensitized to any one of the proteins which are used, there will be a wheal from $\frac{1}{8}$ to $\frac{1}{4}$ of an inch in diameter along the scratch and an areola of redness, at least an half an inch in diameter, about it. It is important not to be misled by a small wheal along the scratch or a small area of redness about it, because the skin of many children is so sensitive that a small wheal and redness will be caused simply by the scratch. In such cases there is, therefore, a small wheal and redness about all of them. It is always advisable to be suspicious, if there is a reaction about more than two or three of the scratches. No reaction should be accepted as positive, unless it is as large as that described. I have seen a number of children almost starved to death because physicians had misinterpreted the results of the tests. Very often there is no response to the tests during or immediately after an attack of asthma, even though the attack is due to protein sensitization, because, at this time, the skin often does not show any reaction. The tests should be repeated between attacks, therefore, if they are negative at the time of an attack. It is not necessary to use all the food proteins, as, usually, one will serve as an indicator for a class. It is advisable, however, to always use the white and the yolk of egg, the casein and lactalbumen of milk, and all of the ordinary grains. Horse dander, cat hair, rabbit hair and feathers should also always be used.

The results obtained with stock bacterial proteins are not, as a rule, very satisfactory, because, even when there is sensitization to bacterial proteins, there is no response, unless the protein from the organism to which the child is sensitized happens to be in the mixture. Autogenous

vaccines should be made, if possible, from the sputum. If this cannot be obtained, they may be made from the material obtained from swabbing the nose and nasopharynx. All the varieties of bacteria in the sputum should be isolated and pure cultures made. Each organism should be put into a separate vaccine of the strength of about one billion organisms to the cubic centimeter. A quarter of a cubic centimeter, or about two hundred and fifty million bacteria, of each vaccine should then be injected subcutaneously. Only those organisms can be considered as possibly causative to which there is a definite reaction after from eighteen to twenty-four hours. This reaction should be at least one inch in diameter, slightly swollen and tender. Nothing less than this is of importance.

Not infrequently all the tests are negative. In all probability, nevertheless, the attacks of asthma are due to some form of sensitization, just the same.

In this type of asthma the evidence of a constitutional neuropathic basis is not as definite. Nevertheless, it is hard to explain many cases without assuming it. It is also very hard to explain the cases that cease when there is a change of residence and recur when the patient returns to the former residence, even when the food, the exposure to animals and the furnishings of the house are apparently the same. A striking example of this type was a girl who lived not more than a mile from the Children's Hospital. On account of the results of skin tests by various specialists, her diet had been cut down until she was bedridden and emaciated, the room had been stripped of all hanging, the family pets all sent away and a floss pillow used. In spite of this, she had asthma, day and night. Within a few hours after being brought to the Children's Hospital, where she had a feather pillow and was given regular house diet, including many of the articles to which she was supposed to be sensitive, all symptoms ceased. When she went to the Convalescent Home, about fifteen miles out into the country, her asthma returned at once. On a number of occasions the symptoms of asthma, which she had at home and in the country, stopped immediately on admission to the Children's Hospital.

Treatment.—If children are sensitive to animal emanations, they should be kept away from the animals to which they are sensitive. They should use floss or hair pillows, if sensitive to feathers, and, if sensitive to rabbit hair, all articles which may possibly contain it should be discarded. If they are susceptible to certain food proteins, these foods should be cut out of their diet and an attempt made to desensitize them. Desensitization is carried out by first entirely cutting out the protein at fault, then by adding infinitesimal amounts of it to the diet and gradually increasing them. This is done more easily with eggs and milk than anything else, but is always a long, slow, difficult process. If the asthma is due to bacterial proteins, a careful search for some nidus of infection should be made and this should be removed. This nidus is usually in the nose, throat or their adnexa. Some cases are cured in this way. If they are not, autogenous vaccines should be tried.

The initial dose should be one quarter of a centimeter, or two hundred and fifty million bacteria, of the culture to which there was a reaction. Doses should be given every five to seven days, the size of the dose depending on the local reaction which has followed the previous dose. It is not advisable, however, to increase the dose more than $\frac{2}{10}$ of a cubic centimeter at a time. It is rarely advisable to give more than one and one half billion bacteria at a dose. If there is no reaction from the injections, they usually do no good. If there is no relief after six or seven

doses have been given, it is hardly worth while to continue the use of that particular vaccine. If, at any time, the reaction is too vigorous, a smaller dose should be given at the next injection. Stock or heterologous vaccines may be used if they cause a reaction. They are, however, not as satisfactory as the autogenous and many organisms are injected which are doing no good to the patient.

If no source of sensitization can be made out, change of residence should be tried. This need not always be to any great distance.

During an attack the inhalation of the fumes of nitrate of potassium paper or of some of the various asthma powders should be tried. These powders are all more or less alike in their composition, being made up, as a rule, of various combinations of nitrate of potassium, belladonna, hyoscymus and stramonium leaves, and opium. The stramonium leaves are probably more efficacious than the others, While these powders are all more or less alike, one combination is more effectual in one case and another in another, so that it is well to try the different combinations until one is found which agrees. Benzyl benzoate, by mouth, and adrenalin chloride, subcutaneously, may be used, as already described. It is not advisable to use any form of opium in children in the treatment of asthma, except as a last resort, because of the great danger of establishing a habit.

BRONCHOPNEUMONIA

Bronchopneumonia is primarily a disease of infancy and one of the most frequent causes of death at this age. It becomes progressively less frequent and less dangerous with increasing years. It almost always develops secondary to bronchitis, which is itself often a complication or manifestation of some other disease, such as whooping-cough, measles, diphtheria or influenza. In young infants bronchopneumonia may, at any rate clinically, apparently be primary, but even then it is probably secondary to a very diffuse bronchitis of the smaller tubes. All grades of severity, from that recognizable with difficulty to the fulminating, so-called, primary type, fatal in a few hours or days, may occur. It is undoubtedly more common than lobar pneumonia in the first few years of life, if all grades of severity are counted. If the cases complicating whooping-cough, measles and diphtheria and the mild cases, which are hardly recognizable, are excluded, it is in my experience, not much more common or fatal in infancy and early childhood than lobar pneumonia.

Etiology.—Bronchopneumonia is, of course, due primarily to bacteria. It may be caused by a great variety of microörganisms. The most common organisms are the staphyloccocus aureus, streptococci, pneumococci and the bacillus of influenza. The tubercle bacillus also may cause bronchopneumonia, as well as several other organisms. In most instances there is a multiple infection. Practically, bronchopneumonia is due to the extension of a bronchitis, which is, in turn, secondary to a nasopharyngitis or a complication of some other disease. If this fact is borne in mind and nasopharyngitis and bronchitis prevented or carefully treated, bronchopneumonia will be less common and will consequently cause less fatalities.

Pathology.—The inflammatory process originates in the bronchi and the alveoli are involved by extension. This extension is usually direct from the terminal bronchioles to the alveoli in which they end, but may be through the bronchial walls to the alveoli surrounding them. Congestion of the bronchi, bronchioles and alveoli precedes the exudation which

is the essential lesion. Death may occur in the congestive stage. The exudate is made up mainly of mucus, serum and polynuclear leucocytes, with some lymphocytes, desquamated epithelial cells and red corpuscles, the relative proportions varying in different cases. The exudate is materially different from that in lobar pneumonia, in which it is fibrinous. It is evident that in the beginning small areas of solidification are formed. If these areas are numerous and coalesce, large areas of solidification result so that a large portion of a lobe may be involved. There are likely to be small areas of emphysematous lung tissue mixed in with the solidified areas, especially in infancy. If small bronchi are plugged with secretion or by the swelling of their mucous membrane, areas of atelectasis also develop. The pathologic process may pass through the stages of congestion, solidification and resolution or may terminate at any stage. Various stages may be found side by side and intermingled, according to the time at which the process in the individual area started. It is evident that all sorts of combinations and all degrees of involvement are possible. Bronchopneumonia being secondary to bronchitis, which is itself almost always bilateral, it is evident that the lesions in bronchopneumonia must usually be bilateral, and that they are likely to be diffuse. The lesions are most common in the lower lobes behind or in both upper and lower lobes behind.

The duration of the pathologic process is indefinite because new areas of solidification may be formed as long as the bronchitis keeps up, which may be for many weeks. Resolution is, moreover, often slow, even though no new areas develop, sometimes requiring several weeks. Recrudescences of the infection are also not uncommon, especially when the infection is due to the influenza bacillus. Resolution sometimes does not take place and chronic interstitial pneumonia develops. A secondary tubercular infection may also take place. Small abscesses, unrecognizable clinically, may develop, their contents be coughed up and a scar result. Large abscesses are very uncommon in my experience, as is also gangrene of the lung, unless the bronchopneumonic process is a localized one as the result of the inhalation of some foreign body. Inflammation of the pleura is often present over large, superficial areas of consolidation and may go on to the formation of a serous, serofibrinous or purulent effusion. Clinically, inflammation of the pleura is seldom recognizable and effusion is most unusual. The tracheobronchial lymph nodes are enlarged and congested in all but the mildest cases.

Symptomatology.—It is almost impossible to describe the symptomatology of bronchopneumonia because the disease varies so much in severity, in the amount of lung tissue involved, the acuteness of the onset and the duration. The symptoms and physical signs may be so slight that the presence of the disease can only be suspected. They may be characteristic, but very irregular, or they may be so severe and acute that death occurs before any characteristic signs develop. Even in the common form, no two cases are exactly alike. Several types of the disease are, however, fairly distinct.

The Acute Fulminating Type.—When there is an acute, diffuse involvement of the bronchioles, as sometimes occurs in the so-called "*capillary bronchitis*," there may be a rapid extension of the pathological process not only to the terminal alveoli but also to those surrounding the bronchioles, so that the greater part of both lungs may be involved at once. In such instances the causative organism is usually some form of pneumococcus. This type is very uncommon and almost never occurs after infancy. The

onset is always acute. The temperature is high and the pulse very rapid. The respiratory rate is very high, 60, 80, or even higher, because of the effort to make up for the interference with the entrance of air into the alveoli by increasing the number of respirations. There is always cyanosis, which is sometimes very marked, because of the interference with oxygenation. There is for the same reason always more or less dyspnoea. There is also retraction of the lower chest and epigastrium because the lungs cannot be properly expanded. Cough is frequent, because of the desire to clear the bronchioles and alveoli, and short, because of the deficient expansion of the lungs. There are usually no abnormal physical signs, except some diminution in the respiratory sound. If the baby lives long enough, there may be slight dullness, bronchovesicular respiration and an occasional fine, moist râle. If it lives three or four days, moist râles become numerous and the physical signs of the common type of bronchopneumonia develop.

Diagnosis.—It is almost impossible to distinguish this type of bronchopneumonia from capillary bronchitis, in which the pathologic process has not extended to the alveoli, as the symptoms and physical signs are essentially the same in both. Fortunately, it is not important to do so as the prognosis and treatment are not materially different in the two conditions. It may be mistaken for acute lobar pneumonia, but ought not to be. In lobar pneumonia, although there may be no physical signs, there is no interference with the entrance of air, and, hence, no retraction. If there is cyanosis, it is toxic, not the result of deficient oxygenation. Dyspnoea, if present, is due to pain, not deficient oxygenation. Cough is less marked, not as frequent, and deeper.

Prognosis and Treatment.—This type of bronchopneumonia is extremely fatal. Most babies with it die in from twenty-four to forty-eight hours, in spite of anything that can be done. If they live three or four days, they are quite likely to recover. The treatment is essentially the same as that described for capillary bronchitis.

The Common Type.—In this type the symptoms are superadded to those of the causative bronchitis, which may or may not be itself a complication of some other disease. There is nothing about these symptoms which serves to distinguish them from those of the bronchitis. The symptoms, which were at first due entirely to the bronchitis, simply become more marked. The temperature is higher, the cough more troublesome, the pulse and respiration more rapid and the patient sicker. The increase in the severity of the symptoms warrants the suspicion that bronchopneumonia may be developing. If the areas of bronchopneumonia are small, scattered or deeply seated, nothing may be found on physical examination to justify the suspicion.

If the bronchopneumonic process is more extensive, dyspnoea and cyanosis may appear. These may become very marked if considerable areas are involved. Retraction of the intercostal spaces, lower chest and epigastrium are often present in severe cases as the result of the interference with the expansion of the lungs. The temperature runs higher and the pulse and respiration are more rapid, the increase in the rate of the respiration being out of proportion to that in the rate of the pulse. The râles due to the bronchitis persist. In addition, the signs of solidification of the lung develop. If the areas of solidification are small or deep-seated, there is no dullness; if they are large and near the surface, there is dullness, sometimes even flatness. If they are at the surface or near it, the respiration is bronchial or bronchovesicular and the râles are likely

to be high-pitched. There is no change in the voice sounds, unless the areas are quite large. There are usually several areas of solidification and they are likely to be on both sides. Furthermore, these areas are

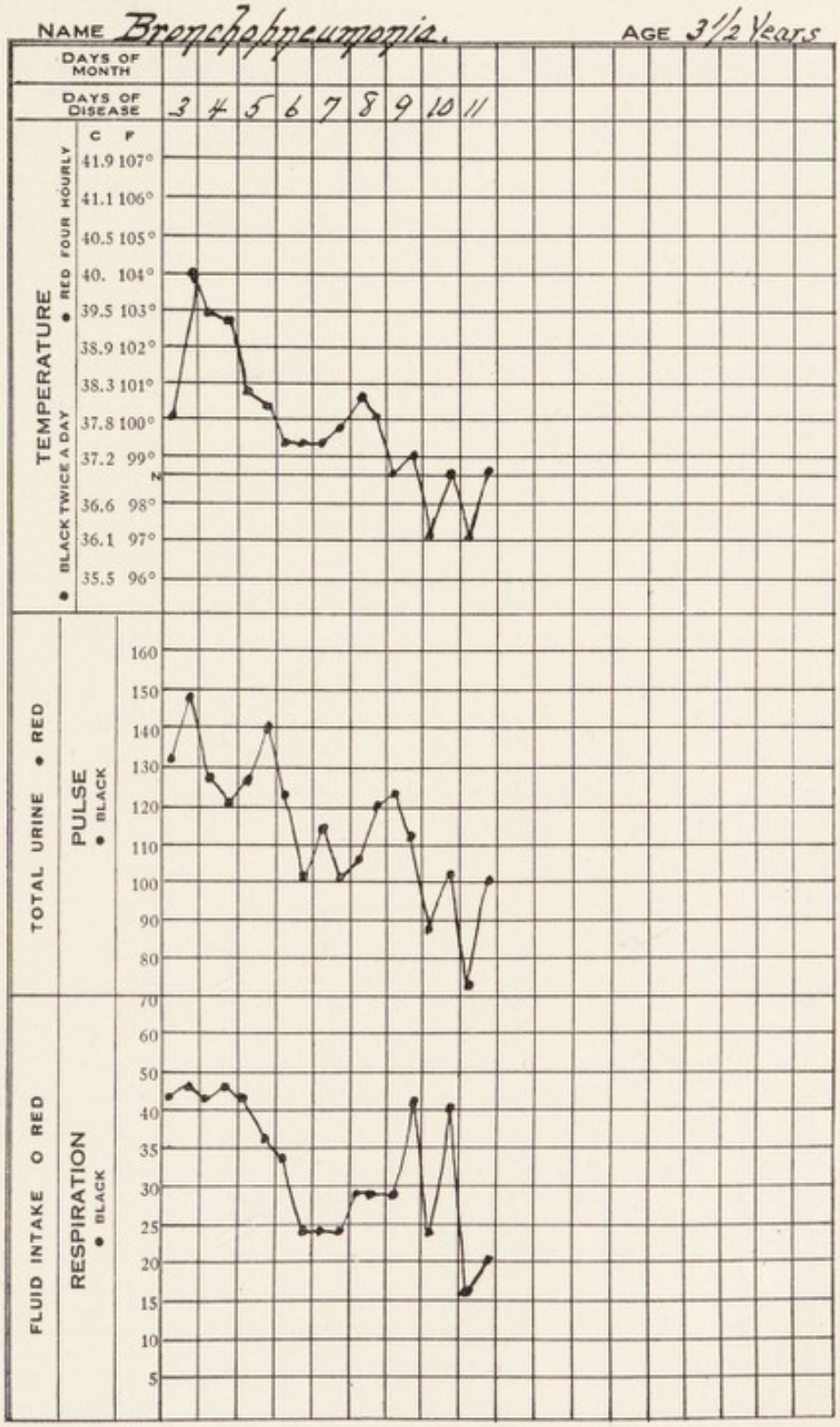

Fig. 109.—Bronchopneumonia.

likely to resolve and new ones develop. Consequently, the signs vary from day to day. The variation in the physical signs is increased by the development and disappearance of areas of emphysema and atelectasis about the areas of solidification. When, as sometimes happens, the

areas of solidification are large and confined to one lobe of one lung, the signs are essentially the same as those of lobar pneumonia, except that there are usually more râles. It is only in these cases that there is much change in the tactile fremitus.

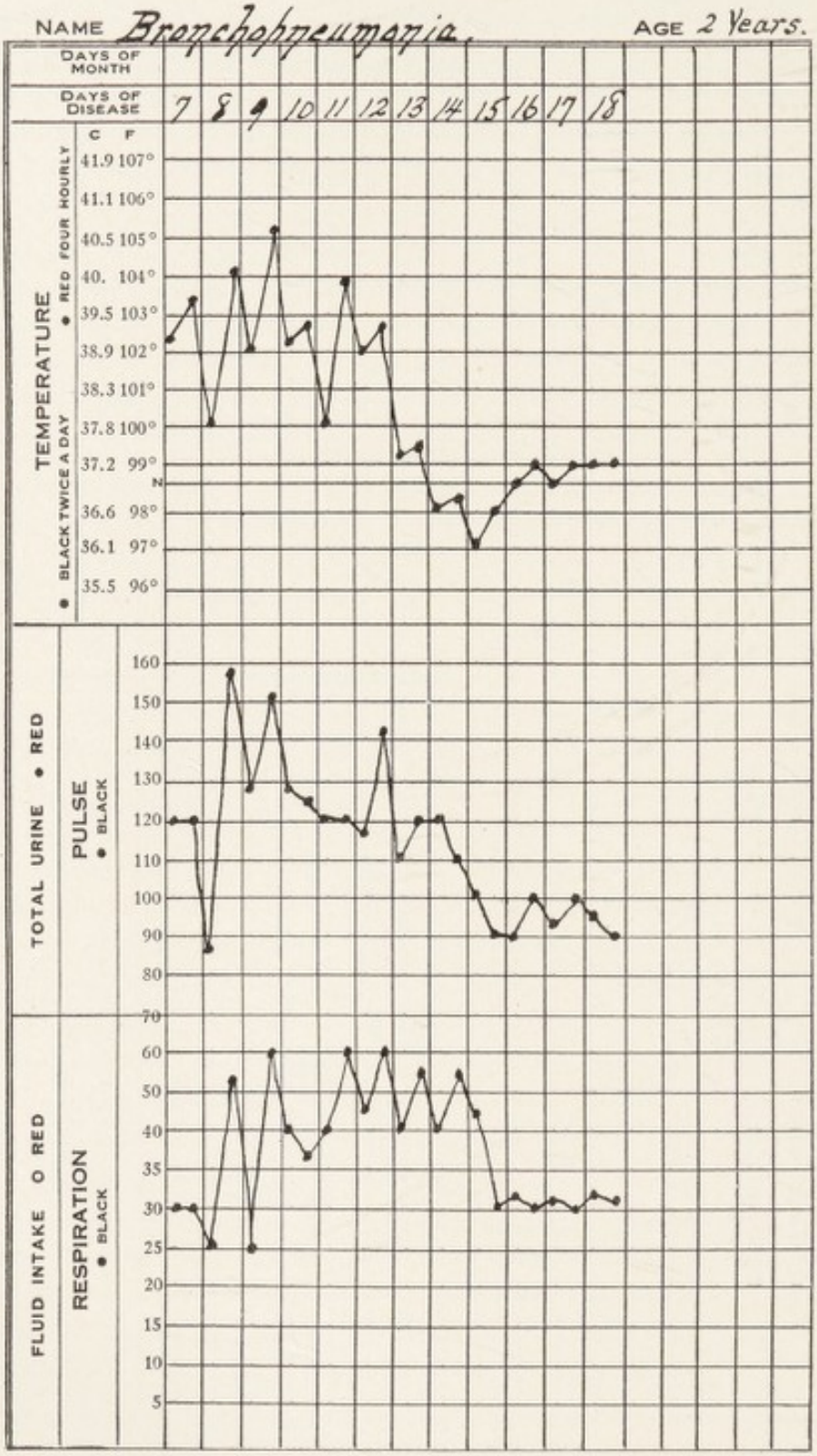

Fig. 110.—Bronchopneumonia.

There is nothing constant about the course and duration of this type of bronchopneumonia. It may be very mild and last but a few days. It may be very severe, and fatal in a few days or a week. It may be

very severe, with or without remissions, and end in recovery. It may be of moderate severity and last for weeks or be mild and suddenly become severe. Every possible variation may occur. The physical signs are quite as variable, as they depend, of course, on how much of the lungs is

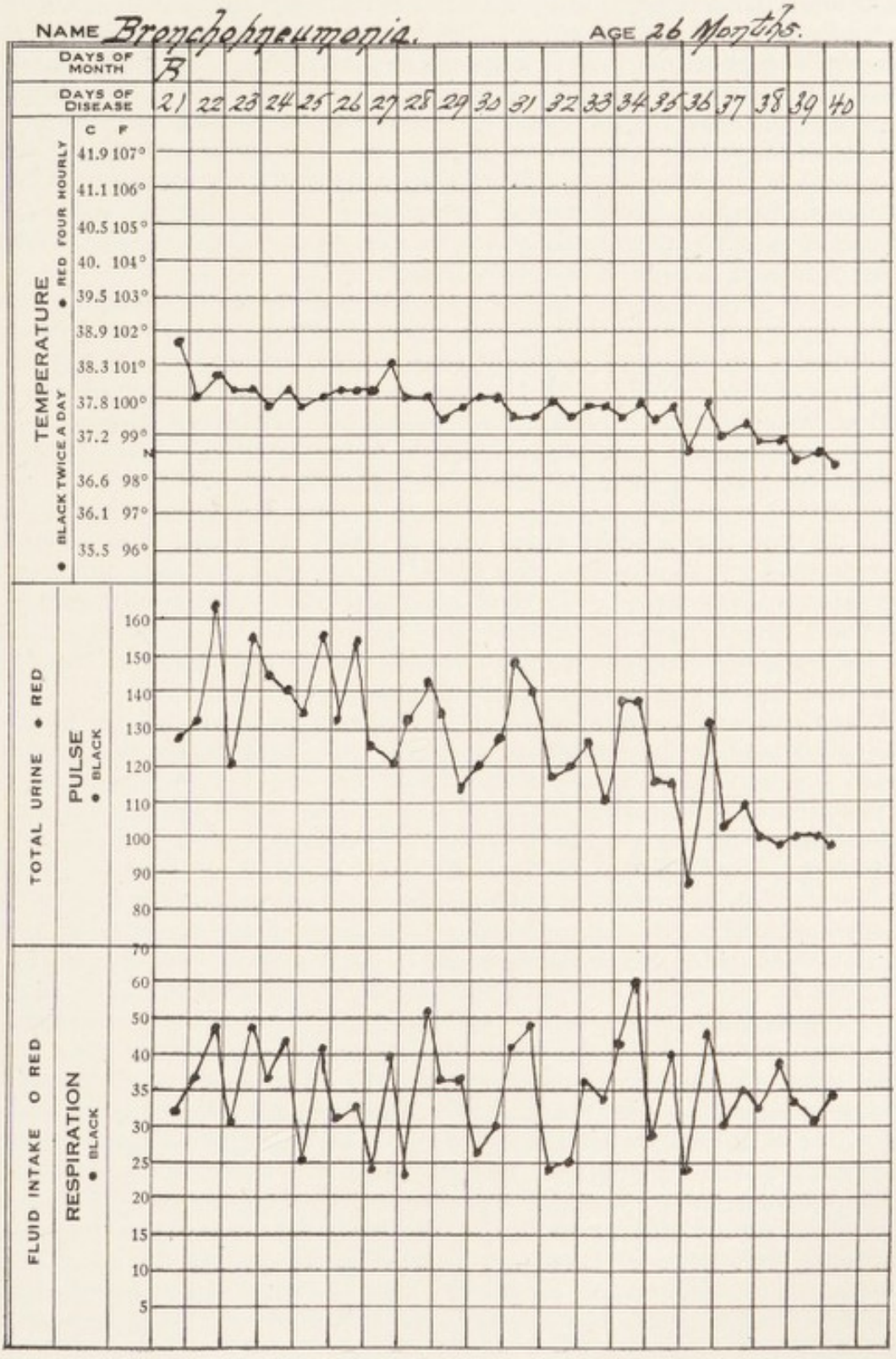

FIG. 111.—Bronchopneumonia.

involved at the given time. The *temperature* varies with the severity of the illness and the amount of lung tissue involved. In general, it is under 103° F., but may reach 105° F. or 106° F. It is almost always

irregular, sometimes markedly so. It is often higher in the morning than in the afternoon, but this fact is of no significance. If the patient is evidently seriously ill, a temperature curve which is constantly under 102° F. is of bad prognostic import, because it indicates severe toxemia. The temperature always falls by lysis, which is usually quite gradual. The *pulse* is rapid and in severe cases more so than would be expected from the temperature. The increase in the rate of the respiration in proportion to that of the pulse is not as constant as in lobar pneumonia. The alae nasi move in severe cases. Expiration is often grunting. In mild cases there is no *dyspnoea* or *cyanosis*. In severe cases, in which large areas of the lung are involved, they are often very marked. Cough is almost always a troublesome symptom. It is usually loose. The sputum is usually swallowed by babies and young children, but when it can be obtained, it is usually mucopurulent.

There is almost always loss of appetite and more or less disturbance of the digestion. Vomiting is not very common, except with cough. Diarrhoea is much more common than constipation. The stools show evidences of interference with the digestion of all the food elements and often of fermentation. Distention of the abdomen is not as common as in lobar penumonia and seldom causes much trouble. The urine frequently contains a little albumen and shows the evidences of acute degenerative nephritis. Otitis media is a very common complication.

In all but the mildest cases and in those in which the patient is overwhelmed by the disease, the blood shows a polynuclear *leucocytosis* of greater or less degree. It is of no importance in either diagnosis or prognosis, except that its absence in a severe case is a bad sign. In all but the mildest cases secondary anemia develops, which in the long-drawn-out cases may become quite severe.

Diagnosis.—It is impossible to determine, if there are no physical signs of consolidation, whether a child with bronchitis has bronchopneumonia as well or not. It should be suspected, however, when the temperature is higher, the pulse and respiration more rapid and the child sicker than would seem likely from the evidences of bronchitis which are found. Nothing can be learned from a count of the white cells, because they are increased in both diseases and the amount of the increase is very variable. An examination of the chest with the Roentgen ray will, however, often show small areas of consolidation which cannot be made out in any other way and thus settle the diagnosis. It is usually easy to distinguish between the common type of bronchopneumonia and lobar pneumonia. Bronchopneumonia develops secondarily to a bronchitis; lobar pneumonia is usually primary. The onset is more sudden in lobar pneumonia, the temperature more regular and usually higher. The areas of consolidation are multiple in bronchopneumonia, single in lobar pneumonia. Dyspnoea and cyanosis are more marked and the cough usually more troublesome in bronchopneumonia. There are many other minor differences in the frequency and severity of individual symptoms, but none of them are characteristic enough to be of much importance in differential diagnosis. Both diseases are accompanied by leucocytosis. The white count is, therefore, of no assistance in diagnosis.

Treatment.—There is no specific treatment for bronchopneumonia. The treatment must necessarily consist, therefore, of general care to strengthen the resistance and to aid nature in overcoming the infection and of measures for the relief of symptoms. Every case, however mild,

must be regarded seriously. Babies and children with bronchopneumonia should be put to bed and kept there until they are well. They should not be taken out of bed and held in the arms or lap, unless it is absolutely necessary to do this to conserve strength being wasted by fretting and tossing about. The position must be changed frequently to prevent congestion and the development of atelectasis and to favor the clearing of the lungs. An abundance of fresh air is very important. Cold air is contraindicated if there is bronchitis of the larger tubes or laryngitis. It is not good for otitis media. When there is otitis media, the relative importance of the bronchopneumonia and the otitis media must be weighed in deciding whether to use cold air or not. In my experience babies and children with bronchopneumonia do not, as a rule, do as well when the air is cold as when the temperature of the room is between 64° F. and 66° F. Sometimes, however, if there is marked cyanosis and dyspnoea, they seem to do better if they are given the cold air treatment, as in lobar pneumonia.

Proper regulation of the feeding is most important. Babies and young children are very likely to refuse food entirely. If they do, they must be made to take it in some way. Babies may be fed with a medicine-dropper or a Breck feeder, if they are unable or refuse to nurse. If they are exhausted by attempts to make them take their food in this way, it is easier for them to be fed with a tube. Babies should be given breast milk, if possible. If this cannot be obtained, they should be given a properly modified cow's milk. Weak mixtures are advisable, because of the enfeeblement of the digestion. After infancy, milk should form the basis of the diet. This may be given in the form of junket or blancmange. Starches may also be given in the form of gruel or cereals. There is no objection, of course, to bread or crackers in various forms, if the patients are able to chew. Broths should not be given, as they contain no nourishment and, when but little food is taken, this should be in a concentrated form. There is no objection to orange juice. It is not advisable, however, to give other fruit juices. The diet should, of course, be varied according to the age of the child and the severity of the illness in the individual case. It must never be forgotten, however, that the digestive capacity is impaired, that it is very easy to disturb the digestion and that it is most important not to disturb it, because the prognosis depends to a considerable extent on whether the child can take a proper amount of nourishment or not.

There are no local applications which can influence in any way the pathological process in the lungs in bronchopneumonia. Heat or cold applied externally may sometimes relieve the pain of a complicating pleurisy, just as in lobar pneumonia. Poultices of whatever sort not only do no good, but do active harm in that they impede the respiration by their weight and tend to keep up the temperature. Applications of scented mud are less harmful because they are not usually made as thick and heavy. Cotton jackets, even when covered with oiled silk, are usually not heavy enough to do much harm by their weight, but do interfere materially with the loss of heat.

The temperature is seldom high enough continuously in bronchopneumonia to require active measures for its reduction. If it is, it should be treated in the same way as in lobar pneumonia. It is almost never safe or advisable to give antipyretic drugs internally to babies or young children ill with bronchopneumonia. They may sometimes be given to older children.

The bronchial element in bronchopneumonia should be treated in the same way as bronchitis without bronchopneumonia. There are no drugs which can influence in any way the pathologic process in the alveoli or in the interstitial tissues about the bronchi. Great care must be exercised in giving drugs for the relief of the cough, which is often so troublesome, not to interfere with the proper clearing of the bronchial tubes. It is very easy to do irreparable harm and to increase the bronchopneumonic process by stopping the cough and thus preventing the clearance of the bronchial tubes. No attempt should be made to diminish the cough, unless it is a useless cough, that is, one which is unproductive and which is tiring the patient out. Proper regulation of the temperature of the room and a change in the amount of moisture in the air is often sufficient to relieve the cough. A change of position will sometimes help. Stimulation, which enables the patient to cough more effectually, is often useful. If it is necessary to use drugs, small doses of one of the bromides, perferably that of sodium, are sometimes sufficient and are less dangerous than opium. If they do not work, some preparation of opium must be used. The best and only safe preparation to use in babies and young children is paregoric. The dose of paregoric is from 3 to 10 drops for a baby and from 10 drops to ½ a teaspoonful for a child, the dose varying, of course, according to the age. A teaspoonful of paregoric contains about ¼ of a grain of powdered opium. For older children codeine or the phosphate or sulphate of codeine, in doses of from 1/16 of a grain to ½ a grain, may be used, according to the age and individual susceptibility of the child. The hydrochloride of heroine may also be used in doses about one-half as large as those of codeine and its salts. Whenever heroine or codeine are used, a small dose should be given first because so many children are susceptible to opium. If that is not sufficient, it may be increased later. It should never be forgotten that any form of opium is dangerous in bronchopneumonia, and that it should never be used, therefore, unless it is certain that it is necessary and that it will not do more harm than good.

Stimulants should be used as in other diseases, if necessary. I do not believe that alcohol is a stimulant and, therefore, do not use it. Small doses may, perhaps, serve as a food. It is rarely advisable to give alcohol for this purpose, however, as food may be given better in other ways. Furthermore, all the preparations of alcohol are likely to disturb the digestion and, if given in large doses, to "dope" the patient. The most satisfactory stimulant in bronchopneumonia in infancy and childhood is strychnine. The beginning dose for an infant is 1–480 of a grain, given at four-hour intervals. The intervals may be diminished to two hours and the dose increased. The beginning dose for a child of four years is 1–120 of a grain. This dose may also be increased as necessary. In general, infants and children need relatively large doses of strychnia. If stimulants are needed, they should be pushed until benefit is evident or until toxic symptoms develop. If toxic symptoms develop, the dose should, of course, be diminished to the point where they disappear. Digitalis should be given if there are evidences of failure of the right heart. It should not be given as a routine measure. If a quick stimulant is needed, citrate of caffeine, in doses of from ¼ of a grain for a baby and of one grain for a child, may be given by the mouth or slightly smaller doses of caffeine-sodium benzoate or salicylate may be given subcutaneously. A grain of camphor in oil may also be given subcutaneously. It must be remembered that these drugs have only a

fleeting action and should be given only in emergencies. If a continued action is desired, it is necessary to give them at least as often as every hour. Given, as they usually are, at intervals of three or four hours, they fit very well the late Dr. H. C. Wood's description of "kangaroo therapeutics." Atropine, in doses of 1–500 of a grain for a baby, or the 1–1000 solution of adrenalin chloride, in doses of from 2 to 5 minims, may be given subcutaneously, if desired, when there is much trouble with the respiration.

It must never be forgotten, however, that, if babies and young children are protected as they should be from exposure to measles and whooping-cough, if common colds and bronchitis are properly treated and if bronchopneumonia is looked after from the beginning, such measures as have been described above are almost never necessary.

No chapter on the treatment of bronchopneumonia is complete without the famous dictum of Dr. Northrup (The Medical News, April 30, 1904) on "How to Kill a Baby with Pneumonia:" "Crib in far corner of room with canopy over it. Steam kettle; gas stove (leaky tubing). Room at 80° F. Many gas jets burning. Friends in the room, also the pug dog. Chest tightly enveloped in waistcoat poultice. If child's temperature is 105° F. make a poultice thick, hot and tight. Blanket the windows, shut the doors. If these do not do it, give coal-tar antipyretics and wait."

Influenzal Bronchopneumonia

The common type of bronchopneumonia is the usual form in influenza. In certain instances, however, especially in times of epidemics, the influenza bacillus produces a special type of bronchopneumonia characteristic enough to deserve separate description. In this type the area involved at one time is usually not very large, but before one area has had time to clear up entirely another is involved and so on for six or eight weeks. The pathological process in a single area usually develops and runs its course quickly. The exudate in the alveoli is purulent and profuse, but the walls are, as a rule, not as much involved as in the common type. Consequently, the pathologic process generally clears up more quickly than in the ordinary type, although, in some instances, small abscesses may be formed which are healed by the growth of fibrous tissue.

Symptomatology.—On account of the consecutive involvement of many areas of lung tissue, the temperature is irregular, rising with the involvement of each new area, and of long duration. It is usually not very high and finally comes to normal after a long and very gradual lysis. The sputum is more abundant than in the common type and purulent. Microscopically, it is made up largely of polynuclear neutrophiles and contains large numbers of influenza bacilli. The only peculiarity in the physical signs is the rapidity with which they vary. Signs of solidification develop in a given area, last but a short time in this area and appear in another area, perhaps before they have entirely disappeared from the first.

Prognosis.—The prognosis in this type of bronchopneumonia is, as a rule, good. It is better than that in bronchopneumonia of the common type of apparently the same severity and far better than in the common type when of the same duration.

Treatment.—The treatment is the same as that of influenza and of the ordinary type of bronchopneumonia. It must not be forgotten, however, that the bronchopneumonia of this type is very contagious and that, therefore, all precautions should be taken to prevent contagion.

Tuberculous Bronchopneumonia

Tuberculous bronchopneumonia, while apparently a primary disease, is, of course, always secondary to some tuberculous lesion elsewhere, ordinarily in the tracheobronchial lymph nodes. Three main types are recognizable, the acute, subacute and chronic.

Acute Tuberculous Bronchopneumonia.—The onset of this type is essentially the same as that of ordinary bronchopneumonia. There are the same physical signs of bronchitis with the formation of more or less extensive areas of solidification. The areas of solidification, however, instead of clearing up, persist, while new ones develop. The temperature is more irregular and remissions nearly to the normal one or more times daily are not uncommon. The respiration and pulse are, as a rule, relatively more rapid than in ordinary bronchopneumonia. The babies, in whom this form is almost always seen, are much brighter and take much more notice than do babies who are as severely ill with the ordinary form. This type may become subacute or chronic, but, as a rule, death occurs in the acute stage.

Diagnosis.—The diagnosis from the ordinary type of bronchopneumonia is almost impossible, because, even if the sputum can be obtained, tubercle bacilli are seldom present in it. The tuberculin test, moreover, is likely to be negative because the baby is overwhelmed with the disease. The symptoms which suggest that the process is tuberculous are the marked irregularity of the temperature, the relatively rapid pulse and respiration and the peculiar brightness of the baby when it is apparently in other ways so seriously ill.

Subacute Tuberculous Bronchopneumonia.—The onset in this type is more gradual than in ordinary bronchopneumonia, although it may sometimes follow acute tuberculous bronchopneumonia. It is also sometimes engrafted on the common type of bronchopneumonia. It is the type which develops after measles and whooping-cough. The duration of this type is usually from six weeks to two months, and the end, death. In rare instances, however, recovery ensues, sometimes with the evidences of the formation of scar tissue in the lungs, sometimes with no abnormal signs remaining.

Symptomatology and Physical Signs.—There is nothing very characteristic about the symptoms, except their persistence, or about the physical signs, except that they do not change from place to place but continue localized and gradually increase. The temperature is very irregular, usually not ranging much over 102° F., often with intermissions to normal, which may last for several days at a time. The rate of the respiration is considerably increased and runs constantly higher than would be expected from the child's conditions and physical signs. The pulse is not relatively as rapid as in the acute type. Cough is ordinarily very troublesome. Just as in the acute type the babies remain almost to the end brighter and more interested in their surroundings than would seem possible from the other evidences of disease which they show. The physical signs are those of solidification in steadily increasing areas. In advanced cases flatness may become quite marked and there may be a considerable increase in the sense of resistance. Small cavities are frequently found at autopsy, but they are usually not large enough to give any physical signs during life.

Diagnosis.—The diagnosis between subacute tuberculous bronchopneumonia and the ordinary type of bronchopneumonia is impossible in the beginning. Subacute tuberculous bronchopneumonia should be

suspected, however, if the physical signs persist in the same area without the development of new areas or the disappearance of the old. Other signs which should suggest it are the long continuance of the illness, the relatively low temperature, the marked intermissions in it, the relatively rapid respiration and especially the clear mentality and lack of evidences of general toxemia, even when there are many other very marked evidences of serious illness. Tubercle bacilli may sometimes be found in the sputum in this type. The tuberculin test may or may not be positive. If positive, it is strong evidence that the process in the lung is tuberculous. If it is negative, it does not by any means exclude a tuberculous process. Examination with the Roentgen ray can show only shadows, which are the same whether the process is tuberculous or not. Some physicians lay considerable stress on the presence of evidences of enlargement of the tracheobronchial lymph nodes, feeling that they count strongly in favor of a tuberculous process. It seems to me that this is not so because the lymph nodes are almost certain to be enlarged when there is any chronic inflammatory process in the lungs. Evidences of calcification would, however, be rather strong evidence in favor of a tuberculous process.

Treatment.—The treatment of both acute and subacute tuberculous bronchopneumonia is essentially the same as that of ordinary bronchopneumonia, except that fresh air is of more importance; that is, as soon as the diagnosis of tuberculous bronchopneumonia seems probable, the treatment of active tuberculosis should be begun.

Chronic Tuberculous Bronchopneumonia.—This type, while pathologically bronchopneumonia, is practically simply chronic pulmonary tuberculosis and is, therefore, taken up under tuberculosis.

Frequency and Prognosis of Bronchopneumonia.—The frequency and fatality of bronchopneumonia are a reflection on the general intelligence of the community and the medical skill of its physicians. Bronchopneumonia is largely a preventable disease, and, even after it has developed, if it is treated early and properly, it should seldom prove fatal. The development of bronchopneumonia can be, to a great degree, prevented by proper measures to prevent the spread of common colds and the proper treatment of colds and bronchitis. The very serious and fatal types of bronchopneumonia which occur in babies in connection with measles and whooping-cough can be prevented by properly protecting babies against infection with these diseases. Tuberculous bronchopneumonia can be avoided by the proper isolation and treatment of adults with open tuberculosis.

LOBAR PNEUMONIA

Acute lobar pneumonia occurs at all ages. If all grades and types of bronchopneumonia are included, lobar pneumonia is less common in infancy than bronchopneumonia. If the cases of bronchopneumonia in which the diagnosis from bronchitis is in doubt and those secondary to measles and whooping-cough are excluded, lobar pneumonia is probably fully as common in infancy as bronchopneumonia. It becomes progressively more common with increasing age until about ten or twelve years, when its frequency diminishes. It occurs most often in the early spring, next in the winter, seldom in the summer. Boys are more often affected than girls. The infection is primarily in the lungs. If there is a bacteriemia or other organs are involved, the infection is secondary to that in the lungs.

Etiology.—Lobar pneumonia, that is, pneumonia in which there is a fibrinous consolidation of the lung, is caused in at least 90% of the cases by some form of the pneumococcus. The pneumococci are, as is well known, now divided into four main classes, which are called types I, II and III and group IV. In adults it has been found that approximately ⅔ of the cases of pneumonia are caused by organisms belonging to types I and II and about ⅕ to organisms belonging to group IV. It has been generally believed that in children a very large proportion of the cases are due to infection with organisms belonging to group IV. This has been found to be especially true in infants. The frequency of infection with group IV in infancy has been thought to be due to the slight resistance of babies to infection. It has also been thought that the low mortality of pneumonia in early life was due in some measure to the frequency of infection with organisms belonging to group IV, because, as is well known, their virulence is less than that of the other types. Lyon, however, in a study of 165 cases of pneumonia and empyema in infants and children at the Boston City Hospital, (American Journal Diseases of Children, 1922, **23,** 72), found that infection with organisms of group IV was relatively not as common as others had suspected, while that with type I was more common.

While lobar pneumonia is unquestionably due primarily to the pneumococcus, chilling of the surface of the body and fatigue, especially if they are combined, do certainly predispose to infection. Generally poor hygienic surroundings also predispose to the disease, perhaps, not because they diminish resistance but because they increase the exposure. Other diseases do not predispose to pneumonia and it occurs just as frequently among the strong and robust as among the feeble and delicate.

So little is accurately known about the matter that it is hardly worth while to discuss why infection takes place, whether to an increase in the virulence of the organisms, to diminished resistance of the child or to local changes in the lungs favoring infection. Whatever may be the cause, practical experience shows that lobar pneumonia is very little, if at all, contagious and that there is little or no danger of contracting the disease by direct contact.

Pathology.—The essential lesion is a fibrinous exudate into the alveoli and bronchioles. The alveoli are filled before the bronchioles. All the alveoli in the affected area are involved. Extension is by continuity. The exudate is made up of serum and polynuclear leucocytes with some endothelial leucocytes and red corpuscles. In most instances fibrin is found in large quantities in the exudate. The area involved is at first much engorged and congested with blood and, consequently, appears much reddened. As the exudate increases, the alveoli become distended and the blood in the capillaries is squeezed out, so that the color changes from dark red to gray. These stages are usually spoken of as those of congestion, red hepatization and gray hepatization. During the stage of resolution there is a marked increase in the number of polynuclear leucocytes, which break down the fibrin. The exudate is carried away by the leucocytes through the lymphatics or is coughed up.

The inflammatory process apparently always begins at the surface of the lung and extends laterally and inward. It may involve a whole lobe or stop at any point short of this. It does not extend directly from one lobe to another, but a new process may start in another lobe. The left lower lobe is the one most often involved. In order of frequency are then the right upper, right lower, left upper and right middle lobes.

The pleura is usually involved. Fibrinous pleurisy is present in the majority of cases but does not affect the course of the disease. There is, not infrequently, a small serous or bloody serous exudate, which is, however, seldom large enough to give definite physical signs. There may be a large serous exudate. If so, it almost always becomes purulent. If the exudate is large, it is usually purulent from the first.

There is somteimes inflammation of the pericardium from contiguity. There is also occasionally inflammation of the meninges, peritoneum and joints. Degenerative changes occur in the parenchymatous organs from toxemia and are most marked in the heart and kidneys.

Pneumococci can be found in the blood in from 10 to 15% of the cases. When found, they are usually of type 1. The prognosis is apparently but little worse in these cases.

Symptomatology.—*Onset.*—The onset of lobar pneumonia is always acute. In rare instances it is a complication of some other disease. The usual story is that a baby or child that is perfectly well suddenly becomes sick and very feverish. The mothers often describe the condition as stupid, drowsy or "dopey." The younger the child, the more often is the onset accompanied by vomiting. Older children often complain of being chilly, but a definite chill is most unusual in my experience. A convulsion at the onset of pneumonia, even in infancy, is also quite uncommon. Pain and cough are almost never present at the onset.

Duration.—The average duration of lobar pneumonia in infancy and childhood, as in adult life, is about seven days. There are, however, many more cases of short duration in early than in adult life, the average being kept up by a comparatively few cases of long duration. The younger the child, the more likely is the disease to be of short duration. It is also true that cases of long duration are more common among babies than children.

Abortive cases, meaning by this term cases in which the duration is from twenty-four to seventy-two hours, while not common, are not very infrequent. In these cases the onset is sudden and characteristic, the temperature is high and the pulse and respiration ratio disturbed. There may be no physical signs in the chest or they may be limited to a diminution of the respiratory excursion or sound on one side with, perhaps, a few fine localized râles. The only difference between these cases and those of frank pneumonia is that, instead of pursuing its usual course, the disease comes to an abrupt close with or soon after the initial stage.

In other instances one spot after another may be involved and the duration many weeks, each area requiring the usual time to run its course. The temperature in such cases remains constantly high unless a crisis or a fall by lysis occurs at the end of the process in one area before another is involved. In other cases the affected area does not resolve for a long time. In such instances the temperature may drop and all symptoms disappear. In others the temperature remains somewhat elevated and the picture strongly resembles that of pulmonary tuberculosis, for which it is often mistaken.

Temperature.—The temperature is of necessity high in lobar pneumonia in early life, temperatures of 104° F. or 105° F. being the rule rather than the exception. A temperature of 106° F. in babies and young children is not at all uncommon, and is of no especial significance unless it is accompanied by marked stupor or evidences of nervous irritation. When the temperature does not go above 102° F. the condition is usually a serious one, because the failure of the temperature to go higher shows a

lack of normal resistance in the patient. While the temperature runs higher, as a rule, in early than in adult life, remissions in the fever, often of several degrees, are much more common. The younger the child, the

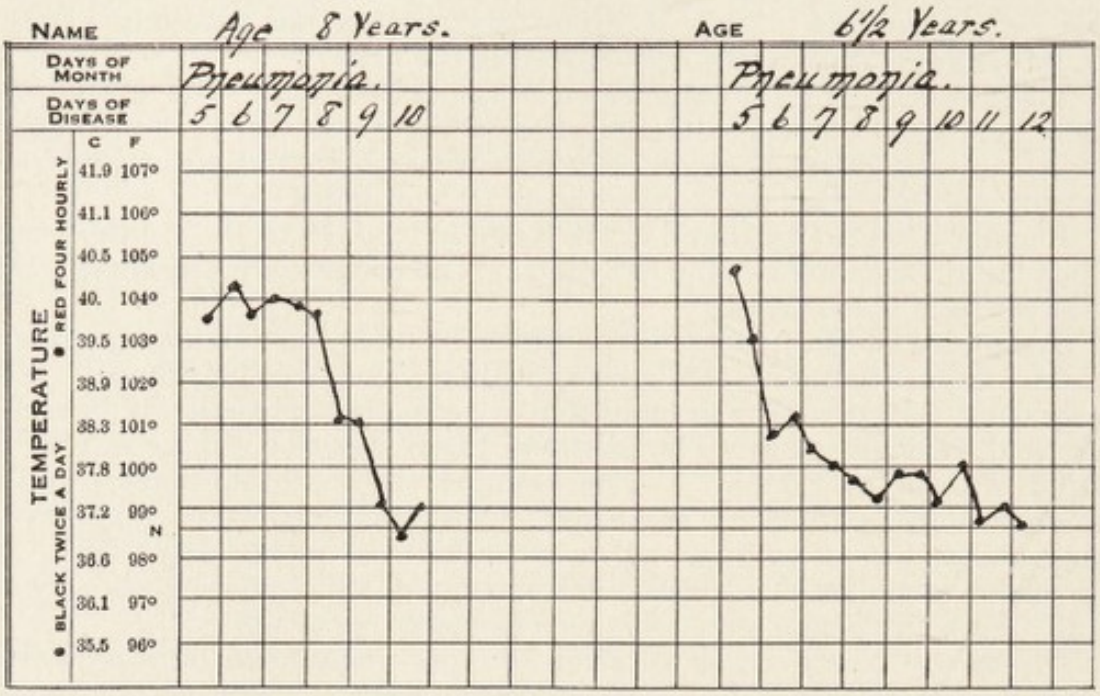

FIG. 112.—Pneumonia. Fall in temperature by rapid lysis.

more likely are such remissions to occur. In babies and young children the temperature often falls to normal by a rapid lysis, occupying two or three days, instead of by crisis. Pseudocrises are also not uncommon and

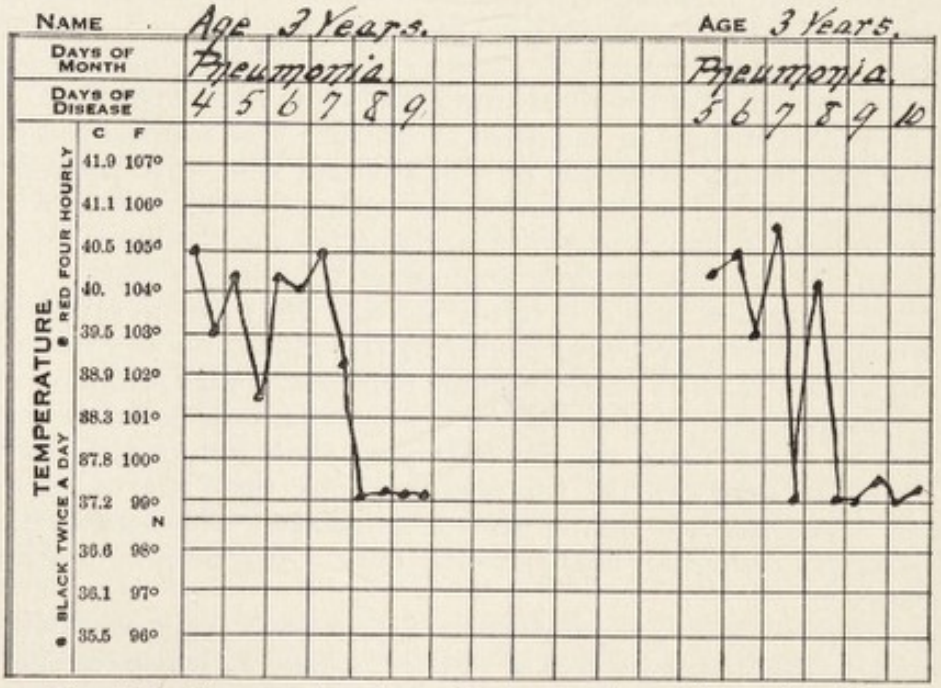

FIG. 113.—Pneumonia. Irregular temperature and pseudocrisis.

in some instances there may be two on successive days. When there is a pseudocrisis the pulse and respiration usually do not fall with the temperature, as they do with a true crisis. In my experience, collapse at the

time of the crisis is most unusual, although most text-books always give warning of it and speak of it as if it were very common.

Pulse and Respiration.—The rate of both the pulse and respiration is much increased in pneumonia in early life. Parents and physicians, as well, are often unduly alarmed by this increase, forgetting how rapid the pulse and respiration are normally in early life, how rapidly and from what slight causes their rate is increased, and that in consequence an increase in their rate is of much less importance than in adult life. The rate of the pulse and respiration should always be compared with the rate normal for the age. A pulse of 160 and a respiration of 60 in a baby are, for example, no higher than a pulse of 100 and a respiration of 30 in an adult. In fact, in infancy the pulse rate need not of itself be a cause of anxiety unless it is over 180 or even approaching 200, while the respiratory rate need not of itself cause anxiety until it is over 80.

The rate of the respiration is almost invariably increased out of proportion to that of the pulse, the normal ratio of 1 to 4 being changed to that of 1 to 3 or even 1 to 2. This change is most characteristic of pneumonia in early life; so characteristic that it is of the greatest importance in diagnosis. If the rate of the respiration is not increased out of proportion to that of the pulse, there is almost never pneumonia. If a child has an acute disease with a sudden onset, a continuous high temperature and a decidedly greater increase in the rate of the respiration than in that of the pulse, the chances always are that it is pneumonia. Under such conditions it is always justifiable to make a diagnosis of pneumonia and to stick to it, even if there are no physical signs of pneumonia, until some other satisfactory explanation of the illness is found.

Coughing and Expectoration.—Cough is, in my experience, rather uncommon in the early stages of pneumonia in infancy and childhood, and, even later, in the course of the disease, it is seldom very troublesome. In some cases, however, it is very annoying, especially in babies, if it is painful, as they do not know how to control it. The absence of cough does not, therefore, exclude pneumonia in a doubtful case.

Babies and young children, if they have been properly brought up, do not expectorate and cannot be made to. Older children expectorate less than adults. Even when they do, the typical prune juice expectoration of pneumonia in adult life is seldom seen in childhood.

Pain.—Although pathologically there is almost always inflammation of the pleura in connection with pneumonia in early life, pain is, on the whole, not very common and not very severe, although there are many exceptions to the rule. When babies have pain with respiration and cough, they do one of two things; they either breathe as superficially as possible and keep as quiet as they can or else they breathe deeply, cough, cry and hurt themselves as much as possible, wearing themselves all out. It must be remembered that infants and young children are unable to localize pain accurately and are far more likely to locate the pain in the abdomen than in the chest. When they do locate it in the abdomen, they are, moreover, quite likely to locate it at the navel. Pain in the abdomen is, however, sometimes localized in the right iliac fossa and may be associated with tenderness and rigidity in that region. They often show pain in the chest by lowering the shoulder and hunching themselves down on the affected side in order to use that side as little as possible in breathing. It is often said that patients with pneumonia are more comfortable when they are lying on the affected side as this prevents motion on that side and makes motion of the good side easier. In my

experience this rule does not hold and they are as likely to lie on one side as the other. Babies, however, will sometimes refuse to nurse unless they are placed on the affected side. In other instances, especially if the pneumonia is located at the apex, children hold the head rigidly retracted or inclined to one side. Headache is not uncommon in older children at the onset, but seldom persists. General muscular pains are also common in older children at the onset, and, with hyperasthesia, not infrequently persist throughout the course of the disease.

Digestive Tract.—Loss of appetite is very common, especially in babies. Vomiting is relatively uncommon, except at the onset. It is a bad symptom when it develops later. Parenteral diarrhea is not infrequent in babies. Older children are likely to be constipated. Distention of the abdomen, as the result of impaired digestion and of weakening of the intestinal walls from the toxemia of the disease, as well as from the impaired motility of the diaphragm, is a not very uncommon and a very serious symptom. It interferes with respiration, impedes the action of the heart, shows a marked degree of toxemia and is very hard to relieve.

Nervous System.—Marked drowsiness, in many cases approaching stupor, is so common in babies and young children ill with pneumonia that it may be regarded as one of the characteristic manifestations of the disease at this age. Delirium is not at all uncommon in older children and is not of great significance. Infants and young children often show very marked evidences of irritation of the central nervous system, such as twitching, rigidity or even convulsions. These symptoms, when they occur, are almost never manifestations of pneumococcic meningitis but are due to toxic irritation of the brain or meninges, with or without an increase in the amount of cerebrospinal fluid. In some instances the increase in the cerebrospinal fluid is sufficient to justify the term of serous meningitis. It is these cases which have been spoken of in the past as cerebral pneumonia. The cerebrospinal fluid obtained by lumbar puncture in these cases shows usually an increase in the amount of fluid, often a slight increase in the number of cells, the increase being in the mononuclears, very rarely a slight trace of globulin, but never any pneumococci. There is no relation between the seat of the pneumonia and the occurrence of these symptoms of cerebral irritation. In other cases the symptoms of cerebral irritation are due to a complicating otitis media. Pneumococcic meningitis, when it occurs, usually develops late in the course of the disease. These other conditions may develop at any time, but more often appear early than late.

Urine.—The urine is scanty, high-colored, concentrated, loaded with urates and the chlorides diminished. It almost always contains acetone and diacetic acid in the early days. It not infrequently contains a small amount of albumen, while the sediment shows the evidences of acute degenerative nephritis.

Physical Signs.—The general appearance of a baby or child ill with lobar pneumonia is, as a rule, quite characteristic; so much so, that it is often possible to make a probable diagnosis without any history or physical examination. The patient is manifestly acutely ill, usually quiet, with brightly flushed cheeks and rapid respiration. In addition, the alae nasi often move and the expiration is grunting.

Cyanosis is not common in pneumonia in infancy and childhood, unless the patient is in extremis. The tendency, especially in infancy, is for the patient to become paler and paler. Flushing of the cheeks is common, especially if the patient is kept out of doors or in a cold room.

It is, however, not in any way pathognomonic, as it is present in many other conditions associated with high fever. I am convinced that the belief that the flushing of the cheeks is more marked on or confined to the side on which the lung is affected is erroneous and that, if this at times seems to be the case, the reason for it is that the child has been lying on the affected side, and that, on this account, this cheek is congested. Motion of the alae nasi is common in pneumonia in early life, being more common in infants than in older children. It is, however, not at all pathognomonic of pneumonia; because it occurs in all diseases of the respiratory tract in infancy and not infrequently in other diseases in which the temperature is high, although the respiratory tract is not involved.

Lungs.—The earliest signs in the lungs are ordinarily a diminution in the motility and in the intensity of the respiratory sound on the affected side without any change in its character. When the history and symptoms are suggestive of pneumonia this diminution in the motility and respiratory sound on one side is sufficient to justify a positive diagnosis of pneumonia. In my experience, crepitant râles are very seldom heard in the early stages of pneumonia in infants and young children, although more often in older children.

It goes without saying that the physical signs of pneumonia in the lungs are those of consolidation of the lung; namely, dullness or flatness, bronchovesicular or bronchial respiration and voice sounds, and increased tactile fremitus, the intensity of the changes depending on the size of the area involved and its situation. In general, oscultation gives more valuable and reliable information in the early diagnosis than does percussion, although there are numerous exceptions to this rule. Light percussion is of more value than heavy percussion at the outset, because the solidification almost always begins at the surface, and, if the percussion is hard, the sound elicited from the normal lung below will obscure that from the solidified area at the surface. Great care must be taken not to confuse the quality of the respiratory sound with its quantity. The quantity of sound is usually diminished on the affected side, while the quality of the sound is changed to or toward the bronchial. On the normal side the quantity of the respiratory sound is increased because of the extra work which this side has to do, while the quality of the sound is unchanged. The normal respiratory sound at this age is higher pitched than in later life, and, to the careless observer, not infrequently seems bronchial, especially if it is unusually loud. I find that many physicians often mistake the exaggerated normal puerile respiration for bronchial respiration and locate the pneumonia on the wrong side, failing entirely to appreciate the difference between the normal puerile respiration and bronchial respiration, and being misled by the quantity of the sound which they hear. Puerile respiration need never be mistaken for bronchial respiration if the precaution is taken to listen over the trachea, either in front or behind. The sound heard there is always purely bronchial and will serve as a guide to distinguish one from the other.

It must never be forgotten that the signs of solidification are often very sharply localized for a considerable time and can only be detected by a very careful and thorough examination. It is not enough to listen only in front or only behind. It is not enough to have the clothes loosened or pulled up. They must be removed. Unless they are, a thorough examination is impossible. The first signs are often heard

in the axilla or in the back, near the median line. These spots should never be passed over. In many instances nothing abnormal can be detected until the child is made to breathe deeply. Whenever, therefore, there is any reason to suspect trouble in the lungs, the child must in some way be made to breathe deeply, even by being made to cry, if necessary. In many instances a change in the voice can be detected before any change in the respiration. The voice should also be tried, therefore, in all doubtful cases.

Signs of solidification are often entirely lacking or very indefinite during the early days of the disease, not appearing until the fourth or fifth day. In some instances they may not appear until at the time of or even after the crisis, and sometimes they may always be lacking. It is, however, perfectly possible to make a diagnosis of pneumonia in the absence of physical signs; in fact, the general picture of the disease is usually amply sufficient to justify a diagnosis and the physical signs in the chest are merely corroborative evidence and serve to locate the lesion. In general, no râles are heard over the solidified area until resolution begins, although there may be. If râles are heard, they are moist and high-pitched. During the stage of resolution they are moist, but become less and less high-pitched as resolution goes on. In uncomplicated cases the rest of the lungs is normal. When the location of the pneumonic process has been determined, it does the patient no good, and may do it much harm, to examine the lungs repeatedly. Once a day is amply sufficient. It makes no difference to the patient whether a part or the whole of a lobe is involved. It makes no difference in either prognosis or treatment whether the area involved is a little more or less dull, or the respiration bronchial or bronchovesicular. The examination merely disturbs the patient.

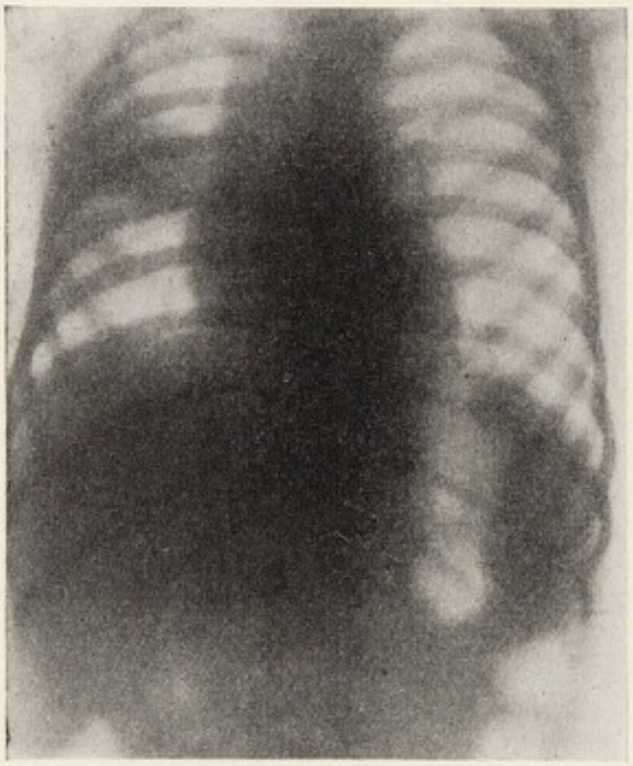

FIG. 114.—Pneumonia. Triangular shadow, with base at surface, in right lung.

Roentgen Ray Examination.—There has been much discussion in the past as to the existence of *central pneumonia* and the reason why the physical signs are often so late in developing. The recent investigations with the Roentgen ray seem to have settled these points very definitely. These show that the pneumonic process never begins in the center of the lobe but always on the surface. The first shadow is triangular in shape, the base being at the surface of the lung. This triangle is often visible when there are no physical signs. In many instances more careful examination in this area will reveal slight signs which were not previously noted. The absence of physical signs may be due to the small size of the area of solidification, to the fact that it does not reach a large bronchus or to its situation high in the axilla, beneath the shoulder joint, or at the base

of the lung. The triangular shadow gradually increases in size, the apex drawing nearer to the root of the lung. Finally it is obscured as the process extends and becomes more general. It reappears during resolution and is the latest, as well as the earliest, picture. A positive diagnosis can often be made with the Roentgen ray several days before it can be made from the physical signs. It is rarely necessary, however, to weary the patient by taking it to the Roentgenologist for examination or to undergo the expense necessary to bring the Roentgenologist to it in order to make an early diagnosis, except in cases where there is doubt as to whether the trouble is pneumonia or appendicitis.

Heart.—The heart should be watched most carefully in lobar pneumonia. The points to be noted especially are its size, the strength of the first sound and the strength of the second pulmonic sound. The most important point to be noted is the strength of the second pulmonic sound. If this is accentuated, it shows that the right heart is meeting the increased pressure in the pulmonic circulation. If it is not accentuated or is weakened, it shows that the heart is not strong enough to do the work which is demanded of it and is a cause for great anxiety. The second pulmonic sound usually begins to weaken before there is any change in the character of the first sound and before there are any evidences, on percussion, of dilatation of the right heart.

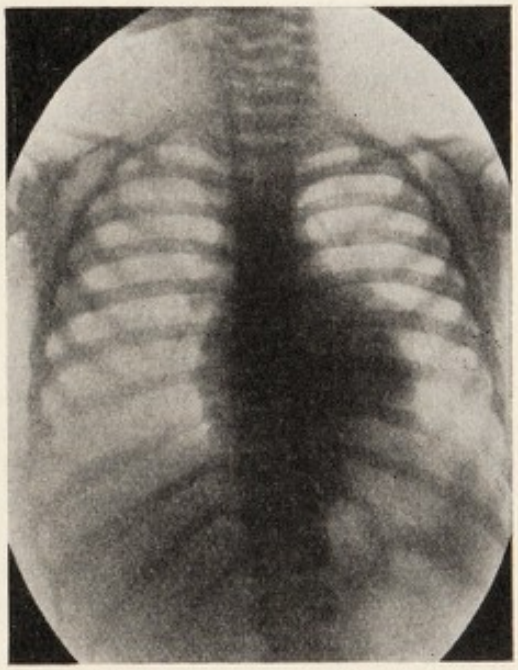

FIG. 115.—Pneumonia. Shadow at left of heart border.

Abdomen.—Distention of the abdomen, as the result of distention of the intestines, is not very uncommon. Enlargement of the liver sometimes occurs. It is sometimes due to engorgement of the organ with blood and sometimes to fatty degeneration as the result of the toxemia. In some instances it is associated with jaundice. When this occurs, both the enlargement and the jaundice are, presumably, due to a catarrhal inflammation of the bile ducts. The spleen is never enlarged.

Extremities.—The knee-jerk is often diminished in pneumonia. This fact is, however, not of great importance and is hardly constant enough to be of much value in diagnosis.

Blood.—There is always a marked polynuclear leucocytosis in pneumonia, except when the infection is very mild or when the patient is overwhelmed by a severe infection. The increase in the leucocytes begins almost immediately, soon reaches its maximum, where it remains until the crisis, after which it usually drops rapidly. The height of the leucocytosis is of no importance. It commonly ranges from 40,000 to 50,000 and may often go over 100,000. The absence of leucocytosis in a child with pneumonia who is evidently not seriously sick is not a bad sign. Its absence in a child with pneumonia who is seriously sick is a very bad sign. The absence of leucocytosis in a child who is suspected to have pneumonia, but is evidently not overwhelmed by any infection, is very strong evidence indeed that the difficulty is not pneumonia.

Complications.—Otitis media is the most common complication of pneumonia in infancy. It is, in fact, found invariably post-mortem in fatal cases. It is usually not due to the pneumococcus but to some other organism. It should always be thought of when the temperature is higher or lasts longer than would be expected or when a baby seems sicker than it ought to be, even if there are no symptoms pointing directly to the ears. There is almost always a dry pleurisy of greater or less extent in pneumonia in infancy and childhood. Nevertheless, a friction rub is almost never heard in infancy and very seldom in childhood. It is not at all uncommon, especially during the latter days of the disease, to have a small serous or bloody serous exudate in the pleura. This usually is not more than two or three ounces. Empyema develops during or after pneumonia in, perhaps, one case in twenty. Parents should always be warned at the beginning of pneumonia that an empyema may develop later, in order to prevent criticism, if it does appear. Pericarditis is an uncommon complication but occurs more frequently in infancy than in childhood. If it occurs, it is likely to be fibrinopurulent. Endocarditis is most unusual. Pneumococcus meningitis occasionally develops late in the disease. So also does pneumococcus peritonitis. Both are purulent. Acute nephritis is not very uncommon. Both parotitis and arthritis may occur.

Diagnosis.—The onset of lobar pneumonia is like that of many other acute infections, such as scarlet fever and tonsilitis or of acute disturbances of digestion. All begin acutely, with high fever, vomiting and general evidences of infection. In pneumonia, however, the increase in the rate of the respiration is decidedly greater than that in the rate of the pulse. If, therefore, when a child has an acute disease with a high temperature, the rate of the respiration is increased out of proportion to that of the pulse, the chances are that it has lobar pneumonia. If, on the other hand, the rate of the respiration is not increased out of proportion to that of the pulse, the chances are that it has not pneumonia. Exceptions to this rule may be acute appendicitis, which often causes an increase in the rate of the respiration, and otitis media or infections of the upper respiratory tract in babies, which also often increase the rate of the respiration. Motion of the alae nasi is suggestive of pneumonia, but nothing more. Flushing of one or both cheeks is of no importance. The absence of cough and pain does not count against pneumonia. Grunting respiration points strongly towards it. The presence of polynuclear leucocytosis is, as a rule, of very little importance, because almost all of the diseases with which pneumonia may be confused have a leucocytosis. The degree of the leucocytosis is of no assistance. The only exception is typhoid fever and possibly influenza. The onset of typhoid fever is usually much slower, however, and the picture of influenza is materially different from that of pneumonia. It should go without saying that repeated careful physical examinations should be made, not only of the lungs for the early signs of pneumonia, but elsewhere for the evidences of other diseases. The early signs of pneumonia have already been described.

Pneumonia with marked cerebral symptoms sometimes closely resembles meningitis. As a rule, the relation between the pulse and respiration will justify a probable diagnosis. An examination of the chest with the Roentgen ray will settle many doubtful cases. In others, however, a lumbar puncture is necessary. If cerebral symptoms occur at the beginning of pneumonia, the chances are that there is no meningitis. If there is, it is usually not due to the pneumococcus. If they occur late and there is a meningitis, it is probably pneumococcal in origin.

It is often difficult in infancy to distinguish in the beginning between otitis media and acute nasopharyngitis and pneumonia. Both otitis media and nasopharyngitis may begin very acutely, have a high temperature with the rate of the respiration increased out of proportion to that of the pulse and be accompanied by motion of the alae nasi and cough, without any definitely localizing symptoms. Careful examination of the ears, nasopharynx and chest will usually quickly settle the diagnosis. Many physicians, however, either forget or neglect to examine the ears and thus make a diagnosis of pneumonia when the trouble is really in the ears.

Another very difficult diagnosis is that between pneumonia and appendicitis in childhood. While the diagnosis seems simple on paper, it is often actually very difficult. It is, moreover, very important to make the right diagnosis, as, if appendicitis is mistaken for pneumonia and operation neglected, the patient will probably die. On the other hand, if pneumonia is mistaken for appendicitis and the abdomen opened, the operation will probably prove fatal. Appendicitis is seldom mistaken for pneumonia, but pneumonia is not infrequently mistaken for appendicitis. The reasons for this mistake are the reference of pain in the chest to the abdomen, the rigidity of the abdomen as the result of diaphragmatic pleurisy, the initial vomiting and the not infrequent distention of the abdomen and constipation in pneumonia. In pneumonia the temperature is usually higher, the leucocytosis is more marked, the rate of the respiration is increased out of proportion to that of the pulse, there is a cough, there is no localized abdominal pain or tenderness, the distention of the abdomen is uniform and there is no localized muscular spasm. Unfortunately, however, the temperature may be very high in appendicitis, the leucocytosis varies markedly in both diseases, the rate of the respiration may be increased in appendicitis out of proportion to that of the pulse because of the abdominal pain caused by deep respiration, cough is often absent in pneumonia, the child is often unable to locate pain and tenderness accurately and the presence or absence of localized abdominal spasm cannot be determined because of the child's resistance to examination. In most instances, however, if the disease is pneumonia, a careful examination of the chest will show some signs of trouble there and reveal the real seat of the trouble. A negative rectal examination is also strong evidence in favor of pneumonia. Such an examination should never be omitted in a doubtful case. An examination with the Roentgen ray will, moreover, almost always settle the question. It must not be forgotten, however, that pneumonia and appendicitis may develop at the same time and that either may follow the other.

The differential diagnosis between lobar pneumonia and bronchopneumonia is taken up in the chapter on bronchopneumonia, and that between pneumonia and empyema in the chapter on empyema.

Prognosis.—Pneumonia is a comparatively mild disease in childhood. It is the exception for a child to die of an uncomplicated pneumonia. In infancy, however, pneumonia is a very serious and often fatal disease. The mortality in hospitals varies between 25% and 33%. It is much lower than this in private practice, but nevertheless high enough to place it among the more serious conditions. It is self-evident that the prognosis is not as good in debilitated and feeble children, in those with deformed chests from rickets and in those who can not receive proper care. It is not quite as good when more than one lobe is involved

at a time, and there is always danger that a child may be worn out or overcome by the toxemia when several lobes are involved in succession.

A high temperature is not a bad sign unless it is continuously over 105° F., and even then most cases recover. An occasional rise to 106° F. or even higher is not of bad prognostic import. If the temperature is continuously under 102° F., the outlook is serious, unless the child is evidently not much sick. Rapid pulse and respiration are a part of the disease. The rate must always be compared with that normal for the age and due allowance made for the ease with which they are increased in early life before an increase causes anxiety. The height of the leucocytosis is of no importance in prognosis. A low white count in an obviously very ill child is, however, of serious import. The prognosis is not quite as good in those cases in which pneumococci are found in the blood as in those in which they are not. In the individual case, however, their demonstration is of very little importance. Convulsions late in the disease are bad signs. The worst signs are cyanosis, distention of the abdomen and weakening of the second pulmonic sound. Resolution usually takes place within a few days or, at most, a fortnight. In some cases, however, it may be delayed for many weeks or even months. In such instances the patient usually has no fever, does not feel sick, but is short of breath on exertion and easily fatigued. When the lung begins to clear, it usually does so quite rapidly. In other rare instances chronic interstitial changes and chronic bronchiectasis develop. Tuberculosis of the lungs is a very uncommon sequela of pneumonia in early life.

Treatment.—The quest for a specific treatment for pneumonia is almost as old as that for the philosopher's stone or that for the fountain of youth, and up to very recently has been as barren of results. It is certain in the first place that no drug, whether creosote, quinine, or any other, has any specific action in pneumonia. It is possible that leucocytic extracts may have some little value, but their action, if they have any, is certainly not specific.

Vaccines.—Monkeys have been protected against experimental pneumonia by the use of living cultures subcutaneously or intravenously, and by intratracheal inoculations. The injection of killed cultures has, in considerable series of human beings, apparently somewhat reduced both the morbidity and mortality of this disease. Immunization against pneumonia by the use of vaccines is, nevertheless, still in the experimental stage and the evidence in its favor is not sufficient to justify its general adoption.

"Preventive inoculation has promise of success because by this means it is possible to stimulate the production of an active immunity. But an active immunity takes time for its development. There is little theoretical justification for the belief and no evidence, direct or indirect, that dead, living, or autolyzed pneumococci used as a vaccine can be of any service whatever in the treatment of pneumonia. In the presence of the existing infection it cannot be assumed that the production of active immunity will be more rapidly induced by the injection of a few million dead organisms under the skin than by the natural infection itself, and there is danger that the patient's power of resistance may be taxed beyond its capacity to respond." (The Commonhealth, 1923, Volume 10, page 55.)

Serum Treatment.—The pneumococci may be divided into three distinct types, Types, I, II, and III, and a group, known as Group IV, made up of a number of individual strains. It is possible to produce

antisera which confer immunity against the special type of organism or a single strain of Group IV. The antiserum is, however, specific for the type of organism and confers no immunity against organisms of any other type. Experimentally, these antisera also have a curative action in animals. A serum sufficiently potent for the treatment of man has been obtained only against Type I pneumococci. As the antiserum is specific, it cannot, of course, be expected to do any good except in those cases of pneumonia which are caused by Type I pneumococci. Not more than ⅓ of the cases of pneumonia in adults is due to Type I, while the proportion is probably considerably smaller in infancy and childhood. Considerable series of cases in adults have seemed to show that the antiserum reduces the mortality, if it is used early. More recent series however, seem to prove that its efficacy has been much overestimated. There are no data sufficient to justify any conclusions as to its usefulness in early life. There is no reason to suppose, however, that its action is any different at this age than later. All agree that to do any good the antiserum must be given during the first three days of the disease and that, the earlier it is given, the more likely it is to do good.

Since the antiserum is specific against Type I only, can do no good when pneumonia is due to organisms of other types, and the intravenous injection of horse serum may cause undesirable, or even dangerous, reactions, it is certainly most desirable, if not absolutely necessary, to determine the type of organism in the individual case before giving the serum. This may be done by using the sputum or the material obtained by lung puncture for injection into mice, or by making blood cultures. The sputum must be obtained from the lungs, not from the upper air passages. A very little will suffice, but it must come from low down. It is hard to get in babies and young children, but can often be obtained if the swab is pushed way back and the patient made to gag and cough. This method is usually more successful than the introduction of a catheter into the upper part of the larynx. No antiseptics should be added to the specimen and it should be kept as cold as possible. It is evident that, unless laboratory facilities are immediately available, the results of the examination will not be known until it is too late for them to be of any practical use.

Everyone who has explored the chest in cases in which it was suspected that there might be a pleural effusion knows that there is but little danger in puncturing the lung. Nevertheless, when the lung is punctured, there is always the possibility that the lung or the pleura may be infected, or a hemorrhage into the pleura or a bronchus result. Furthermore, the operation is painful and causes a certain amount of shock. It hardly seems justifiable to puncture the lung in pneumonia in children, therefore, when the low mortality at this age, the small proportion of cases due to Type I pneumococci, the difficulty in determining the type of organism, unless a laboratory equipped for the purpose is close at hand, and the somewhat questionable results of the serum treatment are borne in mind.

If the antiserum is used, it must be a reliable one and free from hemoglobin and sediment. It must be given intravenously. This is a rather difficult procedure in infants and young children, but it can be done by those who are proficient in the technic. The dose for an adult is 100 c.cm. A corresponding dose for an infant would be between 10 c.cm. and 15 c.cm. and for a child of four or five years, 20 or 25 c.cm. This amount should be given every eight or twelve hours until the infection is overcome or the patient is dead.

The chances of death from anaphylactic shock, while very small, are much greater when large amounts of horse serum are given intravenously, as in pneumonia, than when relatively small amounts are given intramuscularly, as in diphtheria. Anaphylactic shock is especially likely to occur in children who have had asthma or hay fever and in those who have had an injection of horse serum within a short time, provided the interval is not less than ten days. Antipneumococcus serum should never be given to such children, unless it has been previously proven that they are not sensitive to horse serum or they have been desensitized. In fact, it is always inadvisable to give horse serum intravenously, unless it has been shown that the child is not sensitive to it or has been desensitized. Sensitiveness to horse serum may be tested by injecting *into* (not *under*) the skin 0.02 c.cm. of sterile normal or immune horse serum diluted 1:10 or 1:100 with sterile physiologic salt solution. A control injection of an equal amount of sterile physiologic salt solution should be made at the same time. In sensitive children an urticarial wheal, surrounded by a zone of erythema, appears in a few minutes. The zone of erythema gradually increases in size for about an hour, sometimes becoming as large as a 50 cent piece. It then gradually fades out. No such reaction occurs at the site of the control injection. It is most important that the injection is given into the skin, not under it, because no reaction will occur, even in sensitive children, if it is given subcutaneously. Although a negative intradermal test does not absolutely prove that a child is insensitive to horse serum, it is safe for practical purposes to assume that it is and to act accordingly. If the test shows that it is sensitive, it should be desensitized before the serum is given, unless it has a history of hay fever or asthma. If it has, the dangers from the use of horse serum, even with attempted desensitization, are so great that it is hardly justifiable to use it in a disease like pneumonia, in which the chances of benefit from its use are so doubtful. Desensitization is accomplished by the injection *under* the skin of 0.5 c.cm. of normal or immune horse serum. An intravenous injection of antiserum may be safely given in a few hours later.

It is evident, therefore, that when the low mortality from lobar pneumonia in early life, the small proportion of cases at this age due to infection with Type I pneumococci, the difficulties attendant on the determination of the type in children, the difficulties and dangers in the administration of the antiserum, and the rather slight expectation of benefit from its use are taken into consideration, the field for the serum treatment of lobar pneumonia in early life is decidedly limited.

Fresh and Cold Air.—I am convinced from clinical observation that children with pneumonia who have an abundance of fresh, cold air are more comfortable, cough less, have a better color, eat better and show fewer symptoms of nervous irritation than those who do not. I cannot truthfully say, however, that I think this method of treatment has any effect on the mortality from this disease. It was thought at one time that the favorable results obtained with it were due to the raising of the blood pressure, which was supposed to be below normal as the result of vasomotor failure. It has since been shown, however, not only that the blood pressure is not lowered in pneumonia but that it is not raised by exposure to cold air. The explanation of the beneficial action of cold, fresh air is, therefore, unknown, as is likewise whether it is due more to the coolness or to the freshness of the air. I cannot help believing that one of the reasons that cold air does good is that it keeps everyone out of the sick room except those who are obliged to be there.

Babies and children ill with pneumonia should be given the greatest possible amount of cold, fresh air. The only exceptions are feeble and debilitated babies, who do not bear cold well. It is not necessary to put them out of doors nor is it necessary to have the temperature down to freezing. I have, however, never seen any harm come from either. The windows should be wide open, but the patient should, if necessary, be protected from drafts by a screen. It is advisable to get the temperature of the room down to 50° F., if the outside temperature allows it, but there is no especial advantage in having it lower. Sunlight should not be excluded.

It goes without saying, or should, that, when a child is kept in a cold room, it should be warmly covered and should wear a cap, and perhaps mittens, just as if it were out of doors. It should also have a heater of some sort at its feet. Those who are taking care of the child should also be dressed for out of doors. The room must be warmed, of course, when the child is changed or bathed, or it may be taken into another room.

Food and Water.—It is very important to give children with pneumonia sufficient food to keep up their nutrition. There is no especial indication as to the kind of food, except that it must be easily digested, nutritious and not too bulky. Milk should, as almost always, form the basis of the diet. It may be given in many different ways. Gruels and cereals are suitable. Broths have no nutritive value and interfere with the taking of other food. Eggnogs are heavy and indigestible. Care must be taken not to upset the stomach with excessive amounts of the various fruit juices. Children with pneumonia cannot be expected to take much, if any, solid food, as the effort of chewing interferes with respiration and tires them. In general, it is better to give comparatively small amounts at relatively short intervals in order to avoid distention of the stomach and embarrassment of the diaphragmatic respiration. The intervals should not be less than one hour in babies and two hours in children, however, because the too frequent offering and taking of food not only disturbs the digestion, but also fatigues the child and prevents it from getting sleep and rest.

Water should be given freely. It is difficult to say just how much should be taken, but certainly not less than eight ounces a day by babies and a quart by children. They must not be bothered too much to take it, however, because rest is even more important than water.

Quiet.—Children ill with pneumonia should be left alone and disturbed as little as possible. One of the reasons that children with pneumonia do so well in hospitals is that someone is not fussing over them all the time. No one should be in the room except those who are taking care of the patient. Treatment must be given at as long intervals as possible, and, when feasible, given at the same time as food. Frequent examinations are unnecessary and harmful. It is of no advantage to take the temperature every few hours, and disturb the child by doing it, just to mark it down on a chart. Nothing should be done to or for a child sick with pneumonia that is not absolutely necessary. It is not at all impossible to take away a child's chances of recovery in a critical case by too active and energetic treatment and nursing.

Stimulation.—Stimulants should not be given as a routine measure, but only for definite indications. They are not needed in the vast majority of the cases of pneumonia in childhood. Whiskey is not a stimulant and cannot be given in sufficient amounts to be of any use as a food without disturbing the digestion or "doping" the patient. I can see

no justification for the routine use of digitalis in pneumonia. None of the indications for it are present at the height of the disease and it cannot, therefore, do any good. It may sometimes be useful late in the disease, if auricular fibrillation and failure of compensation have developed. Stimulants, when necessary, should be used in the same way as in bronchopneumonia, where their use has already been described.

External Applications.—External applications to the chest can do no good and may do much harm. It is absolutely impossible for any application, whether hot or cold, on the outside of the chest to modify bacterial activity in the lung or to have any effect on an exudation in the alveoli. Poultices may do active harm by embarrassing the respiration. It is difficult enough in any case for a baby or a young child, ill with pneumonia, to breathe. It is much harder for it if it has to lift a pound or two of poultice with each respiration. Poultices tend, moreover, to keep up the temperature and their application is disturbing.

It is equally idle to suppose that the application of mud, whether scented or unscented, under its own name or some other, can have any more effect than poultices. Mud is somewhat less dangerous than poultices, however, in that as it costs more it is usually applied les thickly.

Cotton jackets are less objectionable than poultices and mud in that they are not as heavy and usually are not as hot. If the child is given the cold air treatment, they are not especially objectionable, as a cotton jacket will keep a child warm as well as any other jacket. They are not any better, however, than the child's ordinary clothes.

Treatment of Symptoms.—*Fever.*—It is very seldom that a child ill with pneumonia needs any active treatment for the fever. The temperature is and must be high in pneumonia. In most instances children bear it well. An attempt to reduce it should not be made simply because it reaches a certain point on the thermometer, 103° F., 104° F., or 105° F., as the case may be. If the child is not depressed by the fever, unduly stupid or showing marked symptoms of nervous irritation, the attempt to reduce it will probably do more harm to the child by disturbing it and interfering with its rest than it will do good. In fact, it is quite probable that the fever may be of advantage rather than the opposite. If the child is depressed by the fever, is very stupid, or showing symptoms of nervous irritation which are apparently due to the height of the temperature, rather than to toxemia, an attempt should be made to reduce it. Coal-tar antipyretics should never be used for this purpose in pneumonia. The application of cold externally is the only safe method. Sponging with alcohol and water, equal parts, at 90° F., if properly done, is usually sufficient to bring down the temperature. If it does not, fan baths, which are somewhat more active, may be tried. The most efficient method of reducing the temperature and also the one least objectionable to the child, however, is the cold pack. This is true not only in pneumonia but also in other febrile conditions in early life. (See treatment of typhoid fever for description of the use of cold externally.)

Pain and Cough.—There is no objection to the use of poultices, whether of mustard or flaxseed, temporarily for the relief of pain. A hot water bag or an electric heating-pad is usually equally good and much easier to handle. An ice-bag is, however, often more efficacious. Strapping the chest or a binder seldom afford much relief and, if they are applied tightly, interfere with the motility of the normal side. There is also no objection to the administration of some preparation of opium, provided the rest of the lungs is clear. Opium is also indicated for the

relief of a painful cough under the same restrictions. If the rest of the lungs is not clear, it is very dangerous to use opium, because, if given in sufficient doses to relieve the cough, it prevents the proper clearing of the bronchi and thus predisposes to bronchopneumonia and atelectasis and interferes with the aëration of the lungs. Paregoric contains so little opium that it is not of much use for the control of pain, except in babies. Codeine or the phosphate or sulphate of codeine, in doses of from $\frac{1}{16}$ of a grain to $\frac{1}{2}$ a grain, may be used, according to the age and individual susceptibility of the child. Morphine sulphate, in doses of from $\frac{1}{64}$ of a grain to $\frac{1}{8}$ of a grain, may also be given. Whenever opium or any of its derivatives are used, a small dose should be given first because so many children are susceptible to opium. If the pain is not relieved, the dose should be increased until it is, provided the rest of the lungs is still free.

The various expectorants and cough mixtures are worse than useless in that they cannot have any effect upon a solidified lung and are very likely to disturb the digestion.

Restlessness and Nervous Irritation.—These symptoms are seldom troublesome if the patient has a sufficient amount of fresh, cold air and is not disturbed and worried by an excess of treatment. If they are due to the fever, lowering the temperature by bathing will relieve them. An ice-cap to the head often helps. Sedatives may be given, if required. It is seldom necessary, however, to give any more powerful drugs than the bromides.

Vomiting.—Vomiting is not a very common symptom. It has to be treated by regulation of the diet on the same principles as when it occurs in other diseases. It is often advisable to stop food entirely for six to eight hours. When it comes on late in the disease and is due to toxemia, little can be done to relieve it.

Distention of the Abdomen.—The first thing to do is to regulate the diet, cutting down the carbohydrates, if the stools show evidences of fermentation, and the proteins, if they show evidences of putrefaction. If the distention is marked, it is advisable to give castor oil or some other drug to clean out the bowels. It is not right to repeat the purgation daily, even if the distention persists. The lower bowel should be washed out with a large enema of soapsuds. In my experience the stronger enemas are but little, if any, more effective. Turpentine enemas, however, sometimes afford relief when nothing else will. The enemas may be repeated as necessary. Very little relief is afforded by a rectal tube; in my experience it is hardly worth trying. Hot applications on the abdomen sometimes help. Turpentine stupes are the best.

Finally, judging from the statistics from large numbers of cases collected during many years and treated by many and radically different methods, it is evident that no form of treatment has any materially favorable influence in pneumonia in early life. While no form of treatment does much good, it is however, very easy to do much harm by overzealous and injudicious treatment and to seriously diminish the chances of recovery. In treating pneumonia in early life Baginsky's dictum—"*Nil nocere*"—should always be borne in mind.

CHRONIC INTERSTITIAL PNEUMONIA

In some instances the lung does not clear up after lobar pneumonia and organization of the fibrinous plugs within the alveoli takes place. There is an overgrowth of connective tissue in the alveolar walls, in and

about the bronchi, and in the frame work of the lungs. The same process eventually extends to the pleura. In fact, in some instances it may start from a pleurisy and extend inwards. It also often extends to the mediastinum and to the pericardium. The newly formed connective tissue gradually contracts and compresses what alveolar tissue has not been already involved. The pull of the fibrous tissue on the pleura and the contraction of the pleura itself reduce the size of the lung and pull in the chest wall. The adhesions to the mediastinum and pericardium pull over the mediastinum and heart. The bronchi may or may not be dilated by the pull of the new fibrous tissue. If they are, all the changes of bronchiectasis may develop. Compensatory emphysema develops, of course, in the other lung.

In other instances similar changes may develop more or less rapidly and more or less diffusely throughout both lungs after acute bronchopneumonia or as the result of repeated attacks of acute bronchopneumonia. When this happens, the progress of the pathologic process is much slower and the changes usually much less marked than in the first type.

Symptomatology and Prognosis.—When chronic interstitial pneumonia follows lobar pneumonia, the signs of solidification persist in the lung, the temperature continues to be a little elevated after the crisis, cough and rapid respiration persist, and the child, although no longer acutely ill, does not get well. It finally gets up and about, but is short of breath on exertion, often a little cyanotic, coughs more or less but does not raise much, has a little irregular fever from time to time, and is manifestly not well. If bronchiectasis develops, the symptoms of this condition are added to those of the chronic interstitial pneumonia. In some instances the child becomes bed-ridden after a few years and finally dies of some intercurrent pulmonary infection, if it has not already died of one before it was forced to take to its bed. In other instances, death occurs as the result of chronic septic absorption from the bronchiectatic cavities. It is possible, however, that in very exceptional cases the lung may be so replaced by fibrous tissue that no further changes occur and the child live to adult life, a semi-invalid with one lung and a hampered heart. The course of the disease is the same when it develops as the result of pleurisy.

When the interstitial changes develop as the result of acute bronchopneumonia, the symptoms are very variable and not very different from those of a chronic bronchitis of varying severity. As the pathologic changes increase and more and more lung tissue is invaded and bronchiectatic changes develop, the symptoms are those which must necessarily accompany such a pathologic condition, that is, disturbance of nutrition, loss of weight and strength, irregular fever, dyspnea and finally those of septic absorption. The pathologic process may, however, stop at any stage. If it stops before much damage has been done, the child may be but little injured by it and, although perhaps not of average resistance and vigor, live as long as if it had not had it. In most instances, however, even if the child survives, so much damage has been done that it continues to be a pulmonary invalid or semi-invalid. In the more serious cases death almost always occurs after a number of months or years.

Physical Signs.—In the type following lobar pneumonia and pleurisy the affected side is smaller than the other and its motility is diminished. There is usually some scoliosis of the spine. The cardiac impulse is displaced towards the affected side or absent. There is dullness or flat-

ness over the affected side, the amount depending on the degree and extent of the changes in the lung. The cardiac dullness on the affected side merges with that of the lung, while the opposite border is displaced towards the affected side. The respiratory sound is diminished in intensity, chiefly because of the diminished motility of the chest on the affected side, partly because of the thickening of the pleura. Its intensity also depends upon whether the bronchi are dilated or compressed. Its character is bronchial or bronchovesicular, according to the amount of fibrous tissue present. If there are bronchiectatic cavities near the surface, it may be amphoric or cavernous. The voice sounds vary in the same way as those of the respiration. The tactile fremitus is variable, but usually increased. Because of the associated changes in the bronchi, râles, usually high pitched, are often heard. The sense of resistance may be much increased, almost as much as when there is an effusion in the pleural cavity.

As the result of compensatory emphysema of the other lung, that side of the chest is larger and its motility increased. It is hyperresonant on percussion. The respiration is exaggerated, but normal in character. The voice sounds are loud. There is no change in the tactile fremitus and no râles are heard.

In the type following bronchopneumonia the physical signs are for a long time very slight, usually being limited to a little impairment of the motility of both sides of the chest and of the intensity of the respiratory sound, and those of bronchitis. As time goes on and the fibrous changes become more marked, there is more or less dullness, usually irregularly distributed on both sides, which may eventually here and there become flatness. The respiratory sound is diminished in intensity and changed from the vesicular towards the bronchial in various areas, according to the location and degree of the pathologic changes. In advanced cases in which there are large fibrous areas, the respiration is bronchial, while if there are bronchiectatic cavities, it may be amphoric or cavernous. The voice sounds and tactile fremitus vary with the respiration. Râles are almost always present.

In both types, but more often in the first, there may be clubbing of the fingers. Passive congestion of the liver is not uncommon. If the action of the heart is much impeded or the great vessels compressed by the fibrous changes in the mediastinum, the general manifestations of passive congestion develop and may become very marked.

The Roentgen ray shows but little, if anything, more as to the pathologic changes in this condition than can be discovered by physical examination. In some instances, however, it reveals bronchiectatic changes and cavities which cannot be discovered by physical examination.

Treatment.—There is very little which can be done in this condition except to take the best possible general care of the patient and to protect it from acute infections. Children with it, especially those who have the diffuse type following bronchopneumonia, undoubtedly are more comfortable and do better in high dry climates and, in the winter, in warm Southern climates than they do in the climate of New England and those similar to it. It is possible that residence in such climates may sometimes limit or arrest the progress of the pathologic condition. At any rate, change of climate should be tried, if it is financially feasible. There are no drugs which in any way affect the progress of the disease, although the expectorants and opium, if used as in the treatment of bronchitis, often relieve some of the distressing symptoms.

ABSCESS OF THE LUNG

Multiple small abscesses of the lungs are often found at autopsy in fatal cases of bronchopneumonia, especially if it is due to influenza. It is not known whether such abscesses are present in cases which recover. There is no way at any rate of recognizing them during life.

Single abscesses, which may be either large or small, are not very uncommon. They are usually due to the inhalation of a foreign body and are, therefore, most often situated in the right lower lobe. The objects most commonly inhaled are pieces of tonsils and adenoids. An abscess may also develop as a sequela of lobar pneumonia. An abscess of the lung in childhood is seldom tuberculous. If so, it is sometimes due to the breaking down of tuberculous tracheobronchial glands. It may also be due to the breaking through of an empyema. The organisms found vary with the cause of the abscess. In most cases there is a mixed infection. In some instances, especially in feeble and debilitated infants, the lung may become gangrenous.

Symptomatology.—When an abscess is due to the inhalation of a piece of tonsil or adenoid, from two to four weeks usually elapse before the appearance of any symptoms. The interval may be much longer when the abscess is due to the inhalation of some other foreign body. The first symptoms in these cases are a slight, irregular fever, cough, and sometimes pain in one side of the chest. The temperature becomes higher and more definitely septic and the general consitutional symptoms of a purulent focus in the body develop. If the abscess connects with a bronchus, there is a considerable amount of foul, purulent sputum, which may be coughed up intermittently. If it does not, there is but little sputum.

When an abscess is due to the breaking down of the lung tissue after pneumonia, the symptoms are essentially the same as in empyema, except that, if the abscess opens into a bronchus, the sputum is profuse and purulent instead of slight and non-purulent. If the abscess is due to the breaking through of an empyema, there is no change in the symptoms until the abscess breaks through into a bronchus, when the pus begins to be coughed up. When the abscess is tuberculous, there is nothing about the symptoms to distinguish them from those of the tuberculous process to which the abscess is secondary, unless it be the intermittent raising of considerable amounts of purulent sputum.

Physical Signs.—When the abscess is closed, the physical signs vary according to whether the abscess is near the surface or not. If it is, there is localized dullness or flatness with diminished respiration, voice sounds and fremitus, and an increased sense of resistance. If it is located deep in the lung, the only abnormality is slight dullness on strong percussion. If the abscess is open, that is, connected with a bronchus, the physical signs are the same as in the closed form, when the cavity is full. When it is empty and near the surface, the percussion note is some modification of tympanitic, according to whether there is solidified or normal lung about it; if it is deep in, the tympanitic note is fainter or even absent. If near the surface, the respiratory sound is loud and bronchial, sometimes being modified to amphoric or cavernous. If it is deep, the sound is broncho-vesicular. The voice sounds vary in the same way as those of the respiration. The tactile fremitus is increased, if the abscess is near the surface. Moist râles are often heard.

The Roentgen ray is often of great assistance in locating abscesses deep in the lung, especially in cases in which they are due to the breaking

through of an empyema. It will also show if the abscess is due to a foreign body, provided it is one which casts a shadow.

Diagnosis.—When a child develops the symptoms described above from two to four weeks after an operation for the removal of tonsils or adenoids, the probability is that it has an abscess of the lung. This is also the case, if there is a history of the inhalation of a foreign body, even if it happened some time before. The presence of the symptoms of confined pus after pneumonia, when there are no evidences of empyema outside of the lung, points to either an abscess of the lung or an interlobar empyema. The location of the physical signs in an area corresponding to one of the interlobar spaces is in favor of empyema; their location elsewhere, of abscess. The Roentgen ray will usually settle the question, which is, however, unimportant, as the treatment is the same in both. It is often very difficult to distinguish between a large bronchiectasis and an abscess of the lung. In general, the onset of an abscess is more acute and develops quickly after an operation for the removal of tonsils or adenoids or an empyema. Either may develop after the inhalation of a foreign body. If, after pneumonia, the process in the lungs is localized, an abscess is the more probable; if the whole lung is involved, there is more likely to be a bronchiectasis. If the pneumonia was due to influenza, bronchiestasis is more common than abscess.

When there is reason to believe that there may be an abscess in the lung, exploratory punctures should be made, preferably with the aid of the fluoroscope. It is often very difficult, however, especially if the abscess is small, to locate it, even after several trials.

Prognosis and Treatment.—Recovery is uncommon when an abscess follows pneumonia and practically never occurs without an operation. Recovery is a little more probable when the abscess is due to the breaking through of an empyema. The empyema should always be operated upon. When this is done the abscess usually drains through the pleura, but a secondary operation may be necessary. If an abscess is due to a foreign body and the foreign body is coughed up with the pus, there is a reasonable chance of recovery without operation. If it is not, operation is necessary. When an abscess is due to the inhalation of a piece of tonsil or adenoid, the chances are that the abscess will in time heal without interference. It is advisable, therefore, to be patient in these cases and not to operate unless the child shows marked symptoms of septic absorption. Time, rest, fresh air, sunlight and food do wonders in these cases. Drugs, except for the relief of symptoms, are of very little use.

PLEURISY

The pleura does not extend as high in the neck, as far under the sternum or as low down in the thorax in infancy and early childhood as in later life. The differences are so slight, however, that they are of no practical importance. Inflammation of the pleura occurs from the same causes and under the same conditions as in adult life. The pathologic changes are the same at all ages. Dry, fibrinous, pleurisy is probably as common in childhood as later, but usually causes less symptoms. When there is an effusion, it is more likely to be purulent in childhood. Primary pleurisy is possible in childhood, but probably very seldom occurs. Inflammation of the pleura is usually due to extension from some pathologic process in the lungs or pericardium, or the pleura is infected secondarily as the result of some pathologic process elsewhere.

Dry Pleurisy

This condition is never met in infancy and early childhood as an independent clinical entity, but is occasionally seen in late childhood under the same conditions as in adults. Pathologically, it is present in almost every case of lobar pneumonia and in many cases of bronchopneumonia. It probably accounts for the pain and painful cough in pneumonia. Nevertheless, there are almost never any physical signs to be found. Pericarditis is very likely to be complicated by pleuritis. If so, the symptoms of the pleuritis are overshadowed by those of the pericarditis. Dry pleurisy sometimes occurs in children in connection with inflammation of the throat, joints, heart or other infections, often spoken of as "rheumatic." It sometimes occurs in chorea. It undoubtedly is present in connection with tuberculosis of the lungs, especially in older children. It seldom causes any symptoms in such cases, but may cause pain and an increase in the temperature.

Symptomatology.—The chief symptoms of dry pleurisy are pain in respiration, especially on deep breath, and a short, dry cough. There are no other symptoms, when it is a complication of pneumonia and other acute diseases of the lungs. It may possibly increase the temperature and other general symptoms in tuberculosis.

Physical Signs.—There are almost never any physical signs of dry pleurisy in infancy, seldom in early childhood, more often in late childhood. They are lacking, of course, at all ages if the pleuritic inflammation is over the diaphragm or behind the heart. There is sometimes tenderness and pressure over the inflamed area. There is diminution of motion on the affected side. When a friction rub is heard, it is usually soft, seldom leathery or rough. A friction rub is sometimes increased or brought out by pressure of the stethescope. It is often difficult to distinguish between râles and a friction rub. A friction rub is localized, sounds close to the ear and may be heard in both inspiration and expiration. Râles are seldom sharply localized, sound farther away and are heard only in inspiration. The friction rub disappears with the healing of the process or with the formation of an effusion. There is never thickening enough of the pleura to cause dullness.

Prognosis.—Dry pleurisy does not alter the prognosis in any of the diseases which it complicates. It is, however, liable to result in an effusion, especially when it is a complication of an acute lung condition. Adhesions are usually formed, except when it is associated with pneumonia and bronchopneumonia.

Treatment.—The treatment of dry pleurisy, when it is a complication of pneumonia, has been already described. In other cases the treatment is simply for comfort. The child should be put to bed and kept quiet. Hot or cold should be applied externally, the choice depending on which gives the greater relief. A tight swathe sometimes, but rarely in my experience, helps. Strapping of the affected side may be tried. In severe cases it may be justifiable to use some preparation of opium. I do not believe that external applications have any affect whatever on the process in the pleura. This statement includes iodine in all its forms, whether colored or colorless, in the form of a liquid or of an ointment with a high sounding name. Neither do I believe that any drugs given internally, including the iodide of potash, have any affect on the course of the disease.

Pleurisy with Effusion

Physical Signs.—The physical signs of an effusion in the pleural cavity are the same whatever the character of the fluid, whether it be serous, sanguineous, seropurulent or purulent. The signs vary, of course, according to the amount of the effusion.

Inspection.—When the effusion is small, motion of the affected side is but little, if at all, limited, unless there is an associated dry pleurisy. As the amount of fluid increases, the motion gradually diminishes until, in large effusions, there is scarcely any. Small and moderate effusions do not cause any change in the shape of the affected side. In infants and young children larger effusions produce a general bulging of the affected side instead of a filling or bulging of the intercostal spaces, because, on account of the elasticity of the chest wall at this age, it yields as a whole before the intercostal spaces do. At this age the intercostal spaces bulge only when the effusion is very large. In middle and late childhood the intercostal spaces fill out before the chest as a whole gives way.

Percussion.—There is, of course, dullness or flatness, according to the thickness of the layer of fluid, over an effusion in the pleura. The location of the dull or flat area depends on the amount and location of the fluid. When the fluid is free in the pleural cavity, the upper border follows the lines described by Ellis & Garland, thirty or more years ago, for adults. The upper border of dullness, when the effusion is small, begins at the vertebral column, extends horizontally outward for a distance depending upon the amount of fluid, and then curves rather sharply downward to the base of the cavity. As the amount of the fluid increases, the horizontal upper border of dullness is higher, while the curve is more gradual and extends further anteriorly. When there is a moderate amount of fluid, the upper border of dullness curves upward from the vertebral column, reaches its highest point in the axilla, and then curves rather sharply downward to reach the base of the cavity in front at about the nipple line. When there is a large amount of fluid, the upper border of dullness is approximately level and the same both in front and behind. This border can only be determined by very light percussion. Theoretically, the upper border of dullness should vary with change in the position of the child, if the effusion is not too small or too large. In some instances it does. In most instances, however, especially in small children, I have found this variation very hard to recognize and consequently of very little assistance in diagnosis. The lower border of dullness or flatness cannot, of course, be lower than the bottom of the pleural cavity. This in children is not far from the tenth intercostal space in the scapular line, the eighth rib in the mid-axillary line and, on the right, the sixth rib in the nipple line. Flatness below this line must necessarily be due to some other cause than fluid in the pleural cavity. I have, nevertheless, seen the diagnosis of pleural effusion made erroneously many times, either because physicians did not know where the lower border of the pleura was or because they had not taken the trouble to locate it. Unless the percussion is light, the tympanitic sound from the stomach and intestines may be mixed with, or even overpower, the dull sound from the pleura and cause confusion, not only on the left but sometimes also on the right.

When the lung above the fluid has lost its elasticity as the result of compression, it often gives out a tympanitic note on percussion. This is usually more marked at the apex in front than behind. If the lung is

entirely collapsed, it gives a flat note. If there is much fluid, the other side of the chest is, of course, hyperresonant, because of the compensatory emphysema on that side.

When the effusion is circumscribed by adhesions or encapsulated, the area of dullness or flatness may, of course, be located anywhere. When it is between the lobes, there is often a strip of dullness, at about the level of the fourth rib, in the axilla.

Displacement of Organs.—The heart is easily displaced by effusions in the pleural cavity, provided the fluid is free, or, if encapsulated, close to the heart. The displacement is greater when the effusion is on the left than when it is on the right side. Displacement of the heart is shown by a change in the position or absence of the cardiac impulse and by displacement outward of the border of cardiac dullness on the side opposite to the effusion. The intensity of the heart sounds is also greater on the side of the sternum opposite to the effusion. Displacement of the posterior mediastinum may occur with moderate sized and considerable effusions. This is shown by a triangular area of dullness on the side opposite the effusion. The base of the triangle is at the base of the lung, one side in the median line of the back, the apex in the median line at the level of the effusion. This triangular area of dullness is known as Grocco's sign. Displacement of the diaphragm occurs only when there is a considerable amount of fluid in the pleural cavity. This has to be very considerable to push down the liver on the right side. Displacement of the liver is shown by displacement of its lower border. It must not be forgotten in this connection that the lower border of the liver is normally below the costal border in infancy, and may be even as late as four or five years. Displacement of the diaphragm on the left is shown by dullness of flatness in the half-moon-shaped, or Traube's, space. This is that portion of the lower left part of the thorax which lies below the lung and between the liver and spleen. It is over the stomach and intestines and ordinarily yields a tympanitic note, because of air in these organs. If they do not contain air, the note will, of course, be dull or flat.

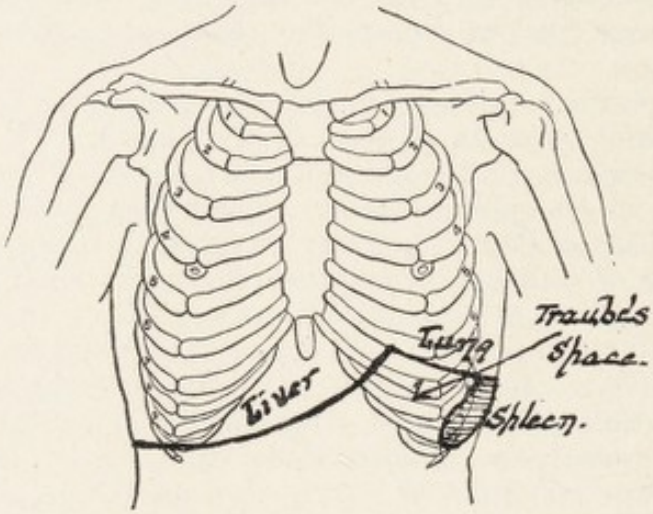

FIG. 116.—The half-moon-shaped, or Traube's, space.

Respiration.—The respiratory sound is diminished over the whole of the affected side, because of the diminished motility on that side. The diminution is greater over the layer of fluid, because of the interference of the fluid with the conduction of sound. In general, the greater the amount of fluid, the greater the diminution in the respiratory sound. When the amount of fluid is small, there is no change in the character of the sound. When it is large enough to compress the lung, the respiratory sound becomes bronchovesicular and, finally, when the lung is much compressed and solid, the respiratory sound, if it is transmitted, is bronchial The respiratory sound above the level of the fluid is vesicular, except that, if the lung is considerably compressed, it becomes broncho-

vesicular. In certain cases, presumably because of the elasticity of the chest wall in childhood, loud bronchial breathing may be heard over the effusion. This is especially likely to happen if the effusion is large. If the effusion is very large and the lung compressed, loud bronchial respiration may be heard over the whole side, both back and front. In other instances the exaggerated vesicular respiration from the normal side may be transmitted without change in character.

Voice Sounds.—The voice sounds vary in exactly the same way, both as to intensity and character, as does the respiration and for the same reasons. The character of the fluid makes no difference in the transmission of either. I have almost never heard the so-called "bleating sound"—aegophony—, which is so often referred to in the books, at the level of the upper border of the fluid.

FIG. 117.—Pleurisy with effusion. Patient flat, face down.

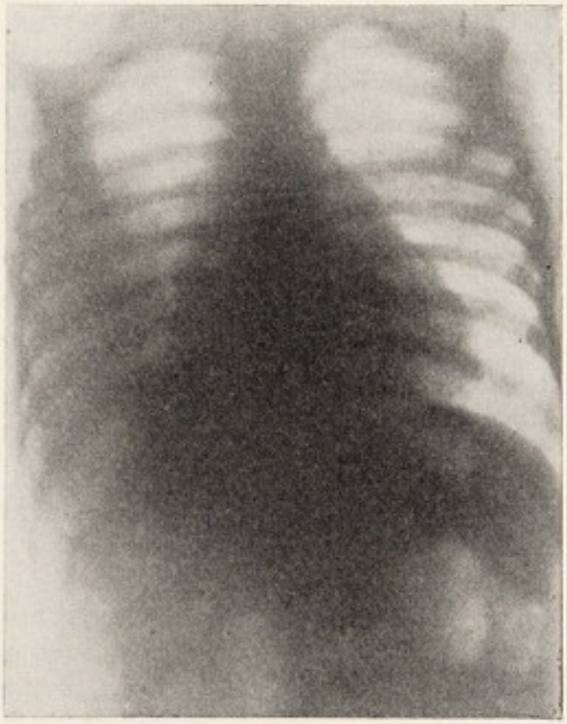

FIG. 118.—Pleurisy with effusion. Patient upright, back to plate.

Tactile Fremitus.—The tactile fremitus is diminished over an effusion, the amount of the diminution varying directly with the thickness of the layer of fluid. It is absent when the effusion is large. It is not changed above the level of the effusion, except that, when the lung is considerably compressed, it may be increased at the apex in front. It is often very difficult to determine the tactile fremitus, especially in infants. In them the cry has to be used instead of the spoken voice.

Sense of Resistance.—If there is more than a little fluid in the pleural cavity, the sense of resistance is increased, the increase varying directly with the amount of fluid. If there is a considerable amount of fluid, the sense of resistance is much more marked than it is over a solidified lung.

Râles and Friction Sounds.—Theoretically, râles should not be transmitted through fluid. Practically, in early life they sometimes are, even when there is a considerable amount of fluid. The presence of râles does not, therefore, absolutely exclude a pleural effusion. Friction sounds may sometimes be heard at the upper border of the fluid. They are

sometimes so moist that it is hard to distinguish them from râles. Their chief characteristics are that they are localized, close to the ear and may occur with both inspiration and expiration.

When the pleural surfaces are adherent in places, or the heart and pericardium fixed by adhesions, the physical signs are modified in various ways, differing in each case. Adhesions of the pleura prevent the free movement of the fluid and the uniform compression of the lung. They allow the transmission of the respiratory and voice sounds, as well as the tactile fremitus, to the surface. Adhesions about the heart and pericardium prevent the displacement of the heart and adhesions in the mediastinum may prevent the development of Grocco's sign.

Roentgenographic Signs.—When the effusion is free in the pleural cavity, there is a diffuse shadow on the affected side when the child is lying down. When it is sitting up, the shadow is, as a rule, not sharply defined and is higher in the axilla than elsewhere. It is comparatively seldom that the upper level of the fluid is distinctly horizontal. Displacement of the heart is evident and the motion of the diaphragm is limited. The Roentgen ray is of more assistance when the fluid is encapsulated, as it then shows definite localized shadows. It shows interlobar effusions, some of which cannot be detected in any other way.

Serous Pleurisy

There is probably a small amount of serum in the pleural cavity in many cases of pneumonia. In some instances, there may be a small amount of blood mixed with it. A large serous effusion very seldom occurs in pneumonia in early life. The younger the individual, the less common it is. In fact, it is one of the curiosities of medicine when it occurs in infancy. If there is a serous effusion, it almost always contains the pathogenic organisms causing the pneumonia and an excess of polynuclear neutrophilic leucocytes. It almost invariably becomes purulent in a few days, but in very rare instances may not and may be absorbed or not reaccumulate after tapping.

Serous pleurisy in childhood, although often apparently primary, is almost invariably secondary to a tuberculous process in the lung. This may be very slight, however, so slight indeed as to be unrecognizable clinically. Tuberculous serous pleurisy is always preceded or accompanied by dry pleurisy.

Symptomatology.—The usual story is that a child, supposedly well, or perhaps a little delicate, begins to have a dry cough or a slight pain in the chest. It seems a little feverish at night and is less active than usual. The pain in the chest ceases after a few days, but the cough continues. The fever becomes a little more marked and the child finally stops playing and wants to keep still or, perhaps, to go to bed. Coincidentally, it loses its appetite and begins to go down hill, losing both weight and color. In some instances pain at the beginning is a more prominent symptom; in others, it is very slight. The cough also varies a great deal. In some cases both cough and pain are almost entirely lacking and the only symptoms are moderate fever and those of malaise.

The leucocyte count in tuberculous serous pleurisy is at all ages either normal or diminished.

The effusion in serous pleurisy is an exudate. The specific gravity is, therefore, over 1.018. When the pleurisy is tuberculous, the effusion contains an excess of cells, which are predominantly mononuclear. When it is due to other organisms, the cells are mainly polynuclear. Tubercle

bacilli are rarely found in the fluid, unless it is previously digested. When the pleurisy is due to other organisms, they are usually very abundant.

Diagnosis.—When there are the physical signs of an effusion in the pleural cavity and a history like the above, it is almost certain that

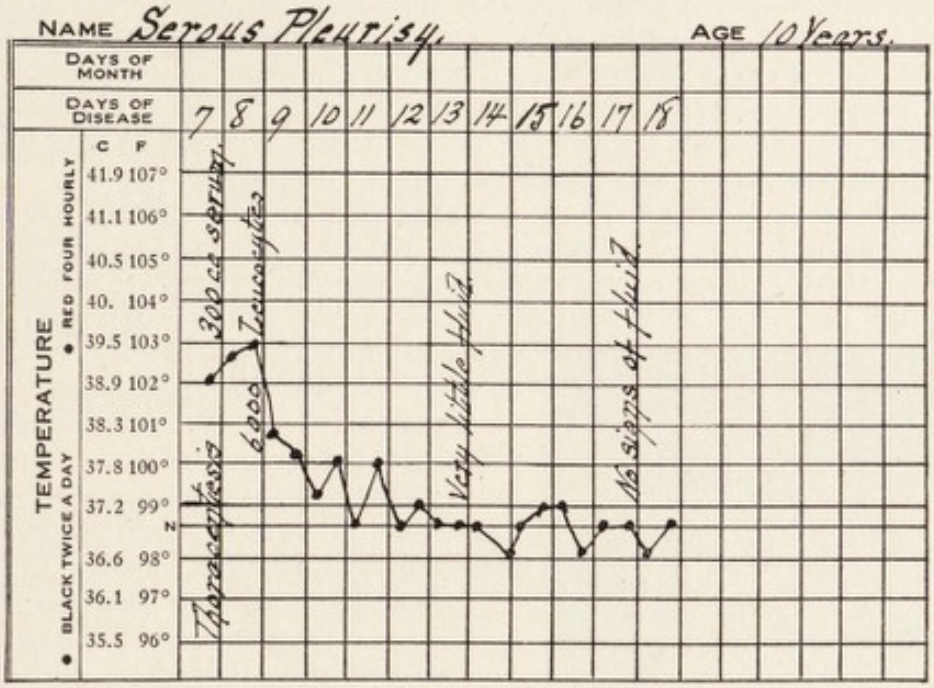

FIG. 119.—Tuberculous serous pleurisy.

the effusion is serous and tuberculous. When the effusion develops in the course of an acute pulmonary disease in childhood, the chances are very much that it is purulent. A low white count is very strong evidence in

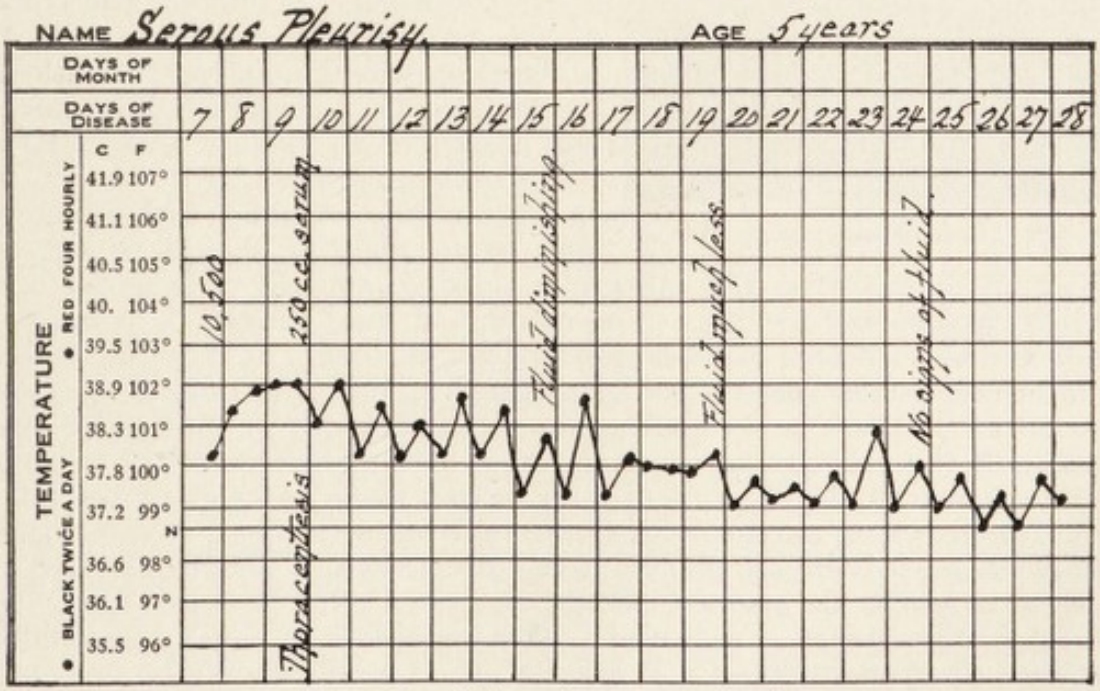

FIG. 120.—Tuberculous serous pleurisy.

favor of a serous and a high white count of a purulent effusion. A high, irregular temperature is in favor of a purulent effusion and a moderate temperature, not as irregular, of a serous effusion, but the effusion may

be purulent when the temperature is but little, or not at all, elevated and serous when it is high and irregular. The only way in which a positive diagnosis can be made, therefore, is by an exploratory puncture.

The effusion is also serous in hydrothorax. Hydrothorax is, however, almost always bilateral, while serous pleurisy is unilateral. Physical examination shows the signs of the lesion in some other organ which is responsible for the hydrothorax. These are absent in serous pleurisy. There is no rise in temperature with hydothorax, while it is usually elevated in serous pleurisy. There is no pain in the chest at the onset of hydrothorax, and the cough is due simply to pressure.

The effusion in hydrothorax is a transudate and its specific gravity is, therefore, below 1.018. It contains few cells, which are usually endothelial, and is sterile. In serous pleurisy, the specific gravity is above 1.018. There is an excess of cells and the fluid is not sterile, although in the tuberculous form it is often very difficult to demonstrate the organisms.

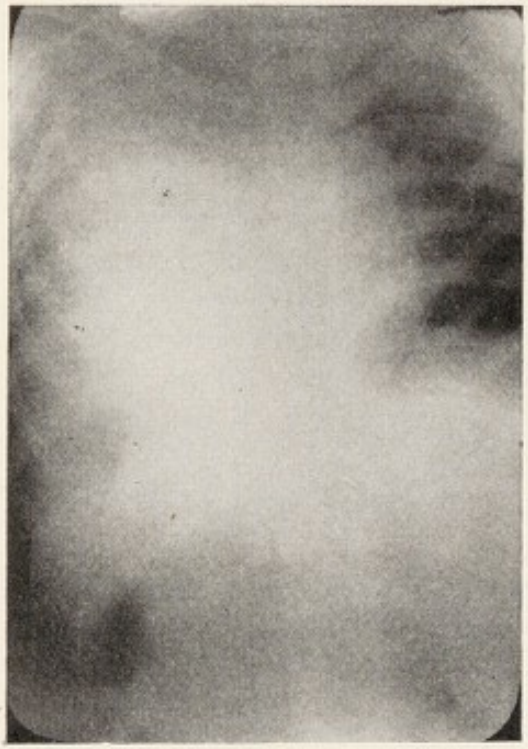

FIG. 121.—Large serous pleural effusion.

Prognosis.—If serous pleurisy develops in the course of an acute pulmonary disease, it almost always becomes purulent. If it is tuberculous in origin, it is in most instances absorbed. If it is not absorbed within a reasonable time and has to be removed, it usually does not return and, if it does, and has to removed several times, it is always finally absorbed. Pleural adhesions are usually formed, but they are less numerous than would be expected and most of them finally disappear. Recurrences are very unusual. Serous pleurisy is one of the least serious manifestations of tuberculosis in childhood and the outlook for recovery from the tuberculous infection elsewhere, to which it is secondary, is but little prejudiced by it.

Treatment.—A child with tuberculous serous pleurisy should be put to bed, kept quiet, given the maximum possible amount of fresh air and sunlight and forced feeding. The amount of fluid should be carefully noted and also whether it is increasing or diminishing. It is not advisable to interfere, unless there is sufficient pressure on other organs to cause discomfort or there is no diminution in the amount of fluid after two weeks. If the pressure on other organs is causing discomfort or there is no diminution in the amount of fluid in two weeks, enough of the fluid should be drawn off to relieve discomfort and allow the heart to come back into place, or, if there are no symptoms of discomfort, about one-third of it, as near as can be guessed, should be drawn off. It is less likely to return if only a part is removed. The remainder is usually gradually absorbed after one tapping. If it is not, the tapping should be repeated as often as necessary, following the same rule.

The patient should be kept in bed until the fluid has disappeared and the temperature is normal. The further treatment is that of tuberculosis in childhood in general.

Purulent Pleurisy. Empyema

Purulent pleurisy in early life is almost always a complication or sequela of lobar pneumonia or, occasionally, of bronchopneumonia. It is due to infection of the pleura with the same organisms which caused the pneumonia. It is said that when pneumonia is due to Type 1 pneumococci it is more likely to be complicated by empyema than when it is due to other forms. In some instances there is a mixed infection. Empyema is sometimes tuberculous and is then due to an extension of the tuberculous process from the lungs or spine. In rare instances it comes on very acutely in the course of measles, influenza or bronchopneumonia, or from some other cause. In such cases there are almost always small multiple abscesses scattered throughout the lung and usually some at the surface. The etiological organism in these cases is almost invariably some form of the streptococcus. This acute type sometimes appears to be primary, but, even so, it is probably always secondary to some small focus on the surface of the lung.

Metapneumonic Empyema

Symptomatology.—When purulent pleurisy is secondary to pneumonia, it seldom develops before the fifth or sixth day of the disease and usually not until a few days after the crisis has occurred. It may not, however, develop for a week or ten days. When it develops before the crisis, there is usually nothing in the symptomatology to call attention to it. The temperature, pulse and respiration may be a little higher and the child may seem sicker, but there are no pathognomonic symptoms to attract attention. When it develops after the crisis, the temperature rises again, as do the pulse and respiration, and the constitutional symptoms return. There is nothing characteristic about the temperature. It is likely to be high and irregular, but may be moderate and not very irregular or, if the pus has been present for some time, there may be no fever at all. The pulse and respiration vary with the temperature. The child shows the common manifestations of a collection of pus anywhere; pallor, loss of weight, anorexia, indigestion and albuminuria. Sweating is not at all common and chills are very unusual. The leucocyte count may increase a little, if the empyema develops in the course of pneumonia but not enough to be of any diagnostic value. If it develops after the crisis, the white count rises again.

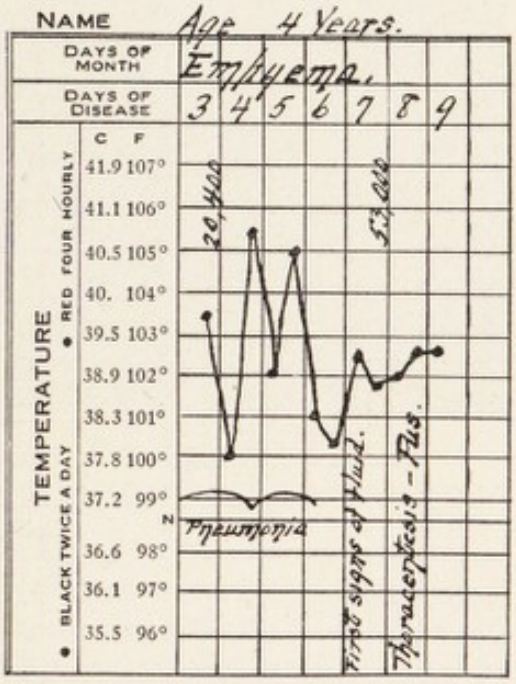

Fig. 122.—Empyema, developing during pneumonia.

The fluid is usually purulent from the first, but in the beginning may be serous or slightly turbid. It quickly becomes thicker, is often greenish, and almost always contains fibrinous masses, which are sometimes quite large. The specific gravity is high, much above 1.018. The cells are almost entirely polynuclear neutrophiles, but sometimes there are many eosinophiles. There are many organisms in the fluid, usually pneumo-

cocci, rarely streptococci, often a mixed infection. The organisms are usually less numerous in neglected cases which are seen late.

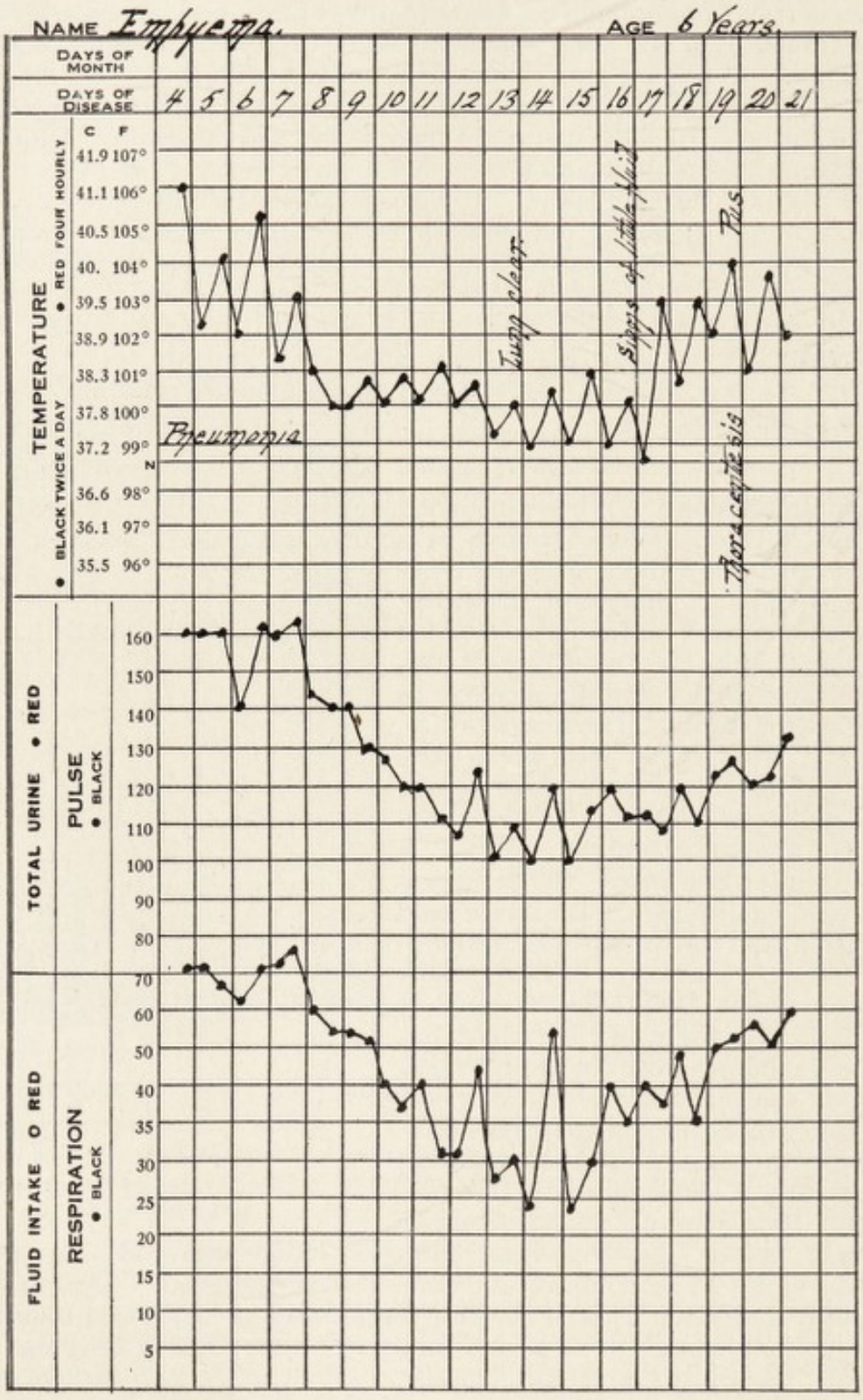

FIG. 123.—Empyema, developing after pneumonia.

Physical Signs.—These have been described under the head of pleurisy with effusion. It is important to remember that there is nothing about the physical signs of empyema which serves to distinguish it from

any other effusion. The character of the fluid can only be determined by exploratory puncture.

Diagnosis.—When the temperature fails to fall at the usual time in lobar pneumonia, remains somewhat elevated after a crisis, or goes up again after it has been normal for a time, empyema should be suspected. When it develops in the course of pneumonia there is usually nothing in the symptomatology to suggest it and it is suspected only because of some change in the physical signs. It must not be forgotten that other complications may keep up the temperature in pneumonia or cause it to rise again. The fever may be due to the development of another patch of pneumonia somewhere else in the lung, to otitis media, or to some involvement of the heart or kidneys.

If there is an effusion, it is almost always purulent rather than serous. Even if it is serous in the beginning it is almost certain to become purulent later. If it is turbid in the beginning it is sure to.

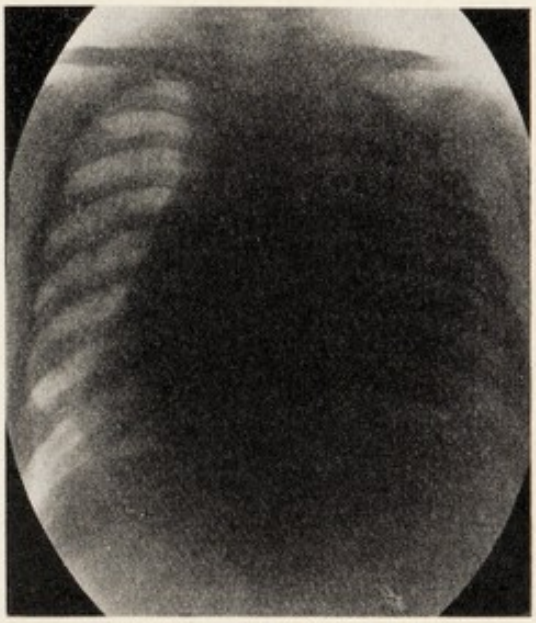

FIG. 124.—Empyema, right.

The most difficult thing to determine, when the temperature fails to come down as soon as it should or rises again, is whether the abnormal signs in the chest are due to solidification of the lung, a collection of fluid, or a combination of the two. There is little or nothing in the history or symptomatology which helps much in the diagnosis. The white count is useless, as it may be, and usually is, high in both conditions.

The diagnosis must be made entirely on the physical signs. Diminution or absence of motility on the affected side is common to both conditions. Enlargement of the affected side and fullness or bulging of the intercostal spaces are in favor of fluid. Displacement of organs, as shown by the position of the heart, Grocco's sign, extension of the liver dullness below the normal and dullness in Traube's space, is almost, but not quite, absolute proof of fluid. It is not absolute, because the heart and mediastinum may have been displaced from other causes in the past and dullness in Traube's space may be due to a full stomach and intestines. The absence of displacement of organs is strong evidence against the presence of any large amount of fluid, but not positive, because the heart and mediastinum may be bound down by adhesions from some previous infection. When the upper border of dullness or flatness corresponds to the border of a lobe, it points to solidification rather than to fluid. When the border does not follow a lobe, but corresponds to the lines described for fluid, it is evidence in favor of an effusion. If the upper border of dullness or flatness changes with change in position, it is very strong evidence in favor of fluid. Tympany in front above the dull area is somewhat in favor of fluid. If the sense of resistance is moderate, it is probably due to solidification of the lung; if it is very marked, there is probably fluid. Absence of the respiratory sound is in favor of fluid, as is also a diminution in it. The respiratory sound is, however, always somewhat diminished in pneumonia, and may be considerably, if the

bronchi are plugged. The presence of the respiratory sound, even if it is loud, does not exclude fluid, because in children it is often transmitted through fluid. The bronchial character of the respiration is not of importance, as it is heard not only over solid lung but may also be transmitted from a compressed lung through fluid. Loud bronchial respiration, especially if it is heard over most of the affected side, not only does not exclude fluid but really suggests it. The importance of the intensity and character of the voice sounds is the same as that of the respiration. The tactile fremitus is of little use. Its absence is in favor of fluid, but its presence does not exclude it. Râles are in favor of solidification of the lung, but may sometimes be heard through fluid. The most important points, therefore, in the diagnosis between solidification of the lung and an effusion are the position of the organs and the sense of resistance.

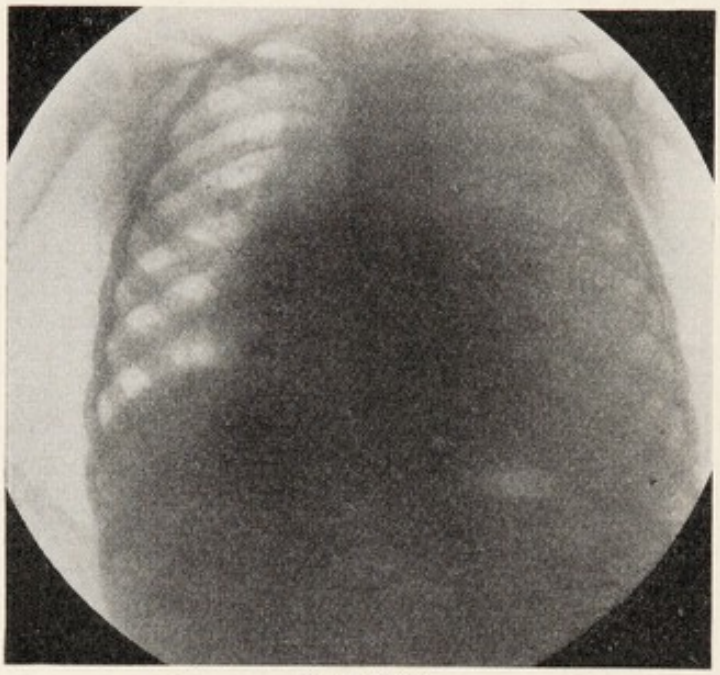

FIG. 125.

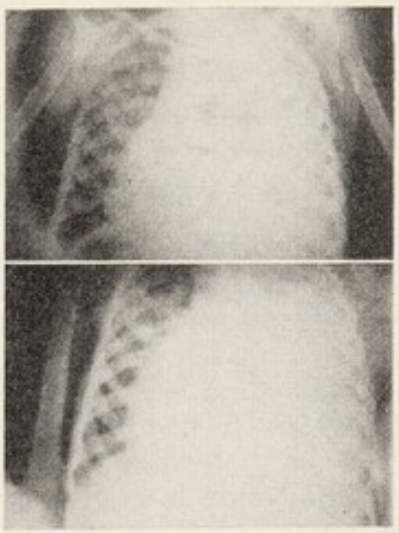

FIG. 126.

FIG. 125.—Empyema, left.

FIG. 126.—Pathologic process in lung. Not a pleural effusion, because no change in picture with change in position. Upper picture with patient prone, lower with patient upright.

Even when the signs are very strongly in favor of fluid, it is often impossible to know whether the lung behind it is solidified or simply compressed.

The Roentgen ray is of a certain amount of assistance in the diagnosis, but of not as much as might be expected, except when the effusion is localized or when it is between the lobes. It is possible with the Roentgen ray to recognize interlobar empyemas when the diagnosis cannot be made in other ways.

If the physical signs seem to show that there is fluid in the chest or if they suggest that there is a reasonable probability that there is an effusion, exploratory puncture should be done. If there is probably a considerable amount of pus in the pleural cavity, one of the best places to introduce the needle is in the seventh or eighth space in the scapular line, because this is an easy place to get at and the intercostal spaces are wide in this region. If the fluid is apparently localized, the needle should be introduced in the place where the signs are most marked, wherever this may be, even if it is high up in front under the clavicle. I prefer

a needle to a trocar. A large needle should be used, because, if the pus is thick, it will sometimes not run through a small needle. I have a number of times known empyema to be excluded because too small a needle was used. It is not advisable to use any local anesthetic, because the child is usually as much hurt and frightened by the anesthetic as it is by the prick of the needle. Judgment must be used as to how far the needle should be introduced. It must be put in far enough to get through whatever fibrin may be on the parietal surface of the pleura and not far enough to go into the lung. I have several times seen an empyema missed because the needle was put through the fluid into the lung. It is advisable to have the needle attached to a syringe when it is introduced, rather than to use an aspirator.

There is really no occasion for making the differential diagnosis between tuberculosis of the lung following pneumonia and empyema, because the condition is almost invariably empyema, not tuberculosis. If such a differentiation is to be made, however, it is simply between solidification of the lung and fluid.

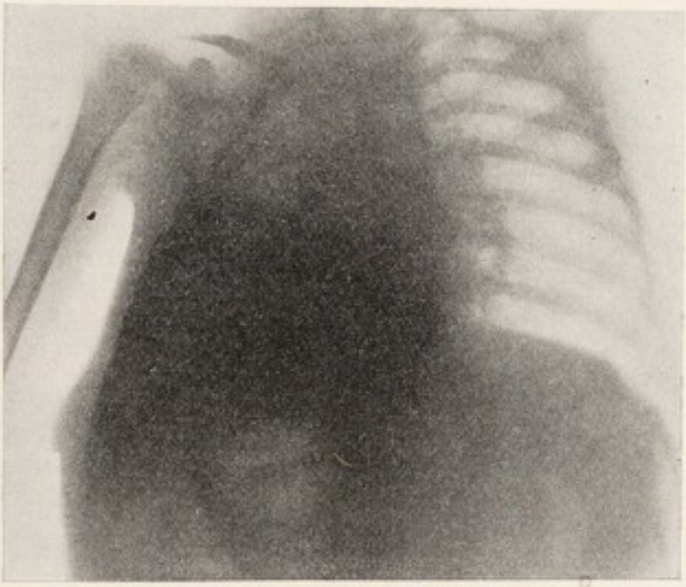

FIG. 127.—Deformity of chest from neglected empyema.

Prognosis.—If an empyema is neglected and left to nature, there is very little hope of recovery. The pus may break through into the lung, form an abscess, and finally break through into a bronchus and be coughed up. In rare instances recovery may eventually take place. It may also sometimes break through externally—empyema necessitans—and be discharged with, in rare instances, eventual recovery. Such a thing should never be allowed to happen, however, as the physical signsof fluid must have been very obvious for a long time previously. In other rare instances, the fluid may be eventually partially absorbed. Adhesions are formed, binding down the lung and contracting the pleural cavity, which result in permanent deformity of the chest, impairment of the expansion of the lung and displacement of the heart. Death, however, usually results from sepsis long before any of these things can happen.

If properly treated, most cases of empyema in childhood recover, usually without deformity or permanent damage. In infancy, however, especially in babies under a year, the outlook is much more serious, because at this age empyema usually shows a greatly diminished resistance to infection, which is not limited to the pleura. These babies, as a

rule, do not die of the empyema but of further and repeated infection of the lungs.

Treatment.—The palliative treatment of empyema is useless. The only thing which does any good is the removal of the fluid and drainage of the pleural cavity. When an empyema develops during the course of pneumonia, it is usually advisable to delay operation until after the time at which the crisis occurs or should occur. If the amount of fluid is so great as to cause symptoms from compression or from displacement of the heart, it may be removed by aspiration, thus relieving the pressure until operation can be performed. It is true that some cases recover after one or several aspirations. In most instances, however, they do not. When empyema develops after the crisis, operation should be performed as soon as the diagnosis is made. In my opinion, a rib should always be resected. The shock of the operation is not much greater than that of incision, drainage is much better and, if the periosteum is preserved, the rib is quickly reformed. The operation should be done in the place which will give the most efficient drainage, that is low down, either in the back or axilla, preferably in the axilla. It is usually inadvisable to introduce any liquids into the pleural cavity. The only reason for doing so is simply to mechanically wash out the cavity. Only unirritating solutions should, therefore, be used.

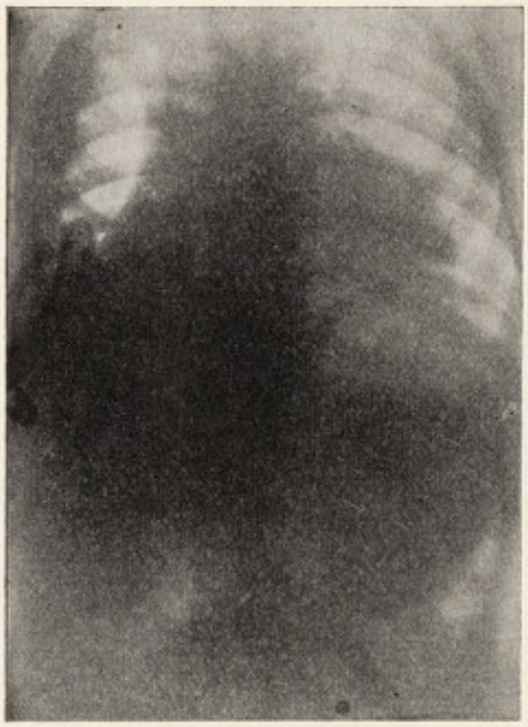

FIG. 128.—Empyema, with drainage tube in place.

About once in five years some new method of continuous drainage or aspiration of the pleural cavity, without resection of the rib or a long incision, is introduced, or some old method is revived and is greeted with enthusiasm. After a year or two it is no longer used and is forgotten and ribs are resected again as usual until the next rediscovery is made, to be again forgotten.

If empyemas are recognized and properly treated at the proper time, the lung almost invariably expands properly, no deformities of the chest result and there is no call for serious operations, such as the Estlander, for the removal of portions of the chest wall. Expansion of the lung during convalescence is helped somewhat by blowing trumpets or horns, or liquids from one bottle into another.

ACUTE STREPTOCOCCUS EMPYEMA

This condition, which was practically unknown to the present generation of physicians before the World War, is very uncommon in early life. When it occurs, it is usually secondary in the course of measles, influenza or bronchopneumonia, especially of influenza. It may come on so early in the course of one of these diseases that clinically it is apparently primary. In any event, it promptly becomes the most important condition and obscures the symptoms of the original disease. In such cases, there are usually multiple abscesses scattered throughout the lung, almost

always as the result of a streptococcus infection. The infection of the pleura is due to the rupture of one of these abscesses on the surface of the lung into the pleural cavity or to the direct extension of the inflammatory process to the pleura. The causative organism is, therefore, almost always the streptococcus.

Symptomatology.—The onset is always very acute, the temperature high, the pulse and respiration very rapid and the patient manifestly very ill. The symptoms are those common to all very severe septic infections, to which are added those of an effusion in the pleural cavity with pressure on other organs. The fluid develops very rapidly. It is a thin pus, of specific gravity well above 1.018, contains little fibrin, is full of polynuclear neutrophilic leucocytes, and loaded with bacteria. The physical signs are those of any effusion. Symptoms of respiratory discomfort and of pressure on other organs develop quickly. These are usually quite severe, because the fluid forms so rapidly that there is not time for them to accommodate themselves to the increased pressure.

Prognosis and Treatment.—The outlook is very grave in all of these cases, partly on account of the condition itself and partly because of the primary disease. Death is likely to occur in a few days. If it does not, there is a reasonable chance for recovery. The longer the patient lives, the better are the chances of ultimate recovery. Death is almost certain to follow quickly, if any radical operation, such as an incision or the resection of a rib, is done. The fluid should be simply drawn off, the aspiration being repeated as often as necessary to relieve the symptoms of pressure. If the patient survives the acute stage of the disease, a radical operation should then be performed. The rest of the treatment is that of any severe septic infection and is described elsewhere.

PNEUMOTHORAX

HYDROPNEUMOTHORAX PYOPNEUMOTHORAX

Etiology.—Pneumothorax in early life is more often due to perforation of the lung with an aspirating needle or trocar than to all other causes combined. It may be due, of course, as in adults, to a perforating wound of the chest wall or the fracture of a rib. It is next most often due to rupture of the lung, either from the inside or the outside. Rupture from the inside may be the result of strain or due to the breaking of an abscess or of a caseous tuberculous focus. The rupture of a caseous tuberculous focus, by far the most common cause of pneumothorax in adult life, is seldom the cause in childhood. Rupture of the lung from the outside is almost invariably due to a neglected empyema.

Infection of the pleura is very likely to occur with the formation of an effusion, which may be either serous or purulent, far more often purulent. The condition is then known as hydropneumothorax or pyopneumothorax.

Symptomatology.—When the pneumothorax is due to a perforating wound of the chest wall or to puncture of the lung with an aspirating needle, the onset is, of course, sudden. When it is due to other causes, the onset is usually sudden, sometimes during exertion, sometimes without it. It may even come on during sleep. The onset is usually accompanied by acute pain, which is quickly followed by marked dyspnea, associated with cyanosis. The pulse becomes rapid and feeble and the respiration rapid and short. In many cases there is marked collapse. In some instances, however, the symptoms may develop slowly or insidiously and the true condition be discovered by accident.

The further symptoms are simply those due to embarrassment of the respiration and to the displacement of the heart. If infection of the pleura takes place, the constitutional symptoms of empyema are added.

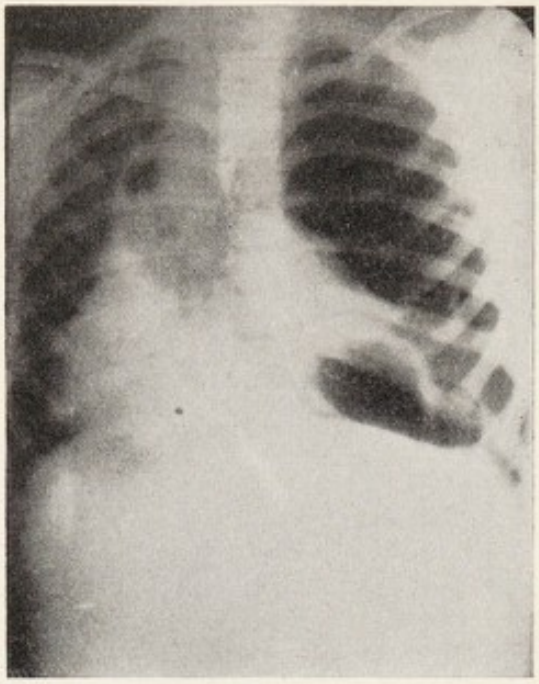

FIG. 129.—Pneumothorax. Dec. 21, 1918. Lung completely compressed.

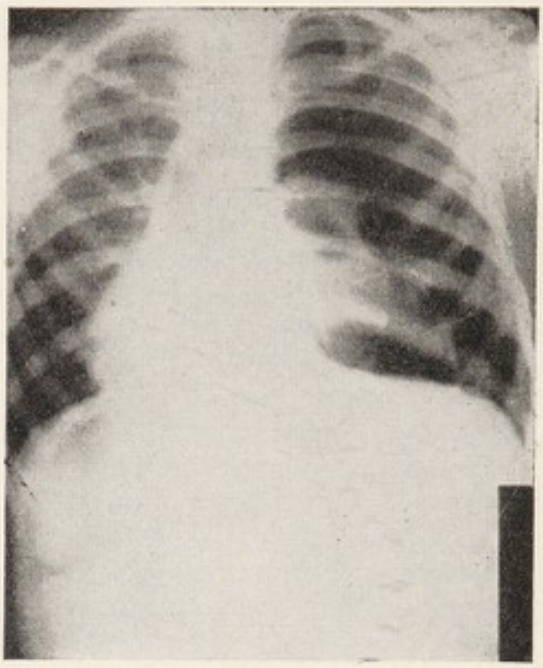

FIG. 130.—Pneumothorax. Jan. 2, 1919. Lung partially expanded.

Physical Signs.—There are three kinds of pneumothorax, the open, closed and valvular. In open pneumothorax there is, of course, connection with the outside air, either through the chest wall or through a bronchus. In the closed there is no such connection. The valvular in a short time becomes closed, because the accumulation of air in the pleural cavity shuts the valve.

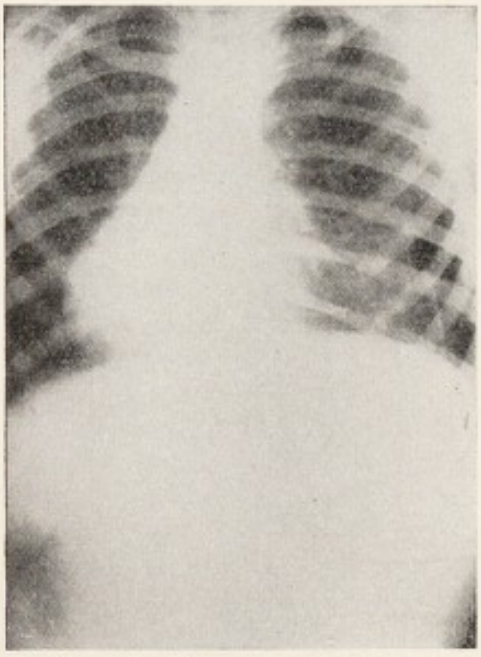

FIG. 131.—Pneumothorax. Jan. 21, 1919. Lung about three-fifths expanded.

If the pneumothorax is open, there is but little enlargement of the affected side. If it is closed or valvular, the affected side is usually considerably enlarged. Motion of the side is, of course, either much diminished or absent.

There is considerable displacement of organs in all cases of pneumothorax. In open pneumothorax this is due to the removal of the negative pressure which helps to hold the organs in position. The lung collapses, partly because of its own elasticity and partly because of the removal of the negative pressure. In closed pneumothorax, especially if it was valvular in the beginning, the pressure is above that of the atmosphere and the organs are still further displaced and the lung more compressed. Displacement of the heart is shown by displacement or absence of the impulse and moving of the dullness of the opposite border further outward. Displacement

of the posterior mediastinum is shown by tympany or exaggerated vesicular resonance on the opposite side of the median line. Displacement of the liver is shown by displacement downward of both the upper and lower borders of dullness. Displacement of the diaphragm on the left is difficult to determine because of the normal tympany in Traube's space.

The percussion note is usually tympanitic when the pneumothorax is open. When it is closed or valvular, it may be either tympanitic or exaggerated vesicular, according to the tension of the walls.

In closed and valvular pneumothorax, if the valve is closed, the respiratory sound is, of course, absent, except that sometimes a slight bronchial sound may be transmitted from the compressed lung, or a slightly vesicular sound from the other side. In open pneumothorax, when the opening is into a bronchus, the respiratory sound is loud and bronchial. This sound may be modified to cavernous or amphoric, according to the characteristics and tension of the walls of the cavity. The voice sounds vary in the same way as those of the respiration.

The tactile fremitus is always diminished. It cannot be transmitted, of course, if the pneumothorax is closed. Even when it is open it is much diminished by the obstacle to conduction furnished by the air in the pleura.

There are usually no râles. If they are transmitted from the lung, they are usually high pitched.

The physical signs vary, of course, according to whether the lung is entirely compressed or only partially. If it is only partially compressed, the signs of pneumothorax are more or less modified. A sign which is not as well known now as it was in the past, and as it should be, is the coin sound. When a silver coin is placed on the chest wall and percussed with another coin, the sound heard over the rest of the chest is a dull metallic sound. When this sound is transmitted through an air cavity, as in pneumothorax, it is clear and ringing like a bell. This clear, ringing sound is what is known as the coin sound.

It is usually possible to make a positive diagnosis of pneumothorax without the aid of the Roentgen ray. It is, however, of considerable assistance in corroborating the diagnosis made on the history and physical signs. The space occupied by the air is, of course, clear and surrounds the lung, which is more or less compressed towards its root. When there is fluid as well as air in the pleural cavity, the line between the fluid and air is very distinct when the patient is sitting up, while it disappears entirely when the patient is lying down. The Roentgen ray is also of great assistance in determining how rapidly the lung is expanding during convalescence.

Prognosis.—When pneumothorax is due to puncture of the lung with an aspirating needle, the prognosis is very good. In most instances no fluid is formed and the air is absorbed within a few days or weeks. An effusion is more likely to occur, if the injury is due to a fractured rib or to a perforating wound of the chest wall. The chances of recovery are, however, very good. If a serous effusion does form, it does not prejudice the prognosis a great deal. When the effusion is purulent and the pneumothorax closed, the prognosis is much the same as in empyema after the pleura is opened. If the pneumothorax is open, the prognosis is much the same as in an abscess of the lung or a neglected empyema which has broken through.

Treatment.—If the onset of the pneumothorax is very rapid and there is much displacement of the heart and mediastinum, it may be necessary

to draw off the air at once. This usually, however, does no good, as the air immediately reaccumulates. If it does, the chest should be opened and a tube inserted. If there is not much displacement of the heart and mediastinum and the patient is not very uncomfortable, it is much wiser to let the patient alone rather than to draw off the air. In most instances the air is finally absorbed. When there is a serous effusion, it should be drawn off, if it is necessary to relieve pressure. It is not advisable to draw off the fluid, unless it is necessary, because of the possibility of converting a closed pneumothorax into an open one and thus increasing the chances of pyopneumothorax. If the effusion is purulent, the chest should be opened and drained.

When the pneumothorax is closed and there is no effusion in the pleural cavity, the expansion of the lung can sometimes be hastened by blowing trumpets or horns or blowing the fluid from one bottle into another.

SECTION XI

DISEASES OF THE HEART AND PERICARDIUM

THE DEVELOPMENT OF THE HEART

After the earliest stages of development the heart is in the form of a tube which, at the upper end, tapers away into the truncus arteriosus, and which at its lower extremity receives the vitteline veins. As the result of excessive growth in length the heart tube is bent upon itself, assuming the form of the letter S placed obliquely. This change in direction is followed by rotation around a longitudinal axis so that the arterial portion is in front and the venous portion behind on nearly the same plane. While these alterations in position are taking place, the arterial part of the heart is being marked off from the venous portion by a transverse constriction, the arterial portion becoming the ventricle and the venous, the auricle, the constricted portion forming the auriculo-ventricular canal, which soon acquires valves. The truncus arteriosus is then marked off from the ventricle by a circular constriction, the proximal portion of the truncus arteriosus dilating and forming the bulbus arteriosus and the truncus arteriosus dividing into the visceral-arch vessels. About the fourth week the single auricle begins to be divided by the growth of a perpendicular ridge from its dorsal and cephalic walls into the right and left auricles. The auriculo-ventricular canal is also divided into the right and left auriculo-ventricular orifices. The separation of the auricles is not complete, however, as an aperture, the foramen ovale, remains. The division of the ventricle follows shortly after that of the auricle and is complete by the seventh week. The closure in this case is from below upward. The truncus arteriosus is also divided by the growth of a vertical septum into the aorta and pulmonary artery. The semilunar valves of the aorta and pulmonary artery appear when the truncus arteriosus divides to form these vessels. The truncus arteriosus is provided with a valve having four leaflets. When it divides into the pulmonary artery and aorta, the lateral leaflets are bisected, the anterior half of each going to the anterior vessel, or the pulmonary artery, and the posterior halves with the dorsal leaflet going to the aorta. At this time, however, there is also a rotation of the heart from right to left which alters the relation of the pulmonary artery and the aorta to the right and left ventricles, thus altering also the relation of the pulmonary and aortic leaflets.

As the result of the atrophy of some and the persistence of others of the visceral-arch vessels, the aorta and its branches are formed. The left fifth visceral-arch vessel is represented in the adult by the pulmonary artery. This vessel is connected with the aorta by a communicating trunk, the ductus arteriosus.

Consideration of the numerous changes which take place in the development of the heart and arterial trunks makes it easy to see how varied the congenital malformations may be, and how easily they may develop.

THE FETAL CIRCULATION

After the completion of the development of the heart and arterial system already described, the circulation of the blood is as follows: The

oxygenated blood passes from the placenta through the umbilical vein to the under surface of the liver. Part of it is carried directly to the liver through branches of the umbilical vein and thence to the inferior vena cava by the hepatic veins. At the transverse fissure the umbilical vein divides into two branches through one of which the blood is carried to the portal vein and through the other, known as the ductus venosus, to the inferior vena cava. The blood then passes from the inferior vena cava into the right auricle. It is then deflected by the Eustachian valve so that it passes through the foramen ovale into the left auricle. From the left auricle it passes through the left auriculo-ventricular orifice to the left ventricle and from the left ventricle into the aorta. The greater part of the blood is then distributed by the vessels of the arch of the aorta to the head and upper extremities, a small part passing down the aorta. The blood distributed to the head and upper extremities returns through the veins to the superior vena cava and passes into the right auricle, whence it passes through the right auriculo-ventricular orifice into the right ventricle and into the pulmonary artery. A small part of the blood passes to the lungs and back through the pulmonary veins to the left auricle. The major part, however, passes through the ductus arteriosus to the descending aorta. The greater portion of the blood in the descending aorta is returned through the umbilical arteries to the placenta; a smaller portion is carred to the legs and returned through the veins to the inferior vena cava. It is evident that no part of the fetal blood, except that in the umbilical vein, is entirely pure, the venous and arterial blood always being more or less mixed. The liver, head and upper extremities receive a greater proportion of oxygenated blood than other portions of the body and, as the result, the head, arms and abdomen are relatively large in the new-born infant.

CHANGES IN THE CIRCULATION AFTER BIRTH

With the beginning of respiration and the detachment of the placenta at birth marked alterations in the circulation occur. The blood in the pulmonary artery ceases almost at once to pass through the ductus arteriosus and is carried to the lungs as in the adult. At the same time the flow of blood through the foramen ovale rapidly diminishes and soon ceases, and the adult form of cardiac circulation is established. The placental circulation through the umbilical vein, the ductus venosus and the umbilical arteries is also cut out. As there is no further use for these structures, they are obliterated in the course of a few days. The ductus venosus and the umbilical arteries and vein are obliterated in from 2 to 5 days and the ductus arteriosus in from 4 to 10 days. The foramen ovale is usually closed by the growth of a septum from below in from 7 to 10 days, but small oblique openings not infrequently persist for six months or more, while in some instances the opening is never completely closed. The function of the Eustachian valve ceases at birth, but its remains may persist indefinitely. The umbilical vein becomes the round ligament of the liver. The distal portions of the umbilical arteries form the anterior true ligaments of the bladder, while the proximal portions remain pervious and are known as the superior vesical arteries. The ductus venosus and ductus arteriosus persist as small fibrous cords.

SIZE AND GROWTH OF THE HEART

Because of the nearly equal work of the two sides of the heart during fetal life the ventricular walls are of nearly the same thickness at birth,

the relation of the walls of the right and left ventricles being approximately as 6 to 7. The weight of the left ventricle is to that of the right ventricle as 13 is to 10, the adult relation being as 26 to 10. After birth the work of the right ventricle is proportionately much less than that of the left, so that at 6 years the thickness of the walls and the energy are no greater than at birth. The thickness of the left ventricle has doubled during this time. The relative thickness of the ventricular walls at this time is about 2 to 1, this relation being retained throughout childhood. The pulmonary orifice is larger than the aortic, especially between 6 and 10 years, there being a difference of 3 or 4 mm. throughout childhood. This difference in size may explain the physiologic reduplication of the second sound in childhood.

The weight of the heart relatively to the body weight is greatest in the new-born, sinks rapidly in the second year, increases again in the seventh year, is smallest in the years before puberty and increases rapidly during puberty, then attaining the adult relation which is the same as in the second and third years. The relation of the weight of the heart to the body weight in the new-born is to that in the adult as 19 is to 15.

The volume of the heart compared with the body weight is greatest in the new-born. It falls rapidly in the first and second years, increasing again in the third year and remaining approximately stationary to the thirteenth year, when it drops for two years to the minimum, increasing again with puberty to the adult relation.

The growth of the heart in weight and volume thus corresponds in a general way to the growth in height and weight. It also corresponds to the increase in the surface area of the body.

The calibre of the arteries in early childhood is relatively large in comparison with the volume of the heart. With the approach of puberty, however, the relation rapidly approaches that of adult life. The diameter of the veins in relation to that of the arteries is, on the other hand, much smaller in childhood than in later life, while the capillary circulation is much more highly developed. All these peculiarities favor the nutrition and growth of the organism. They also explain the low blood pressure, rapid circulation and frequent pulse of infancy and childhood.

Blood Pressure in Infancy and Childhood.—The blood pressure is low in infancy and gradually increases with age. The influence of sex is unimportant. The general condition makes a great difference in infancy, the pressure being much lower in feeble and premature infants. It is lower when a baby is asleep than when it is awake, when it is quiet than when it is active or crying, and before than after feeding. It varies with position and exertion after infancy just as in adults. Approximate average figures in mm. of mercury for various ages are as follows:

TABLE XXVIII

BLOOD PRESSURE IN INFANCY AND CHILDHOOD

Age in years	Systolic pressure	Diastolic pressure	Pulse pressure
Under 2	90	65	25
3–5	92	65	27
6–9	94	67	27
10–11	98	67	31
12–15	99–105	70	30–35

Rapidity of the Circulation.—According to Vierordt the round of the circulation is made in twelve seconds in the new-born, in fifteen seconds at three years, eighteen seconds at fourteen years and twenty-two seconds in the adult.

Pulse Rate.—The rate of the pulse is very irregular in infancy, even under normal conditions. It increases rapidly and often very considerably as the result of exertion or excitement and in febrile diseases. The rhythm of the pulse is also very easily disturbed. Disturbance of the rate and rhythm occurs almost as easily in early childhood as in infancy. It is consequently impossible to give more than approximate figures as to the pulse rate in early life.

TABLE XXIX

PULSE RATE IN INFANCY AND CHILDHOOD

Early weeks	120–140
First year	110–120
Second year	100–110
2–5 years	90–100
5–10 years	80–90

Irregularity of the pulse and an increase in its rate are, therefore, of much less significance in infancy and early childhood than in later life. These facts must never be forgotten in estimating the prognostic importance of a frequent and irregular pulse in early life. The normal rate for the age must always be taken into consideration and the actual rate compared with that. If it is not, the physician, especially if he is in the habit of treating adults, is likely to attach too much importance to a frequent pulse in early life. He is also quite likely to be too much disturbed by an irregularity in the rhythm.

Sinus or Respiratory Arrhythmia.—Sinus or respiratory arrhythmia, that is, an increase in the rate of the pulse with inspiration and a decrease in the rate during expiration, is very common during childhood. It does not mean any disease of the heart whatever and is a perfectly normal condition.

Extra systoles are also not at all uncommon during childhood and have no pathologic significance, unless associated with other definite signs of organic cardiac disease. Dropped beats are also not at all uncommon during childhood and likewise have no pathologic significance.

Paroxysmal Tachycardia is most unusual in childhood. I have seen but three cases. It differs in no way from the same condition in adult life. An infrequent pulse from heart block is also very uncommon.

PHYSICAL EXAMINATION OF THE HEART

On account of the variation in the rate of the growth of the heart, the relative size and shape of the thorax and the size and rate of growth of other neighboring organs, the cardiac physical signs vary greatly at different periods of infancy and childhood. In infancy the comparatively large heart is placed more horizontally in a relatively narrow thorax and the lungs do not cover it as much as later. The physical signs are, therefore, materially different from those in later childhood when the heart is not relatively as large, the thorax is wider and the lungs extend farther forward. The infantile form of thorax is replaced by the adult type at about five years while the diaphragm reaches the adult position about a year later. The lungs are not fully expanded forward until six years. The thymus, which is placed directly over the heart in infancy, modifies the signs for at least three years. The physical signs, therefore,

gradually change from those found in the infant to those found in the adult, being different at each age.

Cardiac Impulse.—The cardiac impulse is seldom visible and often not palpable in early infancy. In childhood it is often more distinct than in adults. On account of the relatively large heart, narrow thorax and high position of the diaphragm, it is at first higher up and farther out than in later life. In infancy it is in the fourth space about one centimeter outside the nipple line and from five to six centimeters from the median line. As the heart becomes relatively smaller and the thorax wider, while the diaphragm descends, the impulse gradually moves downward and then inward, being in the fifth space in the nipple line at seven years and always inside the nipple line by the thirteenth year.

Cardiac Area.—The area of flatness, or absolute dullness, that is, that part of the heart not covered by the lungs, is relatively large during infancy and up to six years, when the lungs are fully expanded. On account of the small size of the parts, it is impossible to determine accurately the area of absolute dullness in infancy, even if the lightest possible percussion is used. If the attempt is made to do so, the area is almost always made too large. The right border of this area is at the left sternal border throughout the whole of childhood. Fortunately, it is practically of no importance to determine this area in early life.

FIG. 132.—Area of relative cardiac dullness in infancy.

It is comparatively easy, however, to determine the area of relative dullness, that is, the absolute size of the heart, which is the important one. The percussion stroke must be light, but not too light. If it is too light, the border of the heart, which is deep and covered by lung tissue, is not reached, while, if it is too strong, the percussion wave extends too far and obscures the sound. The pleximeter finger should be placed parallel to the border of the heart which is being percussed. If it is placed vertically, it is impossible to accurately mark out the border, because both lung and heart are covered by it. Percussion is best performed in infants while they are lying on the back, while in older children the position makes less difference. Comparisons must always be made, however, with the patient in the same position. During the first two years the dullness of the thymus may interfere with the determination of the upper border of the heart. It can, however, usually be made out with the aid of strong percussion.

In infancy the upper border of the relative cardiac dullness is at the lower border of the second costal cartilage or in the second space. The left border is one centimeter outside the left nipple line, or from five to six centimeters to the left of the median line, the right border at or just inside the right parasternal line, or two centimeters to the right of the median line. The greatest width of the heart, therefore, is eight centimeters.

The area of the relative cardiac dullness is so large when compared with that of the front of the chest in infancy that it hardly seems possible

that it can be correct and, therefore, unless a physician is in the habit of marking out and measuring the cardiac dullness, he is almost certain to believe that the heart is enlarged when it really is not.

At six years the upper border of relative dullness is at the upper border of the third rib, the left border just outside the left nipple line, or seven centimeters to the left of the median line, the right border two and one half centimeters to the right of the median line. The maximum width of the heart, therefore, is nine and one half centimeters.

At twelve years the upper border of the relative dullness is at the third rib, the left border one centimeter inside the left nipple line, or eight centimeters to the left of the median line, the right border three centimeters to the right of the median line. The maximum breadth of the heart, therefore, is eleven centimeters. When children are unusually

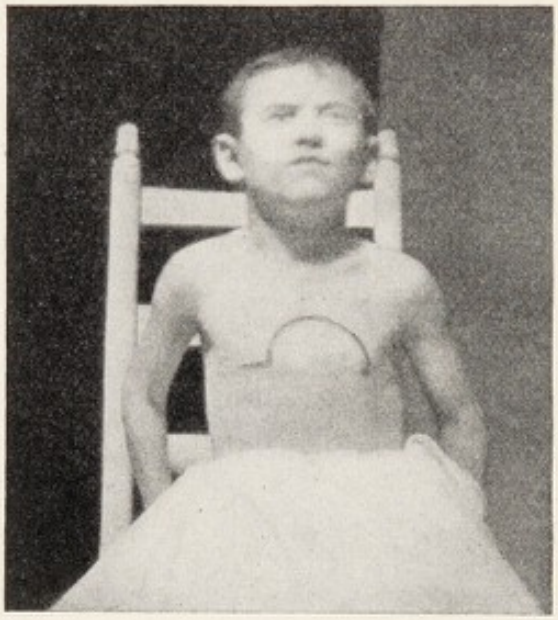

FIG. 133.—Area of relative cardiac dullness at six years.

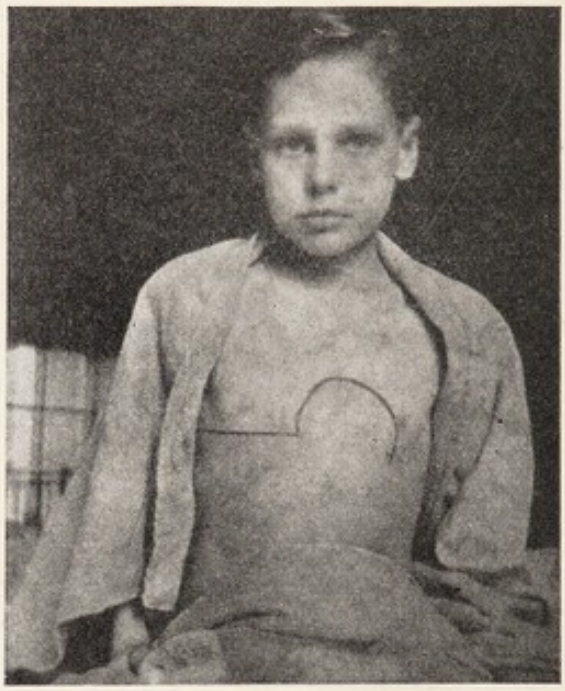

FIG. 134.—Area of relative cardiac dullness at twelve years.

large for their age the right and left borders of the heart may be a little farther out and the breadth of the heart a little greater than the average.

Heart Sounds.—The first sound in early infancy does not have the characteristic booming quality which it has in later life. It is much more like the second sound, so that the sounds often resemble the "tic-tac" heart of the fetus. The first sound acquires the normal booming character during the second year. The first sound at the apex, when compared with the first sound at the base, is relatively much louder in infancy and early childhood than in later life. The second pulmonic sound is louder than the second aortic sound throughout the whole of childhood. Great care must be taken, therefore, in making a diagnosis of an accentuation of the second pulmonic sound. Reduplication of the second sound is not uncommon under normal conditions. It is, therefore, not necessarily pathologic.

General Peculiarities in Early Life.—There are certain general peculiarities in the physical signs of cardiac disease in infancy and childhood. Prominence of the precordia occurs frequently as the result of the pressure of the enlarged heart on the unossified sternum and costal cartilages.

The earlier the onset and the longer the duration of the disease, the more marked is this deformity. Rickets also favors its development. Murmurs are often heard over large areas. Thrills are more common than in later life. Cardiac disease in childhood also interferes with the growth of other organs and causes a general retardation of development. On the other hand, however, because of the reparative powers of growth, the tendency to recovery is greater in early than in later life.

CONGENITAL HEART DISEASE

This term is really a misnomer, because the conditions which are described as congenital heart disease are really abnormalities or malformations of the heart and great vessels.

The causes of congenital heart disease may be divided into three classes: Persistence of fetal conditions, interference with normal development, and fetal endocarditis. The only important anomalies resulting from the persistence of fetal conditions are patency of the foramen ovale and ductus arteriosus. These may be the only lesions or may be the result of other cardiac lesions. The most common abnormalities resulting from interference with normal development are defects in the septa, more often in the ventricular than in the auricular, and stenosis or atresia of the orifices, most commonly of the pulmonary. Malformations of the great vessels are also not uncommon. Fetal endocarditis almost always occurs on the right side and usually affects the pulmonary valves.

Combinations of lesions, as the result of a combination of the causes just mentioned, are very common. Inflammatory lesions and anomalies which result in narrowing of the orifices, and hence in a local increase of intracardiac pressure, prevent the normal development of the heart, especially of the septa, which fail to close because of the necessity for a compensatory circulation. For the same reason, the foramen ovale and ductus arteriosus often continue open after birth.

Hypertrophy and dilatation of the heart, especially of the right side, also develop as the result of the lesions which cause obstruction, increased resistance and increased intracardiac pressure.

The most common lesions are, in order of frequency, defect in the ventricular septum, defect in the auricular septum (including open foramen ovale), pulmonary stenosis or atresia, patent ductus arteriosus, and abnormalities in the great vessels. The most commonly associated lesions are defects in one or both septa in connection with pulmonary stenosis.

Symptomatology.—Barring the cases of uncomplicated patency of the foramen ovale, in which there are almost never any symptoms, the symptoms of congenital heart disease are present at birth or develop during the first month in about 70%. In about half of the others, symptoms appear before the end of the first year, while in a small number they may not develop until after puberty. Infants may die suddenly before having shown any symptoms, or the symptoms may be so slight as not to attract attention until the infant begins to exert itself sufficiently, as in walking or running, to embarrass the circulation. In rare instances, patients live many years with gross defects and without any symptoms.

The most common symptom is cyanosis, which is present in about four fifths of the severe cases, and varies from a slight tinge to a dark leaden color. In many instances it is present only on exertion or during crying and coughing. It may be absent, even when there is some severe lesion. In the majority of cases with cyanosis, there is a lesion of the

pulmonary artery. This corroborates the commonly accepted view that the cyanosis is due to obstruction to the pulmonary circulation rather than to the admixture of venous and arterial blood. The presence of cyanosis in early infancy does not, however, necessarily mean congenital heart disease, as it may be due to a number of other conditions,

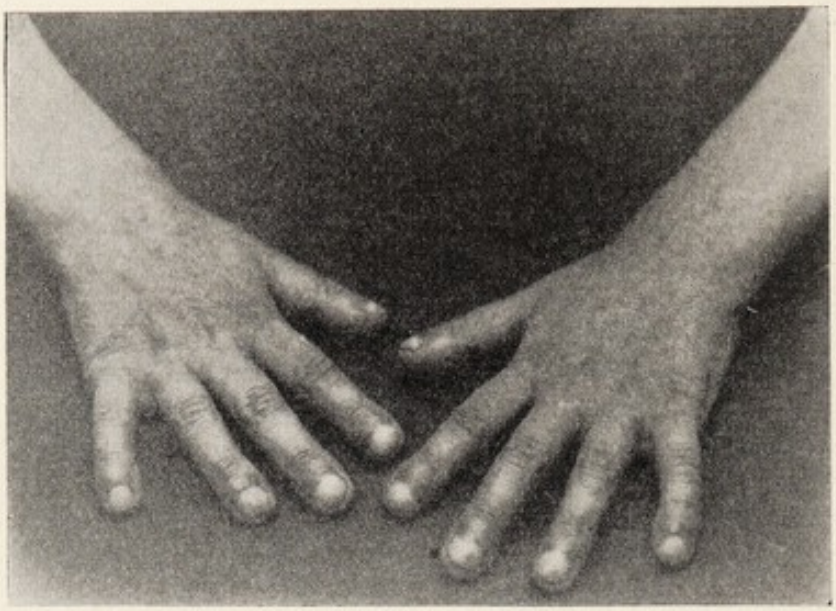

FIG. 135.—Clubbing of fingers in congenital heart disease.

such as an enlarged thymus gland, atelectasis of the lungs, cerebral hemorrhage or sepsis.

Clubbing of the fingers and toes is never congenital, but sometimes develops during the latter part of the first year. It is about as common

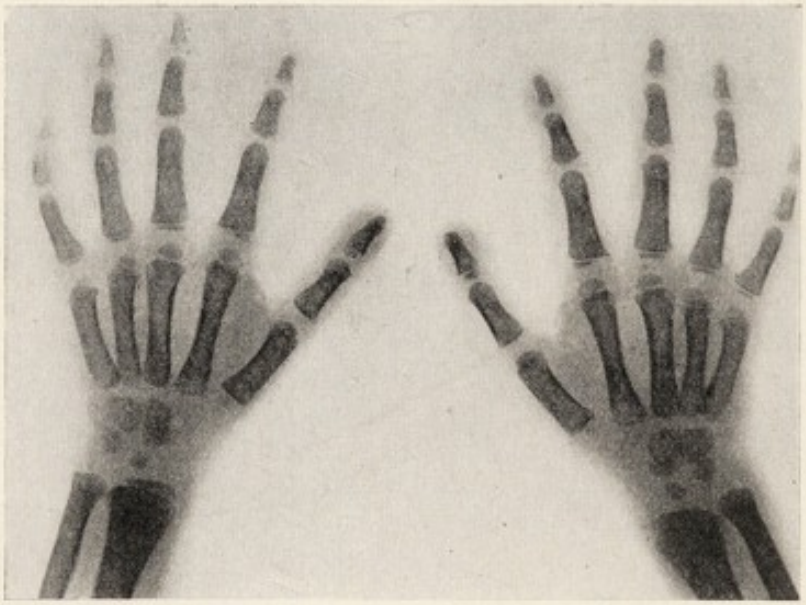

FIG. 136.—Roentgenogram of clubbed fingers in congenital heart disease, showing normal bones. Same hands as in Fig. 135.

as cyanosis in children who have reached the age of four or five years. It is usually due to the swelling of the soft parts alone, but in time the bones may also be involved (hypertrophic osteoarthropathy).

Dyspnea is a very constant symptom, although in many cases it is not noticeable when the child is at rest. As a rule, it varies directly with

the cyanosis. Peculiar attacks of suffocation are not uncommon in connection with cyanosis. They are usually started by coughing or crying and last from five to fifteen minutes. Consciousness is lost. Recovery is the rule, but death is not infrequent in these attacks. Convulsions are not uncommon in connection with these suffocative attacks and sometimes occur independently of them.

Bulging of the precordia usually develops early and becomes more marked as time goes on. Growth and nutrition are almost always affected and those who live usually present the typical picture of infantilism.

If the cases of patency of the foramen ovale, in which murmurs are often lacking, are omitted, murmurs are heard in at least nine tenths of all cases. These murmurs are almost invariably systolic in time, are often diffuse and not infrequently accompanied by a thrill. Their location and transmission does not, as a rule, correspond to those of acquired heart disease. Enlargement of the heart is, in my experience, often absent. If present, it is usually most marked to the right.

Diagnosis.—The diagnosis of congenital heart disease from acquired heart disease is usually not difficult. There are certain main points which are of importance in distinguishing between these two conditions. The mere fact that there are signs of cardiac disease in infancy and early childhood is strong evidence in favor of a congenital lesion. When there is no history of any disease in the past likely to cause cardiac disease, the evidence in favor of a congenital lesion is still stronger. Cyanosis and clubbing of the extremities without heart murmurs or other evidences of disease sufficient to account for them, and cyanosis and clubbing of the extremities without signs of venous congestion in other organs, count strongly in favor of a congenital against an acquired lesion. So also do loud murmurs without enlargement of the heart. Diastolic murmurs are practically never congenital. A combination of physical signs not consistent with those usually found in the various forms of acquired cardiac disease is strongly suggestive of a congenital lesion. The presence of other congenital abnormalities favors the diagnosis of congenital disease, and the presence of the characteristic signs of the various congenital lesions makes the diagnosis almost positive.

The diagnosis between the murmurs due to congenital heart lesions and cardio-pulmonary murmurs is not difficult. The cardio-pulmonary murmurs vary with position or respiration, and with them there is no enlargement of the heart or other symptoms of cardiac disease. Another murmur, which is frequently heard in infancy over or about the manubrium and which is probably due to pressure on the innominate veins, ought not to be confused with the murmur of a congenital lesion, because it is increased by extension of the head and is not associated with any other signs of cardiac disease. A systolic murmur is very often heard in the pulmonary area throughout childhood. The cause of this murmur is not certainly known. It is, however, soft, not associated with a thrill, enlargement of the right side of the heart, or weakening of the second pulmonic sound, one or more of which signs is almost always present when a murmur in the pulmonary area is due to a congenital lesion. Moreover, there are never any other evidences of cardiac disease, such as are usually present with a congenital lesion. The so-called functional murmurs, which may be heard at any of the orifices as the result of lowered muscular and nervous tone, are usually not present in infancy but occur later, when most of the the children with congenital lesions are

already dead. These murmurs are always soft and are not associated with other evidences of cardiac disease, unless possibly slight enlargement of the heart. Their distribution, moreover, is not that of congenital but of acquired lesions.

Cyanosis at birth always suggests a congenital cardiac lesion. If a murmur is heard over the heart, this is the case. If there is no murmur, it may be, but probably is not. The more probable explanation is atelectasis of the lungs or possibly a cerebral hemorrhage. When there is atelectasis, physical examination may or may not reveal it. The Roentgen ray will, however, show positively whether it is or is not present. If there is a cerebral hemorrhage sufficient to cause cyanosis, there will be other definite symptoms of the hemorrhage. The Roentgen ray seldom shows at this time, at any rate, any characteristic picture of a congenital lesion of the heart.

When the diagnosis of congenital heart disease is positive, it is nevertheless usually very difficult, if not impossible, to make a positive diagnosis of the exact abnormality, partly because several lesions may give the same signs, partly because of the frequency of combined lesions and partly because of the occasional presence of most unusual anomalies. Many of the points which are often emphasized in the diagnosis of special lesions are of no practical value. It is not reasonable, for example, to believe that when the ductus arteriosis is patent even if it is enlarged, it will be possible to percuss out a tube less than an inch long and not much larger than a slate pencil deep down in the neck under the manubrium. The Roentgen ray, which theoretically ought to be of considerable assistance in the diagnosis of special lesions, is practically of little assistance even in the hands of an expert.

Fortunately, the diagnosis of the exact lesion in these cases is not of great importance in either prognosis or treatment. The signs of certain lesions are, however, fairly constant and characteristic. A loud murmur, most marked at the center of the heart dullness, not accompanied by a thrill, and without enlargement of the heart, points toward a defect in the ventricular septum; a murmur loudest at the base, accompanied by a thrill at the base and without hypertrophy, points to a patent ductus arteriosus; a murmur loudest at the base, accompanied by a thrill and enlargement of the heart to the right, with a feeble second pulmonic sound and cyanosis, points to a narrowing of the pulmonary orifice.

Prognosis.—The prognosis depends to a certain extent on the nature of the lesion. Patency of the foramen ovale diminishes the expectancy of life but little, and simple defects of the ventricular septum are not incompatible with long life; neither are certain combined lesions in which the lesions to a certain extent neutralize each other. Infants with atresia of the pulmonary artery rarely live over two years, and those with atresia of the aortic orifice seldom over two or three days. The determination of the lesion is so difficult, however, that clinically little reliance can be placed on the diagnosis of the lesion in prognosis, this depending on the general condition and the character of the circulation in the individual patient rather than on special symptoms and signs. In general, about one third die in the first two months, and another one third in the first five years. A few reach adult life. Some infants with what are apparently characteristic symptoms and signs of congenital heart disease recover entirely and develop normally in every way. I have had several such cases. It is hard to reconcile these cases with what we know as to the pathology of congenital heart disease, in which

there is apparently no possibility of the repair of the lesions. It seems as if the diagnosis must be wrong in such cases. Other children with congenital lesions and loud murmurs show no symptoms. I have seen several infants, who are now young adults, in whom the murmurs, which are still audible over the whole chest, were present at birth and have never changed either in intensity or character. None of them have ever had a symptom referable to the heart Some of them have taken ether several times. All of them have been active and played all sorts of games. One of them has been a boxer and two others have taken the course of training in college for the officers' reserve training corps. It is not wise, therefore, when there are no evidences of a congenital lesion outside of a murmur, to give too serious a prognosis.

Treatment.—There is, of course, no curative treatment. There is nothing which will either diminish the deformities or favor the closure of abnormal openings. The treatment must, therefore, be hygienic and symptomatic. Breast feeding, maintenance of the body temperature, and the prevention of infection are of the most importance in the beginning. Affections of the respiratory tract are very dangerous and must be especially guarded against. A comparatively warm, dry climate is, therefore, most suitable. Overexertion and excitement must be avoided in older children. Insufficiency of the heart muscle calls for digitalis, as it does in acquired heart disease. Sudden severe symptoms are to be treated, as in acquired heart disease, by nitroglycerin or the quick cardiac stimulants, such as strychnia and ammonia. The sudden attacks of suffocation and the convulsions are best avoided by the use of bromide.

ACQUIRED HEART DISEASE

Acute Endocarditis

Acute endocarditis seldom, if ever, develops during the first three years of life. It is uncommon during the next two years, but is, unfortunately, very common after five years.

Etiology.—Acute endocarditis in childhood is for all practical purposes always secondary to some infection elsewhere. This infection is, in the vast majority of instances, acute. Infections of the tonsils are the most common cause, but acute endocarditis often develops in the course of infections of the pharynx, nasopharynx and their adnexa. The next most common cause is rheumatism. It must not be forgotten in this connection that the manifestations of rheumatism in childhood are materially different from those in adult life. Swelling of the joints is unusual and redness relatively uncommon. The temperature is seldom high or the constitutional symptoms marked. The patients not infrequently do not feel sick enough to go to bed. In many, or most, instances there is merely a little pain and stiffness in the joints, or even only fleeting pains in the extremities, with an elevation of temperature of only one or two degrees. The endocardium is involved just as often in these mild cases, however, as in the severe. In fact, it is apparently sometimes affected before the joints, thus justifying the statement that in childhood the endocardium acts like a synovial membrane. It is these cases of endocarditis coming on insidiously in mild cases of rheumatism which explain the numerous cases of apparently primary cardiac disease so often seen in later childhood and adult life. It is difficult to state how frequently rheumatism in childhood is complicated by endocarditis, various

authors estimating it at from 50% to 75%. In all probability, however, this proportion is much too high, as the many cases in which endocarditis does not develop are forgotten or overlooked, while, when endocarditis is present, careful questioning is very likely to bring out a history of pains of some sort in the past which is accepted as indicative of rheumatism.

Acute endocarditis is a common complication or sequela of chorea, in which it develops in perhaps one half of the cases. It is a curious fact that in some years endocarditis seldom develops in chorea, while in other years almost every child with chorea also has an infection of the heart. It is almost always found in fatal cases of chorea, in fact, there is no disease in which endocarditis is found so constantly at autopsy as chorea. Endocarditis is apparently no more common in those cases of chorea in which there is an infection of the tonsils or manifestations of rheumatism than in those in which there are not.

Acute endocarditis occasionally develops in the course of scarlet fever, but in such cases there is almost always an associated infection of the throat to which the inflammation of the endocardium is probably due. It may also be secondary to acute stomatitis or gingivitis. Occasionally it is apparently due to chronic suppurative processes in or about the teeth. It may develop in the course of chicken-pox or of those skin diseases in which there is suppuration. It occasionally complicates acute infections of the bone and joints. In childhood it is very seldom due to pneumonia or infections of the pleura.

The causative organism is in most instances some form of streptococcus, although any of the cocci, and occasionally other bacteria, may be the causative agent.

Pathology.—There is nothing characteristic about the pathologic lesions of endocarditis in early life. In childhood, however, acute endocarditis is almost always vegetative in type, the ulcerative form being extremely rare, so rare, indeed, that it is hardly necessary to take it into consideration. I can remember but two cases in my own experience.

After the acute stage is passed the vegetations may gradually disappear and the valve be restored to a normal condition, at any rate functionally. This is more likely to happen in early life, because the growth of the parts may correct any slight cicatricial changes which may have occurred. On the other hand, although the vegetations may become organized and disappear, sclerotic changes are usually set up which result in permanent deformity of the valves and orifice. The mitral orifice is affected at least ten times as often as the aortic and, if the aortic is involved, it is almost always in connection with the mitral. Unfortunately, repeated infections are likely to occur, each one of which increases the pathologic changes.

Although acute endocarditis is usually described as an individual entity, it is very often associated with pericarditis and myocarditis, especially myocarditis, in early life, far more often indeed than is the case in adult life. This association is so common that the English often speak of the "acute carditis of childhood," meaning by this term the simultaneous inflammation of all parts of the heart, endocardium, myocardium and pericardium. Unless this association is kept in mind, the course and symptomatology of acute cardiac disease in childhood are likely to be misunderstood.

Symptomatology and Physical Signs.—The onset of acute endocarditis is almost always insidious. In most instances there is merely a rise of temperature. This is seldom high, often irregular, not infrequently

ephemeral. There may or may not be an increase in the pulse rate out of proportion to the rise in temperature. Symptoms pointing directly to cardiac involvement are uncommon. Pain, oppression, anxiety and distress are most unusual and, when present, are almost always due to a complicating pericarditis. In some cases there is slight dyspnea, especially on exertion. To sum up, in the vast majority of cases of acute endocarditis in childhood there is nothing whatever to direct attention to the heart, the only symptom being a rise in the temperature. This being the case, the heart should always be examined repeatedly in all diseases of childhood likely to be complicated by endocarditis. In many cases a change in the character of the first sound can be noted before the development of a murmur. In most, however, the first thing detected is a murmur. This murmur, whether it originates at the aortic or mitral orifice, is almost always systolic in time.

The temperature is likely to be elevated for several weeks. Sometimes, however, it drops to normal in a week or ten days, while in other instances it persists for months. It is usually not very high, ranging between 100° F. and 101° F. at night. Sometimes it is lower and sometimes higher. Not infrequently after an interval of days or weeks the temperature will rise again for a time without any other apparent change in the symptoms. The rate of the pulse is also a little increased, but usually not much when the disease is progressing favorably. If children with acute endocarditis are carefully treated, there is usually not a great deal of enlargement of the heart, the increase in breadth generally not being more than two or three centimeters. The first sound, as a rule, becomes weaker and shorter and the murmur increases in intensity. As improvement takes place, the heart decreases a little in size, the first sound becomes better, the murmur diminishes somewhat or remains the same and, if the lesion is at the mitral orifice, the second pulmonic sound becomes more and more accentuated. In the cases that are doing well there are usually no constitutional symptoms, except the slight fever, and the children feel perfectly well. They usually do not lose much weight, but not infrequently become pale. In other instances, however, in spite of the greatest care, the temperature keeps up, the pulse is frequent, the heart enlarges more and more, the sounds become weak and, if the lesion is at the mitral orifice, the second pulmonic sound becomes and remains weak. In most cases of this sort the temperature finally comes down to normal after many weeks or months. The rate of the pulse, however, while it diminishes, remains increased. The heart diminishes in size, but, nevertheless, remains considerably enlarged. The heart sounds become stronger or normal, the murmurs louder and the second pulmonic sound accentuated. In still other instances, fortunately very rare, in spite of the most careful treatment, the symptoms persist and increase, the heart becomes enormously enlarged and the sounds very feeble while all the symptoms of failure of cardiac compensation develop, passive congestion, enlargement and tenderness of the liver, passive congestion of the kidneys with albuminuria, edema of the extremities, ascites, edema of the lungs, cyanosis, cough and dyspnea. Such cases are quite likely to die. Nevertheless, after weeks or months some of them improve and finally are left with a greatly enlarged heart which is not competent to meet the demands of everyday life.

Children who are not properly treated sometimes do as well and show no more symptoms of their cardiac disease than do those who have the best of care. Usually, however, after an interval of a few weeks they

develop symptoms of cardiac distress or of beginning failure of compensation. Examination then shows greater enlargement of the heart than is usual when children are carefully treated, with more weakening of the sounds and more extensive murmurs. Even if careful treatment is then begun, the heart almost always remains larger than it would have, if it had not been subjected to undue strain in the beginning. In neglected cases all the symptoms of failure of compensation may develop quite rapidly.

There is nothing very characteristic about the physical signs of acute endocarditis in early life. The time, location and transmission of murmurs in relation to the orificial lesions are the same as in the adult. As in the adult, murmurs are due to eddies in the blood current. These eddies may be set up by the narrowing of an orifice, the roughening of an orifice, or the reflux of blood through a partially closed orifice. Loud murmurs may be produced, for example, when the blood is forced powerfully by a strong heart muscle through an orifice which is normal except for vegetations on the edges of the valves, just as a rapidly flowing brook makes a lot of noise when a branch sticks into it. In neither case is there much interference with the flow. If the heart muscle is weak and the blood current less forcible, the roughening of the orifice does not make such large eddies and the murmur is not as loud. A twig projecting into a creek does not make even a ripple. There may be a very considerable reflux of blood through a dilated orifice, which the heart muscle is barely able to overcome. If so, there will not be many eddies formed and but little sound produced. If the heart muscle is strong, the reflux is met by a strong current flowing in the opposite direction. Eddies are formed and considerable sound produced. The intensity of murmurs depends, therefore, on two factors, the condition of the orifice and the strength of the heart muscle. Both must be taken into consideration in estimating the significance of a heart murmur. It is evident that a loud murmur may often be of less significance than a feeble one and an increase in the intensity of a murmur a good sign. A diminution in its intensity may be either a good or a bad sign. It may mean an improvement in the condition of the orifice or a weakening of the heart muscle.

Enlargement of the area of relative cardiac dullness, which represents the real size of the heart, while enlargement of the area of absolute dullness, or flatness, merely shows that less of the heart is covered by the lungs, may be due to either dilatation or hypertrophy of the heart. In mild cases of acute endocarditis, in which there is but little obstruction and little or no regurgitation, the reserve power of the heart muscle is often sufficient to take care of the additional work. In such instances there is no increase in the area of dullness. On account of the extra work there is often, however, if the lesion is at the mitral orifice, a slight accentuation of the second pulmonic sound. There is, of course, no weakening of the first sound. If the obstruction or regurgitation develops slowly, as it usually does, and there is no associated myocarditis, the heart muscle is usually able to hypertrophy rapidly enough to meet the extra work, and, therefore, although there is an increase in the area of dullness, the first sound remains strong and, if the lesion is at the mitral orifice, the second pulmonic sound increases in strength. If, however, there is an associated myocarditis or the lesion at the orifice increases rapidly, the heart muscle is unable to meet the demands made upon it and gives way. The area of dullness is increased as the result of the dilatation of the heart from weakness. The first sound is, therefore, feeble and the

second pulmonic sound, if the lesion is at the mitral orifice, also feeble. If hypertrophy takes the place of dilatation, the area of dullness may or may not become smaller, but the first sound will increase in strength and the second pulmonic sound become louder. The intensity of the murmur is likely to increase at the same time. When the lesion is at the aortic orifice, the increase in the area of dullness is mostly to the left, because the extra work is thrown on the left ventricle. The impulse is likewise displaced downward and outward. When the lesion is at the mitral orifice, the increase in the cardiac area is chiefly upward and to the right, but is also somewhat to the left, because, while the strain falls first on the left auricle, it has to be met by the right side of the heart, and, when it is met, some extra work also has to be done by the left ventricle.

Diagnosis.—There is little danger of overlooking the development of acute endocarditis if the heart is examined repeatedly, as it should be. There is, however, the possibility of mistaking other abnormal sounds for those of endocarditis and, consequently, of drawing erroneous conclusions.

Pericardial friction sounds may be so soft in childhood as to resemble endocardial murmurs. They are not, as a rule, so definitely associated with a single phase of the cardiac cycle, are more superficial, are not transmitted, are increased by the pressure of the stethoscope and are often modified by change in position.

Functional murmurs, presumably due to lowered muscular and nervous tone, often appear during the course of acute diseases. They are usually most marked at the pulmonic area and are accompanied by a venous hum in the neck and by pallor. The cardiac area is not increased and the second pulmonic sound is not accentuated.

Acute febrile diseases are often the cause of myocarditis, which results in dilatation of the heart, usually most marked in the left ventricle. This causes a relative insufficiency at the mitral orifice which is, of course, accompanied by a systolic murmur at the apex. The only sign of any value in differentiating between myocarditis with relative insufficiency at the mitral orifice and acute endocarditis is the cardiac area, which is always increased in myocarditis with relative insufficiency and may be increased or normal in endocarditis. If the area is normal, the first sound of fair strength and the second pulmonic sound normal or accentuated, the condition is almost certainly endocarditis. If the area is increased, the first sound feeble and the second pulmonic sound diminished, there may be myocarditis, endocarditis with dilatation or a combination of myocarditis and endocarditis. If there is myocarditis with cardiac enlargement and relative insufficiency, it is, of course, impossible to determine whether there is or is not also endocarditis.

The murmurs which are most often mistaken for those of acute endocarditis in children are the cardio-pulmonary. These are usually due to the squeezing of the air out of the thin edges of the lungs over the heart by the cardiac systole, and are consequently systolic in time. They may be loud and long. They are usually most noticeable during inspiration, when the lungs are distended. They are more marked when the heart action and respiration are rapid, as they are in fever. They are to be differentiated from endocardial murmurs by their variability with respiration and with the pressure of the stethoscope. Slight pressure tends to increase them; strong pressure often stops them. There is, of course, no increase in the cardiac dullness and the heart sounds are not changed.

Prognosis.—The prognosis has been discussed in considerable detail in describing the symptomatology. In general, however, with reasonably careful handling the immediate prognosis of simple, uncomplicated acute endocarditis is good. A fatal outcome is most unusual and, when it occurs, is usually due as much or more to the disease responsible for the endocarditis than to the endocarditis itself. In rare instances death comes suddenly from embolism. The outlook for the future, however, is not so good. In most instances the cicatricial changes following the involvement of the endocardium result in permanent lesions of the valves and orifices. The chances of complete involution and of the restitution of normal conditions are better in childhood than in later life, however, because of the great reparative power at this period and because of the changes consequent on growth. A small proportion recover entirely. It must be remembered in this connection that the murmurs due to the acute endocarditis frequently disappear, to be followed later by those due to the cicatricial changes at the orifices. The disappearance of the murmur does not, therefore, justify a favorable prognosis. This can only be given when the murmur has not reappeared after an interval of one or two years.

The ultimate prognosis depends to a considerable extent, of course, on how much damage has been done at the cardiac orifices. It also depends very largely on whether the hypertrophy required to compensate for the lesion at the orifice is much or little. If the lesion has reached its maximum development and is compensated for by a small amount of hypertrophy, there is no reason why the child should not lead a normal life, except that extreme strain should be avoided, and live for many years without any symptoms of its cardiac lesion, perhaps even as long as if it did not have one. Moreover, if for any reason compensation begins to fail, it can usually be readily reëstablished by a moderate amount of additional hypertrophy. If, however, the heart is dilated and compensation established only as the result of considerable hypertrophy, the child, while it may be able to lead a quiet life, cannot play and live like other children without failure of compensation developing. When this happens, there is less opportunity for further hypertrophy and it is harder to reëstablish compensation. In such cases the child becomes more and more incapacitated and finally dies, not of acute endocarditis, but of the results of acute endocarditis. The amount of dilatation and hypertrophy, other things being equal, depends very largely on the care taken during the acute and convalescent stages of the disease. The ultimate prognosis is dependent, therefore, to a considerable degree on the treatment during these stages. A week in bed at this time may well mean months of life later, and months, many years.

Children who have had one attack of acute endocarditis are liable to have further attacks, unless the cause of the original attack is removed. If this cause is removed, they are but little more likely to have recurrences than are children who have normal hearts. Every recurrence of acute endocarditis does further damage to the heart, of course, and, as this damage increases the lesion, it necessarily increases the hypertrophy necessary to compensate for it, thus at the same time making the ultimate prognosis worse.

Treatment.—The ideal treatment of acute endocarditis would be to stop the heart and thus prevent all irritation of the endocardium and strain on the myocardium until the inflammatory process had run its course. Such treatment being obviously out of the question, the next

best thing is to diminish the number of heart beats as much as possible. This can only be done by cutting down or out those things which increase the rate of the heart. They are physical exertion and nervous excitement. Rest, both physical and mental, is, therefore, the one important thing in the treatment of acute endocarditis in childhood. Everything else is subordinate. The child must be kept in bed, not only during the acute stage, but for months longer. The acute stage is certainly not over until the fever is gone and the rate of the pulse has come back to, or nearly to, the normal for the age. In many instances, if not in most, it will, of course, always remain a little accelerated. It is not safe, moreover, to even consider allowing the child to leave the bed before the first heart sound is strong and the second pulmonic sound, if the lesion is at the mitral orifice, normal or accentuated. Three months in bed is the minimum, no matter how well the child seems to be doing. In the beginning it must be kept flat; later, as nearly flat as circumstances will allow. As time goes on the child begins to sit up in bed for a short time each day, then for a longer time and finally it sits up in a chair. How rapidly it progresses must necessarily depend on the conditions in the individual case. When children have been badly brought up and spoiled, the attempt to keep them flat may disturb them so much that the rate of the heart is lower when they are sitting up than when they are lying down. In such instances the child should be allowed to sit up in bed part of the time. The object of the bed treatment being to diminish the number of heart beats to the minimum, judgment must be used in carrying out the details of the treatment not to excite the child by it and thus to increase the number of beats instead of diminishing them. The children must, of course, be read to and quietly amused when they are kept flat. When they begin to sit up the toys and amusements which are given to them must be suitable for their condition. It is not advisable to give drums and trumpets to children flat in bed with acute endocarditis, as I have seen done several times on Christmas. There is nothing, of course, about the treatment of acute endocarditis by rest which diminishes the importance or abrogates the necessity for good food, fresh air and sunlight. It is not as difficult to carry out this line of treatment as many physicians suppose. Children that have been properly brought up accommodate themselves to it at once, while those that have been badly trained and spoiled quickly yield to judicious handling and firmness. A child will almost invariably do what he is told to do, if he is convinced that the person who tells him to do it really intends and expects him to do it. Children almost always make better patients than adults and object much less to a routine.

It is just as important to watch children carefully after they are out of bed as when they are in bed. Exercise must be begun very gradually and the heart watched carefully for evidences of overstrain, such as an increase in the rate of the pulse, slight increase in the area of dullness or weakening of the first sound. Exercise should at first be limited to walking on the level, the distance traveled being gradually increased. Walking upstairs should not be allowed for some time. At least a year should be allowed in which to get back to the ordinary life of the child, but even then extreme exertion must be forbidden. In many instances two or three years are necessary.

It is probably advisable in those cases which develop during the course of rheumatism and chorea to give some form of salicylic acid during the acute stage, preferably aspirin. It is doubtful whether it does any good,

but it may possibly limit the process to some extent. Restlessness and discomfort call for the bromides or some form of opium. Opium, as always in early life, must be used with care. Digitalis can do no good in the acute stage of endocarditis, in fact, it is contraindicated. Digitalis, moreover, is useless in myocarditis. It may possibly be of some use in neglected cases and in cases which have done badly in spite of good treatment, if there is dilatation of the heart and failure of compensation from overstrain, not from myocarditis. The so-called cardiac stimulants, such as caffeine and strychnia, are seldom needed. In most instances they do harm rather than good, because rest of the heart, not stimulation, is demanded.

All foci of infection should be looked for, and removed as soon as is possible or feasible. It is very hard to know whether it is advisable to remove infected tonsils and adenoids or diseased teeth in the early stages of acute endocarditis. In most instances the heart, although acutely diseased, is able to stand the strain of anaesthetization and operation without any damage. In fact, the heart is often better able to stand operation in the beginning than it is later. While it is, of course, always important in all conditions to remove a focus of infection, nevertheless, there is a certain amount of danger, if the focus is quiescent, of opening the portals and causing reïnfection by an operation. I have seen this happen not infrequently. I have tried having infected tonsils and adenoids removed early, soon after the very acute stage was passed and during convalescence. My feeling is that the best time to remove them is just after the very acute stage is over. I make it a rule in every instance to explain to the parents that while the acute endocarditis is secondary to the focus of infection in the throat, and there is constant danger of further infection while it remains and that it must, therefore, be removed, there is, nevertheless, always a possibility that there may be an immediate reïnfection of the heart as the result of the operation. I have, on the other hand, never seen reïnfection occur as the result of the removal of abscessed teeth. I, therefore, have them removed immediately.

CHRONIC ORIFICIAL LESIONS OF THE HEART

Chronic orificial lesions are often spoken of as chronic valvular disease of the heart or chronic endocarditis. Both terms are misnomers, especially chronic endocarditis. The ending "itis" implies inflammation. There is no inflammation in these conditions. They are simply deformities of the orifices, resulting from previous diseases. It is true that the valves are also usually involved. The important point, however, is not the condition of the valves, but the condition of the orifices resulting from the condition of the valves. If it is borne in mind that these conditions are not diseases and not inflammatory, it is much easier to understand them. Chronic orificial lesions in childhood are almost invariably the result of acute endocarditis. The causes of chronic arteriosclerosis, except syphilis, are not active in childhood. Moreover, syphilis in childhood rarely attacks the blood vessels or the heart. Since the chronic orificial lesions are consecutive to acute endocarditis, which is very uncommon in the early years of life, they are seldom seen before the age of five years. For the same reason the mitral orifice is much more commonly involved than the aortic, the proportion being nearly as 20 to 1. Obstruction at the mitral orifice is much less common than leakage.

Symptomatology and Physical Signs.—The symptomatology and physical signs of chronic orificial lesions in childhood are on the whole much the same as in adult life and, therefore, hardly seem worthy of detailed description. There are, however, certain points of difference which should be mentioned. The orifice involved being far more often the mitral than the aortic, the symptoms and signs, of course, are more often like those of lesions at the mitral orifice in adults than of those at the aortic orifice. In this connection it should be remembered that, especially in childhood, it is very easy to mistake a murmur at the very beginning of systole for a presystolic murmur and in consequence to make a diagnosis of mitral stenosis when the true condition is mitral regurgitation. Just as in adults also the symptoms depend on whether the orificial lesion is compensated or not rather than on the lesion itself. The subjective symptoms are, as a rule, less than in adult life, because children apparently do not notice slight degrees of discomfort as adults do and complain only when they are marked. On this account medical advice is quite likely to be sought for them considerably later than for adults.

Interference with growth is, of course, peculiar to childhood. It may be very marked, enough so to justify at times the term of cardiac infantilism. Deformities of the chest are also peculiar to childhood. These deformities are, of course, due to the yielding of the cartilaginous sternum and costal cartilages as the heart enlarges. The amount of the deformity of the chest varies according to the age incidence of the cardiac disease, the earlier the onset, the greater being the deformity. Clubbing of the fingers with chronic orificial lesions is also peculiar to childhood. It is quite likely to be associated with cyanosis of the extremities. Neither the clubbing nor the cyanosis of the extremities are, however, often as marked as in congenital cardiac lesions. Children sometimes also have a peculiar cachexia as the result of chronic orificial lesions. They are pale and poorly nourished, irritable and subject to repeated attacks of indigestion.

The murmurs due to chronic orificial lesions in childhood are, as a rule, louder and more diffuse than in later life. They are not infrequently heard in the back and sometimes over the whole chest. Great enlargement of the heart is also more common than in later life. The heart is not infrequently so large that it fills a considerable portion of the left chest. As the result of the great enlargement of the heart, the left lung is not infrequently compressed. The result of this compression is an area of dullness and bronchial respiration, not infrequently of considerable size, in the lower left back. This area extends outward from the median line to the inner edge of the scapula or to the scapular line. It is usually located below the sixth rib, but may extend higher. It may or may not extend to the base of the lung. The voice sounds also are bronchial and likely to be increased in this area. The tactile fremitus is also sometimes increased. Râles are rather uncommon. This area is often thought to be due to a complicating pneumonia and sometimes leads to the suspicion of a pericardial effusion. It is usually easy to distinguish it from pneumonia, because, while the physical signs are consistent with pneumonia, there are no other evidences of the disease. Furthermore, the location of the signs is not that usually found in pneumonia, while it is the characteristic one of compression from the enlarged heart. There is nothing about the physical signs to show whether the compression is due to an enlarged heart or an effusion in the pericardium.

The diagnosis must be made on the history and the other signs in the heart.

Failure of compensation in childhood, outside of the changes in the cardiac physical signs, is usually first shown objectively by enlargement of the liver. Enlargement of the liver may develop very rapidly and is not infrequently enormous. It is usually accompanied by tenderness on pressure and sometimes by pain. Enlargement of the spleen is relatively uncommon. It is not at all uncommon, moreover, to find the liver greatly enlarged without any edema of the lungs or extremities. In general, edema of the lungs apparently occurs relatively later in childhood than in adult life. Auricular fibrillation and its symptoms are quite uncommon in childhood.

Prognosis.—It is impossible to make any general statements which are applicable to individual cases of orificial disease in childhood. The smaller the lesion and the less the amount of hypertrophy which is required to compensate for it, the better is the prognosis, and vice versa. The prognosis is better when one than when two orifices are involved. It is better when there is not and has not been a failure of compensation, when all the foci of infection which might cause another attack of acute endocarditis and further damage to the heart have been removed, when the child is in good circumstances, has intelligent parents and can have its life regulated to its cardiac condition. In general, the prognosis is better when the disease develops after than before eight years.

While recovery is not so very uncommon in acute endocarditis, it very seldom takes place after the residual changes resulting from it have been established at the orifices. There is, of course, no possibility of the repair of these lesions, but it is conceivable that the growth of the parts may be so great as to overcome them and prevent leakage or obstruction at the orifices. If the lesion is a small one and but little hypertrophy has been required to compensate for it, there is a very fair chance that it will not cause any trouble for many years, perhaps never, provided the child's life is regulated to its cardiac condition and it does not have further attacks of acute endocarditis. These can be largely prevented by the removal of foci of infection. If the lesion is a large one and the heart is much enlarged, failure of compensation is likely to occur at any time, especially if there has been dilatation in the past. The course is then much the same as in adult life. Compensation may be reëstablished several times, but each time there is a further increase in the size of the heart. The child becomes more and more of a cardiac invalid and finally dies, usually before reaching adult life, as the result of failure of compensation.

The prognosis is dependent to a considerable extent on the care which the child can receive. The establishment and maintenance of a good condition of general nutrition has a very important bearing, as the nutrition of the heart is closely related to that of the individual as a whole. The prognosis is also much better if all discoverable causes of acute endocarditis are removed. Failure of compensation is especially likely to develop at puberty, when the demands on the heart are greatest because of the rapid growth at that time. Many children begin to go wrong rapidly at this time. Sudden death as the result of chronic orificial lesions is very unusual in childhood.

Treatment.—Children with chronic orificial lesions must be kept under constant observation in order that the progress of the disease may be watched and their life properly regulated. They should be seen and

examined at least every two or three months. In this way only can they be expected to do well. Parents, often with the best intentions, do not understand what to do and what not to do. They are almost certain to make gross errors, unless they are carefully instructed. It is rather difficult to get continuous coöperation on the part of the patient during early childhood. Later, most will help. More difficulty, however, is met with young adults.

When compensation is present, the treatment must be directed to the maintenance of the general nutrition and to preventing overstrain of the heart. This is especially necessary at the time of puberty. Every detail of the child's life must be laid out, for example, the time allowed for study, sleeping, exercise and amusement. When the patients are boys, the most difficult thing to regulate is the amount of exercise. The fundamental principle is that there shall be no prolonged strain. Football, swimming and all competitions must be interdicted. Mild exercise, like horseback riding, walking and golf is allowable. When the patients are girls, much dancing must be prohibited. It is very important in endeavoring to prevent overstrain not to go to the other extreme and not allow sufficient exercise. Proper exercise is most beneficial in that it not only keeps up the general nutrition but keeps the muscles, including the heart muscle, in good condition.

Boys should be trained for some occupation which does not demand too hard strain, preferably one which requires some exercise and much life out of doors. Too sedentary a life is almost as bad as too active a one. In most instances it is unwise for girls to marry, because the strain of pregnancy and labor is very likely to bring on failure of compensation and death. They should be brought up with the understanding that they probably will not be able to marry. It is also important to guard against rheumatism and its causes, as far as possible, as well as exposure to the infectious diseases.

Failure of compensation is shown in children, as in adults, by dyspnea, coughing, cyanosis, weakness, nausea, vomiting, enlargement of the liver, edema, ascites, diminution in the amount of urine, frequent pulse, enlargement of the relative cardiac dullness and weakening of the heart sounds. It may develop gradually or come on suddenly; usually it develops slowly.

The first thing to do in the treatment of failure of compensation is to diminish the work of the heart, that is, to put the child to bed. If it is not uncomfortable when lying down, it should be kept flat. If there is orthopnea, it should be allowed to sit up. The next thing to do is to give some drug which will help the heart to do its work. The one drug which does this well is digitalis. No one of the other drugs of this class—strophanthus, squill, apocynum and convallaria—can do anything which digitalis cannot do and no one of them does the things which digitalis does as well as digitalis does them. They should all, therefore, be discarded. Digitalis depresses conduction and, therefore, induces heart block. It also acts directly on the myocardium and strengthens the systolic contractions. It is doubly useful, therefore, when there is auricular fibrillation with frequent and irregular pulse. It is very useful also when there is no auricular fibrillation, as there usually is not in childhood with failure of compensation, because of its action on the myocardium. It is indicated, therefore, if there is failure of compensation, when the pulse is not much increased in rate or even when it is below the normal rate.

If the digitalis leaves are of proper strength, it makes very little difference whether digitalis is given in the form of a pill, as the powdered leaf, or as the tincture. It is largely a matter of fashion. The chief thing is that the preparation is potent and standardized. The amount of digitalis which is needed is that which will relieve the symptoms of the failure of compensation. The dose should be increased until there is relief or until some of the toxic affects of digitalis appear. These are nausea, vomiting, certain arrhythmias, such as bigeminal pulse and heart block, diarrhea and diminution in the amount of urine after it has been increased. The nausea and vomiting are due to the direct action of the digitalis on the vomiting center in the brain and not to a local action on the gastric mucosa. It is idle to think, therefore, that any preparations of digitalis are more or less likely to cause nausea or vomiting than others. If they do not cause nausea or vomiting, when given in sufficient doses, it is because they are not potent.

In most instances of failure of compensation in childhood the failure develops slowly and it is not necessary to saturate the child with digitalis at once. Consequently, small doses may be given three or four times daily, and increased until the desired result is obtained. In very urgent cases massive doses, sufficient to saturate the system in forty-eight hours, should be used. In less urgent cases a little longer time may be taken. It is almost never necessary to give digitalis intravenously in childhood. In order to saturate the system 0.1 gramme, or 1.5 grains, of the leaf per 10 lbs. of body weight is required. One half of this amount may be given at once, $\frac{1}{4}$ after six hours, $\frac{1}{8}$ six hours later and so on, or it may be divided into six equal doses and given three times a day for two days. One cubic centimeter, or 15 minims, of the tincture is equivalent to 0.1 grammes or 1.5 grains of the leaf. Ten minims is equivalent to one grain and one minim to $\frac{1}{10}$ of a grain of the leaf. One minim of the tincture is equal to about three drops. It must never be forgotten that the drops of the tincture of digitalis are so small. Unless this fact is remembered, or minims are used instead of drops, insufficient doses will be given. It is important to maintain saturation after it has been obtained. To do this enough must be given to make up for that which is excreted. This, in the adult, is the equivalent of from $1\frac{1}{2}$ to 3 grains of the leaf or from 15 to 30 minims of the tincture daily. I know of no accurate figures as to the excretion in childhood. It is probably somewhere between $\frac{1}{3}$ and $\frac{1}{2}$ as much as in the adult.

The other so-called cardiac stimulants are of but little use in failure of compensation Caffeine sometimes relieves respiratory distress. Strychnia is contraindicated in auricular fibrillation. Given in the form of nux vomica, it may possibly help the appetite. Camphor and epinephrine have little or no place in this condition.

The diminution in the excretion of urine in failure of compensation is due, of course, to passive congestion of the kidneys. Digitalis, by increasing the strength of the heart and thus relieving the passive congestion in the kidneys, is the best drug to increase the excretion of urine. Certain other drugs, however, by their direct stimulant action on the renal epithelium also tend to increase the amount of urine. These drugs are caffeine and theobromine. Theobromine is better than caffeine in that it is less likely to excite the patient and interfere with sleep. Theobromine, being insoluble and irritating to the stomach, is best given in the form of one of its salts. Theobromine-sodium acetate, also known as agurin, or the double salt of sodium-theobromine and sodium salicylate,

commonly known as diuretin, are the salts most commonly used. The doses of these salts for children are from 3 to 5 grains, three times daily. Theocine sometimes is more effective than the salts of either caffeine or theobromine. The dose is from ⅓ to ½ as much. It is useless, of course, to give these diuretics unless there is edema or ascites.

Edema, ascites and also passive congestion of the liver and other abdominal organs may also often be considerably relieved by catharsis. It is not wise, however, to use catharsis as freely in childhood as in adult life. Drugs which produce large watery stools are the most suitable. Among these are the salines, compound licorice powder and jalap.

If the patient is restless or uncomfortable, it is not only advisable but most helpful to relieve him by the use of bromides or some form of opium. Opium is not a cardiac depressant, as is often supposed, but in small doses is a cardiac stimulant. The usual precautions in the use of opium in childhood should be observed.

When there is failure of compensation with passive congestion of the abdominal organs, digestion is impaired. The diet, therefore, must be simple and easy to digest. It is inadvisable, also, to give large amounts of fluid. Small amounts of food should be given at relatively short intervals, so as not to embarrass the heart action by a distended stomach.

MYOCARDITIS

Children do not have fibrous myocarditis for the reason that the chief cause of this condition, coronary disease, does not occur in childhood. Chronic myocarditis, secondary to acute myocarditis, develops in rare instances. Acute myocarditis, most often parenchymatous, not infrequently interstitial or mixed, is very common in the course of the acute infectious diseases and intoxications. It is due to the presence of toxins in the circulation. In some instances, however, an acute suppurative interstitial form may be the result of the direct action of bacteria. Acute myocarditis is most common in diphtheria. It is next most common in pneumonia and scarlet fever. It is less frequent in connection with typhoid fever and influenza in children than in adults. Acute myocarditis is also an almost constant accompaniment of endocarditis and pericarditis.

Symptomatology and Physical Signs.—When the myocarditis is not very severe, there may be no symptoms or merely a little irregularity and variation in the pulse rate, with possibly a little change in the character of the first sound or a slight murmur as the result of weakness of the papillary muscles. If the infection of the myocardium is more severe and there is dilatation of the heart as the result of myocardial weakness, the area of relative cardiac dullness is increased, the heart sounds are feeble and there is often a murmur. This is usually systolic in time and not very loud. The frequency of the pulse is usually increased and it is likely to be more or less irregular. In the most serious cases the pulse becomes very frequent and irregular and at times is not palpable at the wrist. If, however, the sino-auricular node or the bundle of His is involved, the rate of the pulse is diminished. When there is marked myocarditis the children are likely to become pale or cyanotic. They are not infrequently faint and sometimes lose consciousness. These symptoms are sometimes very slight and fleeting, sometimes more marked and intermittent, sometimes continuous and ending in death. It must not be forgotten, however, that death may occur suddenly, usually as the result of exertion, before any of the more serious manifestations of myocarditis have appeared.

Diagnosis.—The presence of myocarditis should always be suspected and looked for in the course of the acute diseases in which it occurs. It is safe to assume that it is present whenever the rate of the pulse is quicker than would be expected from the height of the temperature, and when it is irregular. It is almost certainly present, if the rate of the pulse is much slower than would be expected under the circumstances. It is certainly present when the area of relative cardiac dullness increases in connection with a weakening of the heart sounds. The presence of a murmur is corroborative evidence. Further evidences of its existence are pallor or cyanosis and attacks of faintness or syncope.

The diagnosis of myocarditis is more difficult when it occurs as a complication of acute endocarditis or as a part of a general affection of the heart, which has been described as "acute carditis." The differential diagnosis between acute endocarditis and myocarditis has been described in discussing acute endocarditis.

Prognosis.—Most children with acute myocarditis, even if of a severe type, recover. Recovery occurs not infrequently when the outlook seems absolutely hopeless. In other instances, however, death occurs suddenly and unexpectedly when the symptoms and physical signs have not been at all marked. The prognosis in an individual case, therefore, should always be guarded, because death may occur suddenly in any case at any time until the heart muscle has returned to normal. In general, an infrequent pulse is a more serious sign than a frequent and irregular pulse. Pallor, cyanosis and faintness, even if slight and fleeting, are always of serious import.

Treatment.—The only thing which does much of any good in the treatment of acute myocarditis and myocardial insufficiency is rest. Rest is even more important than in the treatment of acute endocarditis. It must be as nearly absolute as possible. The child must not be allowed even to raise its head or to turn over. It must be kept absolutely still, not only as long as symptoms persist, but for some time longer. It is impossible to overestimate the necessity of absolute rest and the dangers of any exertion, when there is evident involvement of the myocardium, it being impossible to determine clinically how serious the involvement of the myocardium really is. Every case in which there is myocarditis should be treated as if the involvement of the myocardium was serious. Absolute, or nearly absolute, rest should be kept up, not only as long as the symptoms of myocarditis persist but for some time longer. The child should not be allowed to begin to sit up, even in bed, until the pulse has returned to its normal rate and become regular, the cardiac area returned to normal and the sounds become of good strength. Convalescence is usually quite rapid and relapses unusual. The period of absolute rest is usually not more than two or three weeks, almost never over six weeks. The same precautions during the early stages of convalescence should be taken as in acute endocarditis. The recovery is usually complete, however, in a few weeks or, at most, a few months.

Drugs are of practically no use in the treatment of acute myocarditis. Alcohol is useless, unless as a food, and in large doses undoubtedly does harm. Digitalis cannot act on an acutely degenerated muscle and, therefore, can do no good in a severe case of acute myocarditis. In a mild case, it is not needed. It is possible that strychnia, caffeine or camphor may help to tide over an emergency. Nitroglycerine and the other nitrites are dangerous, as they tend to produce vasometer paralysis.

It is sometimes necessary in the beginning to give bromides or some form of opium in order to keep the child quiet.

DILATATION OF THE HEART

Dilatation of the heart may occur in connection with acute endocarditis or myocarditis, or as the result of the weakening of the heart muscle in chronic orificial lesions. Dilatation of the heart may also occur in children as the result of undue and excessive physical exertion. The volume and weight of the heart relatively to the body weight are small in the years before puberty and increase rapidly during puberty, reaching the adult relation soon after. That is to say, the heart is relatively smallest in comparison with the body weight at the time when the child is growing most rapidly. The heart at this time is less able to meet the demands made upon it than at any other time in the life of the individual. In many instances it is about all that it can do to meet the physiologic demands for the maintenance of life and growth. It has no reserve power to be expended in work or play. It is presumably partly for this reason that growing children are often so indolent and lazy. Under these conditions there is great danger of the development of dilatation of the heart as the result of overstrain. Irreparable injury may be caused by overexertion. At this period, therefore, children should be watched and guarded very carefully, especially if they are easily fatigued or appear below par in any way. If they are not protected, but are allowed to lead their usual life, harm may be done which cannot be corrected for years and perhaps never. If they are looked after properly for a few years and tided over this critical period, the heart will then catch up with the body and they can resume their normal lives.

Dilatation of the heart at this age is more likely to occur in boys than in girls, because of the strain put on the heart as the result of overindulgence in athletics in schools, summer camps, gymnasiums and boy scout organizations. Masters, coaches and leaders alike fail to appreciate the physiologic conditions and urge or compel growing boys to take part in contests and to continue in games when they are in no condition to do so. At this age the heart should not be subjected to the strain of long races and tramps or of severe contests, like football and rowing. I have seen a considerable number of boys and some girls seriously injured in this way.

Symptomatology and Physical Signs.—The symptoms of dilatation of the heart from overstrain are frequent pulse, shortness of breath, cough, weakness and general malaise. Physical examination shows the cardiac area increased, the pulse frequent and the first sound weak. There is often a systolic murmur in the mitral area or at the apex as the result of a relative insufficiency of the mitral valves.

Prognosis.—In most instances, with proper care and treatment, the dilatation will gradually diminish and finally disappear without permanent enlargement and hypertrophy. If the condition is neglected, however, permanent enlargement of the heart may result.

Treatment.—If the dilatation is marked, as it is sometimes when it has developed acutely, the child must be put to bed and kept there until the size of the heart has returned to normal or nearly normal, the pulse has come down to normal and the murmur has disappeared. If the dilatation has not been as acute or as marked, it may be sufficient to cut out all violent exercise and to have the child rest several hours a day. In every instance all contests and violent exercise must be stopped, not to be taken up again until the child has regained its strength and has stopped growing so rapidly. In many instances extreme care must be taken for several years. I have kept children in bed on this account for months and

kept them out of competitive athletics for years before they were able to take them up again. Drugs, except for symptoms, are of little help in this condition.

In certain instances, in which there is no dilatation of the heart, the heart shows the strain by an increase in the rate of the pulse, which, while not as serious, does mean that the same care must be taken to avoid dilatation as would be if it had already occurred.

FUNCTIONAL DISORDERS OF THE HEART

Functional disorders of the heart are very uncommon before middle childhood, but become increasingly frequent up to puberty, when they are most common. They are often due to indigestion or overstrain, especially mental or nervous. The exciting cause is occasionally tea, coffee or cigarettes. These disturbances are usually associated with other evidences of disturbed nervous equilibrium. The most common symptom is attacks of palpitation. At times more marked symptoms, such as oppression, dyspnea or faintness, appear. The pulse is usually increased in rate and often irregular. The cardiac area and sounds are normal.

These disturbances, as already noted, are especially likely to appear at the time of puberty. This is probably because of the rapid growth of both body and heart at this time, that of the heart usually lagging a little behind that of the body, and the general disturbance of the nervous system. Enlargement of the area of dullness and murmurs, probably due to relative insufficiency at the mitral orifice, are not infrequently added to the usual signs and symptoms, especially if there has been in addition physical overstrain.

Diagnosis.—If this condition is not borne in mind, it is easy to mistake it for organic disease of the heart. If it is borne in mind, the character of the onset taken into consideration and the physical signs studied carefully, it is, however, not difficult to recognize it.

Treatment.—The treatment is, of course, in the main, the removal of the cause. If there is dilatation with relative insufficiency at the mitral orifice, the treatment is essentially the same as in dilatation of the heart from physical overstrain. Rest, as in all cardiac conditions, is of the greatest importance. Bromides are useful in some cases, while tonics, like nux vomica, help in others.

INORGANIC CARDIAC MURMURS

There are a considerable number of different murmurs which may be heard in the precordial region in childhood which are not due to either acute endocarditis or chronic orificial lesions. It is very important to recognize these murmurs because, if they are not recognized, it is very easy to make erroneous diagnoses and prognoses.

Cardio-pulmonary Murmurs

A considerable number of the murmurs heard in the precordial region in early life are made outside of the heart. They are rare in infants and most common in middle childhood. They are usually heard at the apex or along the left border of the heart, where the lungs cover it, but seldom at the base. They are almost always systolic in time, sometimes diastolic, seldom double. They are generally soft and blowing, but may be quite loud. They are usually sharply localized, but are sometimes transmitted. The intensity of these murmurs is often changed by the pressure

of the stethoscope, the intensity, as a rule, being weakened. The most characteristic point about these murmurs is that they vary with respiration. They are sometimes louder in inspiration, sometimes in expiration. They cease when the breath is held. They are presumably due to the squeezing of air out of the alveoli at the edge of the lung, but may, perhaps, sometimes be due to the rushing in of air. This explanation may, however, not be the correct one. Further evidence to show that these murmurs are made outside the heart is that there are no other evidences of cardiac disease. The cardiac area is normal, the rate normal, the action regular, the sounds strong, and there is no change in the second pulmonic sound. Furthermore, there are no subjective symptoms. These murmurs are of no importance whatever.

Another extracardial murmur, which is also probably pulmonary in origin, is one which varies with the position of the child. It is usually most marked when the child is lying on its back, diminishes when it sits up, and disappears when it stands up. Sometimes, however, it may be heard loudest when the child is standing. This murmur is more common in boys than in girls, and is heard most frequently in middle and late childhood. The murmur is the only sign. There are no other evidences of cardiac disease. It is of no importance.

Still another murmur, which, while it is probably made in the vessels, is almost certainly due to pressure from outside, is a murmur which is very often heard over or above the manubrium in infancy and sometimes in early childhood. It is most marked when the head is extended, and often disappears when the baby is lying down or the head is flexed. It is almost certainly due to pressure on the innominate veins. This pressure, in some cases, is undoubtedly due to enlarged bronchial glands or to an enlarged thymus. The murmur can be brought out, however, in almost every infant by complete extension of the head when it is in the upright position. It is certain that not all infants have enlarged bronchial glands or an enlarged thymus. This sound is of no value, therefore, in the diagnosis of these conditions, and, in fact, has no pathologic significance.

Systolic Murmur in Pulmonary Area

A systolic murmur in the pulmonary area, that is, in the second and third left interspaces, is very often heard throughout early life. It is so common that it may be regarded as physiologic. I do not know, and doubt if any one does, whether it originates inside or outside of the heart. It is always systolic in time and soft and blowing in character. It is usually rather limited in its distribution, but may be heard both below and to the right of the pulmonary area. It is seldom heard in the neck. There are no evidences of cardiac disease, either subjective or objective, associated with it. The heart is normal in size, the rate and rhythm are normal, the sounds of good strength, and the second pulmonic sound unchanged.

The only cardiac lesion with which it can possibly be confused in childhood is a congenital lesion at or near the pulmonary orifice. If there is such a lesion, there is often a thrill, and the murmur is rough, usually loud, and transmitted into the neck and back. The right side of the heart is often enlarged and the second pulmonic sound almost invariably weakened. There are usually other evidences of cardiac disease, such as cyanosis, clubbing of the extremities, bulging of the precordia and disturbance of nutrition. Confusion is hardly possible if the characteristics of the murmur and the differences in the condition of the heart and circulation in the two conditions are borne in mind.

Functional Murmurs

Intracardiac murmurs are due to interference with the normal flow of the blood through orifices which are narrowed or roughened, or to the formation of eddies in the flowing blood as the result of the reflux of blood through orifices which the valves do not entirely close. The failure of the valves to close may be because the orifice is dilated, the valves shrunken or the nervous or the muscular contol of the valves imperfect.

It is evident that when intracardiac murmurs are functional they must be due to the failure of the valves to close the orifice, and that this failure can only be due either to dilatation of the orifice or to the imperfect action of normal valves. Functional murmurs due to leakage at the mitral orifice are, therefore, systolic in time and are heard in the mitral area and at the apex; those at the tricuspid orifice, systolic in time and heard under the sternum; those at the aortic and pulmonary orifices, diastolic in time and heard in the appropriate locations. Functional murmurs are often associated with blowing sounds over the great veins of the neck. These are usually systolic in time, but may be almost continous. They are especially common when there is anemia. It is not probable that either the murmur over the heart or the blowing sound in the neck, are due directly to the anemia, that is, to any change in the blood itself. They are simply manifestations of the lowered mucular and nervous tone produced by the anemia. The cardiac muscle and the nerves controlling the heart are affected, like other muscles and nerves, in disturbances of nutrition. Such disturbances are especially likely to occur at or a little before puberty. At this time, moreover, as the heart does not increase in size as rapidly as the body, it is less able to meet the demands made upon it. Its walls and orifices not infrequently yield a little to the strain, and murmurs develop.

There is nothing about the character of functional murmurs which serves to disinguish them from organic murmurs. Either may be short or long, faint or loud, transmitted or localized. When murmurs are functional, the heart may or may not be of normal size, according to whether the leakage is due chiefly to incoördination of the valves or to dilatation of orifices as the result of a general dilatation of the heart wall. If the murmur is systolic, the cardiac area normal, the first sound of good strength, and there is no accentuation of the second pulmonic sound, the murmur must be functional, because, if the leakage is due to an organic lesion, the extra work thrown upon the right ventricle, even if it does not cause enough hypertrophy to increase the cardiac area, will necessarily increase its strength enough to accentuate the second pulmonic sound. If the cardiac area is increased, the first sound strong, and the second pulmonic sound accentuated, it is evident that the murmur is not functional but is due to an organic lesion at the mitral orifice, which is compensated for by hypertrophy of the right ventricle. If the cardiac area is increased, the first sound of fair strength, the second pulmonic sound normal, and there are no evidences of failure of compensation, the murmur is almost always functional, because, if it is due to an organic lesion at the mitral orifice, the enlargement of the cardiac area must be due to failure of compensation on the part of the right ventricle, which will necessarily result in a weakening of the second pulmonic sound and systemic evidences of failure of compensation. The presence of a blowing sound over the vessels of the neck always counts in favor of a functional murmur.

If the murmur is diastolic and in the aortic area, it is almost certainly functional, if there is no enlargement of the cardiac area, because organic, aortic insufficiency is usually due to a serious lesion and causes enlargement of the left ventricle. If there is enlargement of the cardiac area, the chances are that the murmur is organic, unless there is marked anemia. If there is marked anemia, the murmur is almost always functional, even if it is accompanied by a Corrigan pulse, high pulse pressure, and a double murmur in the groins.

ACUTE PERICARDITIS

Acute pericarditis is very unusual in infancy. Its frequency increased directly with age. In infancy it is usually pyemic in origin, in early childhood secondary to inflammatory processes in the neighboring parts and in middle and late childhood to rheumatism and the causes of rheumatism. It is less frequently secondary to pneumonia and more often tuberculous than in later life. It is often associated with endocarditisand myocarditis in acute inflammatory conditions of the heart. It may precede, accompany, or follow the endocarditis. The tendency to effusion is greater than in adults. Owing to the nature of the exciting cause, the effusion is usually purulent in infancy, while throughout childhood there is a greater tendency for it to become purulent than in adults. A blood tinged exudation is more common than in later life and less indicative of tuberculosis or malignancy. An effusion often forms very rapidly in childhood. Acute pericarditis is never primary, although it sometimes seems as if it were. It is always secondary to some infection elsewhere which, however, may be so slight as to be overlooked.

Acute pericarditis is, of course, bacterial in origin. The etiologic organisms are either those which cause the original infection to which the pericarditis is secondary or secondary invaders, usually the pus organisms. When pericarditis is a part of a general inflammation of the heart, the so-called "carditis" of childhood, very often no organisms can be found.

Dry Pericarditis

Pathology.—The lesions in dry, or fibrinous, pericarditis vary materially in degree and may be either localized or general. In the mildest cases the pericardium simply loses its lustre and is a little roughened as the result of the formation of a thin fibrinous coating. When this fibrinous coating is thicker, the movement of the pericardial surfaces over each other throws it into ridge-like folds or gives it a honey-combed appearance. When the exudate is still greater, the surfaces resemble slices of bread and butter which have been separated, the fibrin being in shreds and giving a shaggy appearance. The membrane below the fibrinous exudate is injected or ecchymotic. In general, however, the fibrinous exudate in childhood does not become very thick. There is usually also a slight excess of fluid in the pericardial cavity. During convalescence adhesions are quite likely to be formed between the inflamed surfaces. This does not always happen, however, and the pericardial surfaces may be restored to normal.

Symptomatology and Physical Signs.—When dry pericarditis is due to the extension of some acute inflammatory condition in the neighborhood, such as pneumonia or pleurisy, there often are no new symptoms to call attention to it. It is, in fact, often unsuspected until it is discovered at the autopsy. The temperature may perhaps be a little higher, the pulse

a little more frequent, the cough a little more troublesome and respiratory distress a little more marked. In other instances the symptoms may be quite severe and like those in the apparently primary cases. Physical examination may or may not reveal a friction rub. It is surprising how often no sound is heard in childhood when the pathologic lesions found at autopsy would seem to show that a friction rub ought to have been heard. The friction rub may be located anywhere, according to the location and distribution of the pericardial inflammation. It is generally stated that it is most often heard at the apex or in the mitral area. In my experience it is heard most frequently at the base of the heart. If the pericardial inflammation is limited to the diaphragmatic portion, it is, of course, impossible to hear a friction rub. It is said that a friction rub may sometimes be heard in the back when the pericardium is involved posteriorly. I have never heard a pericardial rub in this situation. When the pericarditis is secondary to inflammation in the pleura, the pericardial rub is likely to be obscured by the pleural friction rub. The character of the friction sound varies with the condition of the pericardium. It may be very soft and shuffling, when there is but little roughening of the pericardial layers, or rough and creaking, when there is considerable thickening which is relatively dry. When the thickening is softer, the friction rub may be fairly loud but soft. It may be lacking when there is a considerable amount of soft fibrinous exudate. The friction sound is, of course, audible in both inspiration and expiration. It sounds close to the ear, is often increased by pressure of the stethescope and often varies in intensity with change of position.

When dry pericarditis is a part of a general acute inflammatory affection of the heart, "carditis," the symptoms are hard to distinguish from those due to the associated endocarditis and myocarditis. There is, however, more likely to be precordial pain, a sense of constriction in the chest, cough and an increase in the rate of the respiration. The physical signs are the same as when the pericarditis is due to other causes.

When it develops in the course of chorea, rheumatism or one of the infections which causes rheumatism, and, as sometimes happens, there is little or no involvement of the endocardium and myocardium, the symptoms are quite characteristic. They are essentially the same as in those cases which are apparently primary, but which are undoubtedly really secondary to some mild infection elsewhere. The onset is acute with a sharp rise in the temperature to from 102° F. to 104° F., with a somewhat relatively greater rise in the rate of the pulse and respiration. There is usually pain in the precordia and a sense of constriction of the chest or of oppression. Cough is not infrequent. Dyspnea is sometimes marked. Younger children, however, are quite likely to locate the pain in the epigastrium, and there may be, as in pneumonia, rigidity of the abdominal muscles. In other instances, not very uncommon, the child complains of pain only on swallowing. In such instances, of course, the inflammation of the pericardium is located posteriorly. In such cases also friction rubs are often inaudible. This combination of an acute febrile disease without any physical signs and with pain on swallowing is almost pathognomonic of acute dry pericarditis. In other instances there may be no pain and no symptoms pointing towards the heart, except a disproportionate increase in the rate of the pulse and rapid and guarded respiration. In many instances children with dry pericarditis have a peculiar, anxious expression and look sicker than would be expected from what can be found on physical examination.

The course of dry pericarditis is very indefinite. The symptoms and physical signs may last but a few days or may persist, perhaps intermittently, for two or three weeks. In the great majority of instances the symptoms subside and the friction rub disappears without the formation of an effusion. The disappearance of the friction rub may be due either to the healing of the lesions or to the formation of adhesions. It may also be due to weakness of the heart. In a small proportion an effusion, either serous or purulent, is formed. When this happens, the friction rub usually disappears, but may persist, usually at the base. Dry pericarditis is almost never a fatal disease but, when it is a complication of other diseases, makes the prognosis of these diseases somewhat worse.

Diagnosis.—The diagnosis of dry pericarditis on the symptomatology has already been mentioned. It is usually easy to recognize the friction rub of pericarditis, but it is possible to confuse it with endocardial murmurs. Friction sounds are close to the ear, and, while they are usually rather sharply localized, their position changes from time to time. They are often increased by pressure of the stethoscope and not infrequently vary in intensity with change in position. Endocardial murmurs are not close to the ear, are always in the same location, which is characteristic for some lesion or lesions at a given orifice. They are not increased by pressure of the stethoscope and do not vary with change in position. Endocardial murmurs are closely connected with the heart sounds. Pericardial friction sounds are not in close conjunction with the heart sounds, but between them, either during systole or more often in both systole and diastole. Sometimes they occur just before and just after the second sound and sometimes they obscure the first sound. In rare instances they may be palpable. The endocardial murmurs which are most likely to be mistaken for pericardial friction rubs are those connected with a simultaneous stenosis and regurgitation at the aortic orifice. Such an orificial lesion almost never occurs in childhood, however, and, when it does occur, there are other signs, such as a pistol shot or a double murmur in the groins, a Corrigan pulse and an increased pulse pressure, in the peripheral circulation, all of which signs are lacking in acute pericarditis. Pericardial friction rubs may also be confused with the so-called pleuro-pericardial friction rubs, which are not truly pleuro-pericardial, but, when the pleura is inflamed, are made in it, as the result of the beating of the heart. They occur, therefore, only where the pleura overlies the heart, while pericardial friction rubs may occur anywhere in the cardiac area. They vary in intensity according to the phase of the respiration, sometimes being louder or lacking in inspiration, sometimes in expiration. The intensity of pericardial friction rubs on the other hand is independent of the phase of respiration. It is possible also to mistake the peculiar, fine crepitations, which are sometimes heard in mediastinal emphysema, or even those heard in subcutaneous emphysema, for pericardial friction rubs, if one is not on his guard.

Treatment.—The treatment of dry pericarditis is mainly of the causative condition. In the cases which are associated with rheumatism or with the infections which cause rheumatism, it is probably worth while to give some form of salicylic acid, preferably aspirin. An ice bag to the precordia almost always makes the patient more comfortable, if there is pain, usually reduces the rate of the heart and possibly may have some curative action. An ice bag ought never be applied directly to the skin, but several layers of flannel should be placed between it and the skin. If the ice bag is heavy and rests directly on the chest, it may cause much

discomfort. It should always be suspended in some way so that the full weight of the bag does not come directly on the chest. It must also be remembered that the continuous application of cold to an infant or young child may cause serious depression and even collapse. In some instances hot applications give more relief. Blisters should never be used.

If the heart action is rapid and turbulent, it is sometimes useful to use aconite to depress it. The tincture of aconite may be given in doses of from ¼ of a drop to one drop every hour, according to the age of the child. If children with dry pericarditis are restless, it is advisable to use the bromides. If they do not quiet them, some form of opium must be used. Morphine controls pain better than any other. It should be given in doses of from $\frac{1}{32}$ of a grain to ¼ of a grain. As always, when opium is given to children, a small dose should be given first and then increased, if it is not efficient.

PERICARDITIS WITH EFFUSION

Pericarditis with effusion usually follows dry pericarditis, but may sometimes develop insidiously. If so, it is likely to be tuberculous. The effusion may be serous, sero-fibrinous, hemorrhagic or purulent. Purulent effusions are not uncommon in infancy. In childhood, however, although purulent effusions are more common than in adult life, in the vast majority of cases the effusion is serous or sero-fibrinous. Hemorrhagic effusions are said to be more common in childhood than later. I have never seen but one and that was caused by a piece of a needle in the anterior chest wall. It is probably true, as is the case with all hemorrhagic effusions in early life, that they are of less serious import than in adults and more likely to be due to tuberculosis than to cancer.

Symptomatology.—The pain due to the antecedent dry pericarditis usually diminishes and finally ceases as the fluid accumulates. In some instances, however, if there is not much fluid, it persists. The fluid usually collects rather slowly, but sometimes increases in amount very rapidly, so rapidly indeed that urgent symptoms from pressure may develop in a few hours. It is usually rather slowly absorbed, but sometimes disappears quite rapidly. As the result of the pressure of the fluid on the heart there is a sense of oppression in the chest, and respiratory discomfort and finally distress develop. There is more or less dyspena and in severe cases orthopnea. The patient is usually restless and has a rather characteristic anxious expression. There is usually some cyanosis. In some instances it is very marked. The cyanosis is probably due in part also to pressure on the veins and lungs and to passive congestion of the lungs. Pressure on the veins may cause distention of the vessels of the neck and marked cyanosis of the face and head. Pressure on the esophagus may cause dysphagia, on the trachea, cough, and on the recurrent laryngeal nerve, hoarseness or aphonia. Pressure on the lungs may compress them, especially at the left base posteriorly. All the symptoms of passive congestion from a weak heart may finally develop. The temperature is usually obscured or modified by that of the causative disease. If not, it is as a rule moderately elevated and somewhat irregular. If the effusion is purulent, it is usually more irregular and may be higher. There may also be chills and sweating. The pulse rate is always increased. If the effusion is small, it may not be increased out of proportion to the fever. It usually is, however, and is often quite frequent, small and irregular. I have never seen the pulsus paradoxus, that is, the weakening or disappearance of the pulse with inspiration, in peri-

carditis with effusion, although it is said to be not uncommon in this condition. The respiration is, of course, rapid. In addition there are all the symptoms common to the acute febrile diseases.

Unless the effusion is tuberculous, there is always some leucocytosis. It is usually more marked when the effusion is purulent.

Physical Signs.—The physical signs necessarily depend on the amount and location of the fluid in the pericardium and the position of the heart in the fluid. It is evident, when the attachments of the heart to the great vessels are borne in mind, that the heart can neither float nor sink in the fluid, although the apex may perhaps be rotated to a certain extent. It is also evident that, because of the short distance between the posterior surface of the sternum and the anterior surface of the vertebral column, the heart cannot be much displaced either anteriorly or posteriorly. The fluid must necessarily, therefore, surround the heart, and there is no reason why a friction rub should not continue to be heard, even when there is an effusion, unless it is very large.

Recent investigations seem to show that in pericarditis with effusion the fluid accumulates first between the heart and the diaphragm, about the apex, and along the left border. The result is to increase the cardiac dullness slightly upward and to the left, mostly upward. The earliest change in the shadow shown by the Roentgen ray is also a slight extension upward. Another of the results of the accumulation of fluid in this position is to push down the left lobe of the liver. The liver is not pushed down as a whole, but the left lobe is rotated downward. This is shown by a depression of the lower border of the liver dullness on the left. This low position of the lower border of the liver dullness is of little value, unless it is known that the lower border was in the normal position before the onset of the illness and also that there is no passive congestion of the liver from cardiac failure. It is of even less value in children, because the lower border of the liver may normally remain low, as in infancy, for four or five years.

The next place that the fluid accumulates is apparently about the great vessels at the base of the heart. This accumulation results in an increase in the normal dullness in this region with an extension laterally in both directions. The next change in the shadow shown by the Roentgen ray is also a widening of the shadow at the base of the heart about the great vessels.

It seems probable, in the light of these investigations, that the change in the cardio-hepatic angle, described many years ago by Ebstein, from an acute to a right and then to an obtuse angle, occurs only when there is a considerable amount of fluid in the pericardium. This also seems true of Rotch's sign, described in 1878, of flatness in the fifth right space extending more than two centimeters to the right of the sternum. Recent studies, both experimental and with the Roentgen ray, also seem to show that both Ebstein's and Rotch's signs may often be lacking, even when there is a large amount of fluid in the pericardium. Nevertheless, I have often found them present and have learned to consider them to be of considerable importance in the diagnosis of pericardial effusions.

When there is a large amount of fluid in the pericardium it is distended in all directions, and the area of cardiac dullness is consequently much enlarged in all directions. It may extend well into the axilla and even behind the posterior axillary line. The effusion may compress the lower lobe of the left lung sufficiently to cause marked dullness or flatness, with bronchial respiration and voice sounds, in the lower and middle left back.

In some instances in young children the dullness may simulate that of pleurisy with effusion. The right border of dullness, in my own experience, usually forms an obtuse angle with the liver, except in the case of very large effusions, when the angle may be acute again. With large effusions the Roentgen ray is likely to show a large, somewhat globular shadow.

The cardiac impulse is usually weakened or absent when there is more than a small amount of fluid in the pericardium. If it persists, it is usually not the apex impulse which is seen or felt, but that from some other part of the heart. The heart sounds may be quite distinct, even with considerable amounts of fluid. Eventually, of course, they become weakened. This weakening is, however, often due as much to weakening of the heart muscle from exhaustion as to the fluid. Friction rubs often persist in spite of the effusion, because the effusion does not cover the whole heart anteriorly.

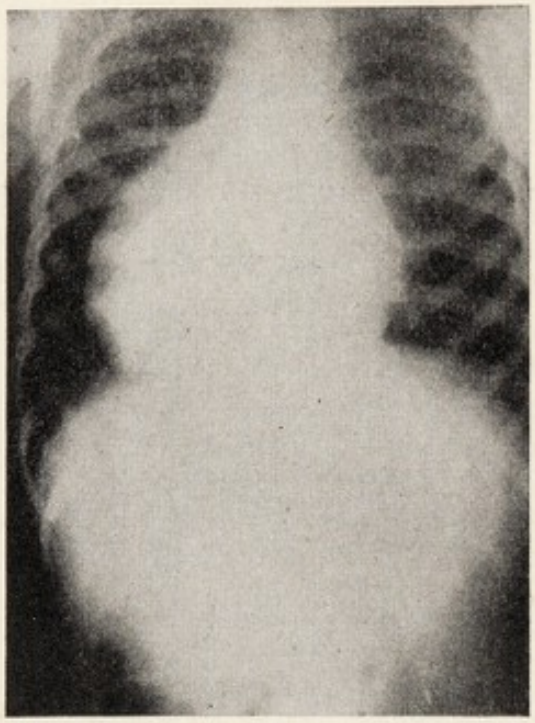

FIG. 137.—Dilatation of heart.

Diagnosis.—When an effusion develops in the course of or as a sequela of dry pericarditis, there is usually little difficulty in the diagnosis. The first signs to be looked for are a slight increase in the area of cardiac dullness upward, depression of the lower border of the liver dullness on the left and, a little later, an increase in the dullness and a widening of its area in the region of the great vessels at the base of the heart. The Roentgen ray also shows an increase of the shadow upward and then widening at the base of the heart. It is very easy to overlook small effusions, however, even when great care is taken. Fortunately, it makes very little difference whether they are recognized or not.

It is often very difficult, when a child is seen for the first time and there is no history of dry pericarditis, to distinguish between a dilated heart and an effusion in the pericardium. If there is a pericardial friction rub, the chances are in favor of an effusion, but it is possible to have a friction rub with a dilated heart and no effusion. The impulse in dilatation is more likely to be visible and palpable than in pericarditis with effusion. It is, moreover, diffuse and wavy while in pericarditis with effusion it is localized. The area of dullness is increased in both. It is somewhat more triangular in effusion than in dilatation, but not enough so to be of very great importance. An obtuse cardio-hepatic angle counts strongly in favor of an effusion. An acute angle does not rule it out, however, being consistent with either. Flatness in the fifth right space more than two centimeters to the right of the right sternal border is almost positive proof of an effusion. Its absence does not, however, exclude one. Change in the position of the upper border of dullness with change in position is strong evidence in favor of an effusion. The heart sounds are usually shorter, clearer and nearer in dilatation than in effusion, in which they are likely to be distant and muffled. The second pulmonic sound is usually

more distinct in effusion than in dilatation. The Roentgen ray is sometimes of great assistance and shows outlines in effusion inconsistent with dilatation. In other instances, however, there is nothing characteristic about the shadows.

A very large pericardial effusion may sometimes be mistaken for an effusion into the left pleural cavity or an encapsulated pleural effusion. The physical signs may be very much the same. When there is a pleural effusion, however, the heart sounds, while displaced, are louder than when the effusion is in the pericardium and the impulse, while also displaced, is usually more distinct. A pericardial friction rub is strong evidence in favor of a pericardial effusion. It is also sometimes very difficult to determine whether there is an effusion into the pleura as well as into the pericardium. I must confess that in the days before the Roentgen ray was available I have several times drawn a serous fluid from the left chest

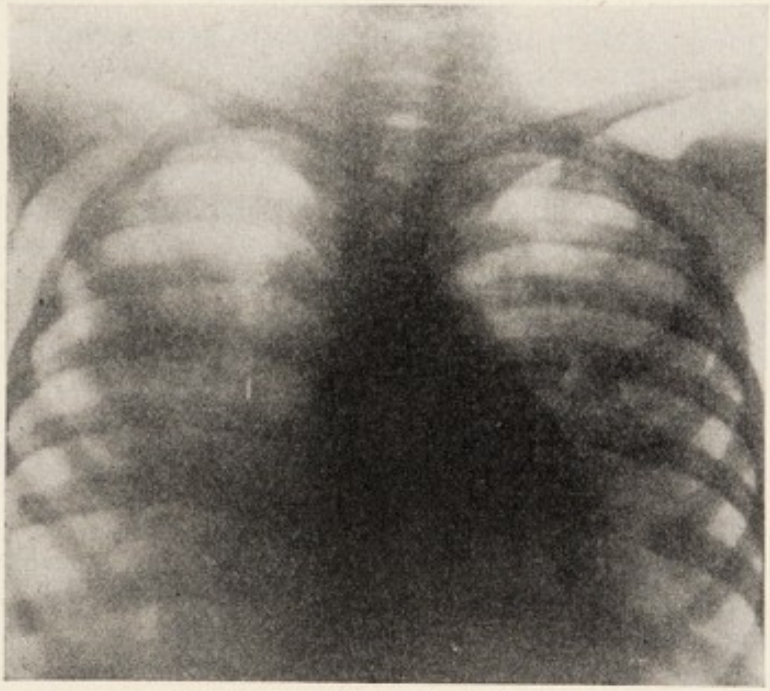

FIG. 138.—Pericardial effusion.

without knowing whether it came from the pleura, the pericardium or both.

There is nothing in the physical signs to show whether an effusion is serous or purulent. If the effusion develops in connection with chorea, rheumatism or any of the causes of rheumatism, it is almost certainly serous and usually does not contain much fibrin. If it develops in the course of pneumonia, it is usually serous, but may be purulent. If it develops in infancy or in the course of a septic infection of any sort, it is generally purulent. If it is probably tuberculous, it may be either serous or purulent, perhaps hemorrhagic. An irregular septic temperature is in favor of a purulent effusion, as are chills and sweating. A very high white count is in favor of a purulent effusion, but is not of much value, because the white count may be quite high when the effusion is serous or sero-fibrinous. The only way, therefore, in which a positive diagnosis can be made is by puncture of the pericardial sac.

Prognosis.—The prognosis in infancy is very grave, partly on account of the age and partly because of the usual etiology at this time. When the effusion is purulent in childhood, the chances are considerably against

recovery, as much because of the cause of the pericarditis as because the effusion is purulent. Occasionally, however, even when the effusion is purulent, recovery occurs, if the pericardium is opened and drained. When the effusion is serous or sero-fibrinous, recovery is the rule in childhood. Death sometimes occurs, however, in neglected cases as the result of the pressure of the fluid. More often it is due to associated conditions in the endocardium and myocardium. Unless children with a serous effusion in the pericardium are carefully watched, death may occur suddenly as the result of a very rapid increase in the amount of fluid. Adhesions of the pericardial layers almost invariably result. In many instances adhesions between the pericardium and pleura and between the pericardium and the mediastinal tissues also develop, if there has been a complicating pleuritis or mediastinitis.

Treatment.—The first element in the treatment of pericarditis with effusion is rest. The patient should be allowed to take the most comfortable position, whether lying down or sitting up. An ice bag should be applied to the precordia, the same precautions being taken as when it is used in the treatment of dry pericarditis. It probably does diminish the inflammation to a certain extent and certainly in most instances makes the patient more comfortable. If the child objects to an ice bag, heat in some form may be used. Irritating applications externally, blisters and leeches can do no good and may do much harm by increasing the child's discomfort. It is idle to suppose that an increased number of stools, as the result of purging, or an increased excretion of urine, as the result of diuretics, can draw any of the inflammatory exudate out of the pericardium, when there is so much water in the tissues which is so much more easily reached and eliminated. Purgatives and diuretics can, therefore, do no good, but merely serve to disturb the child and diminish its chances of recovery.

When pericarditis with effusion is associated with rheumatism or the conditions which cause rheumatism, it is probably advisable to give some form of salicylic acid, preferably aspirin, in the same way in which it is given in rheumatism. When pericarditis is secondary to other diseases, there are no drugs which can have any effect on the course of the disease.

If the child is restless, bromides or some other sedative should be given. If there is pain or distress, nothing will relieve it but opium. The best form to use in these cases is morphine, which should be given in doses varying from $\frac{1}{32}$ to $\frac{1}{4}$ of a grain, according to the age and the severity of the symptoms. As always in childhood, a small dose should be given first, because so many children are susceptible to opium. The dose may be increased later, if necessary. Cold externally is safer and just as efficient in quieting the heart as aconite and similar drugs. It seems hardly reasonable to give digitalis to help the heart to do the increased work demanded of it, when the cause of the increased work can be removed by the withdrawal of the liquid.

The diet should be the same as in other acute febrile conditions in childhood. It is important to give small meals at relatively frequent intervals in order not to push up the diaphragm and thus embarrass the heart's action. It is not necessary to limit liquids.

A careful record must be kept of the area of dullness and the other physical signs in order to know whether the fluid is increasing or diminishing. In most instances it is not necessary to draw off the fluid, if it is serous, even when the effusion is considerable. The guide as to whether the pericardium should be tapped or not is not the size of the effusion,

but the symptoms produced by it. If the child is unable to lie down, is cyanotic, very restless and uncomfortable and the pulse frequent and feeble, the pericardium should be tapped. If the child is not cyanotic, not very uncomfortable and the pulse reasonably good, it should not be tapped. In every case of pericarditis with effusion, however, everything should be kept ready for the immediate tapping of the pericardium, because the fluid is liable to increase very rapidly, sometimes so rapidly as to make immediate intervention necessary after an hour or two.

There is much difference of opinion as to where the pericardium should be tapped. The most logical situation seems to be in the left chrondroxyphoid angle, as, when the needle or trocar is directed upward and a little backward, it passes through only connective tissue and enters the pericardial cavity at the bottom, where there is no possibility of wounding the heart. I have, however, never personally tapped the pericardium in this position. The needle or trocar may also be introduced in the fifth right space, not less than two centimeters to the right of the right border of the sternum. It may also be introduced in the fourth or fifth left space, just inside the border of dullness. There is no danger of hitting the internal mammary artery in this location. I have obtained fluid in all of these places. It is wiser to use a trocar than a needle. It is not advisable to introduce it deeply, partly because it is unnecessary, and partly because of the danger of wounding the heart. As a matter of fact, however, it really does not do much harm to stick a needle into the heart. It must not be forgotten, however, that sometimes, although there is a considerable amount of fluid in the pericardium and the pericardium is properly tapped, none of it will flow out through the needle. If the pericardium is tapped, it is advisable to draw off as much of the fluid as possible. It is seldom necessary to aspirate the pericardium more than once, but it may be done as often as necessary.

When the effusion is purulent, little relief can be expected from aspiration. The pericardium should be freely incised and drained. It may be necessary to resect a rib. In one instance at the Children's Hospital in which the pericarditis was secondary to pneumonia recovery was apparently hastened by the injection of bile, which is inimical to the growth of pneumococci, into the pericardial cavity.

The same care should be taken during convalescence in pericarditis with effusion as in acute endocarditis.

ADHERENT PERICARDIUM

Pericardial adhesions are usually sequelas of dry pericarditis or of pericarditis with effusion. In rare instances they may be due to a chronic tuberculous infection of the pericardium. When there has been no inflammation outside of the pericardium, the adhesions are limited to the layers of the pericardium and result simply in an obliteration of the pericardial sac. When there has also been inflammation about the pericardium in the pleura or mediastinal tissues, the pericardium may be bound down to the pleura and the chest wall as well as tied up with the mediastinal tissues. Adherent pericardium is often spoken of as chronic adhesive pericarditis. This term is an erroneous one in that there is no inflammatory process going on. That has been in the past. It is, moreover, no more reasonable to speak of the results of an acute inflammatory process as chronic than it is to speak of the loss of a leg as chronic. The only condition to which the term chronic adhesive pericarditis is at all applicable is a slowly progressive tuberculous process in the pericardium.

Symptomatology and Prognosis.—The adhesion of the layers of the pericardium to each other with obliteration of the pericardial sac usually causes little or no trouble. It does not interfere to any great extent with the work of the heart and has little or no influence on the duration of life. When the pericardium is adherent to the pleura and chest wall or tied down to the mediastinal tissues, the work of the heart is mechanically hampered and, in consequence, it has to work harder. Hypertrophy and dilatation of the heart in various degrees develop, with the symptoms due to these conditions. There are no symptoms characteristic of adhesions of the pericardium, whether they are of the layers to each other or of the pericardium to the surrounding tissues. All the symptoms are referable to secondary lesions in the heart itself and are characteristic of such lesions. The only exception is the symptom-complex seen in the peculiar form of cirrhosis of the liver which sometimes develops as the result of an adherent pericardium, especially if the pericardial condition is due to tuberculosis. In this form of cirrhosis of the liver the liver is much enlarged and the spleen moderately. Ascites develops early, always before edema of the extremities and usually without other symptoms of passive congestion. The first symptom usually noted is enlargement of the abdomen as the result of ascites. The presence of ascites in children, associated with an enlargement of the liver and spleen, without edema or other signs of passive congestion, should always suggest this condition. If there are also signs of cardiac disease, the diagnosis is almost certain. These cases always eventually die. The prognosis is also grave when the adhesions between the pericardium, pleura, chest wall and mediastinal tissues are sufficient to bring on dilatation of the heart. Death usually finally results in these cases from cardiac failure.

Physical Signs.—Simple obliteration of the pericardial sac without adhesions to the surrounding tissues causes no change in the physical signs. When there are adhesions to the surrounding tissues, a considerable number of characteristic physical signs may develop. In general, however, they are conspicuous by their absence. A systolic retraction in the neighborhood of the apex is of itself not of great importance. It must be carefully differentiated from the systolic retraction of the intercostal spaces with marked apex beat, which is found in cardiac hypertrophy. Retraction of several spaces or of the ribs and lower portion of the sternum is very characteristic and a very important sign. It may, however, be due to mediastinal adhesions alone without pericardial adhesions. The cardiac impulse is often fixed and does not vary with change in position. Broadbent's sign, a systolic retraction in the eleventh space in the left back, is very characteristic, but is seldom seen in children. The pulsus paradoxus, that is, the weakening or disappearance of the pulse at the end of inspiration during deep breathing, is an important sign. It is probably due to distortion of the large arterial branches as the thorax is broadened and the diaphragm depressed in inspiration. Diastolic collapse of the cervical veins, Friedreich's sign, is sometimes seen, but is not of great importance. A divided second sound is of no more.

When the heart enlarges from either hypertrophy or dilatation, there may be in young children bulging of the precordia. The cardiac impulse is likely to become more extensive and is often undulatory or wavy. If the dilatation of the heart is sufficient to cause a relative insufficiency at any of the orifices, the murmurs characteristic of such insufficiencies develop. A peculiar presystolic murmur has been described as rather

characteristic. I have never heard it. All the signs of passive congestion from cardiac failure may finally develop.

The Roentgen ray is often of considerable assistance in the diagnosis of adhesions of the pericardium to the surrounding tissues. It shows that the heart is bound down and immovable.

Treatment.—Nothing can be done to modify in any way the adhesions which have formed between the layers of the pericardium and between it and the surrounding tissues. Treatment must be devoted to diminishing the work of the heart, and in this way preventing hypertrophy and dilatation, and to the treatment of dilatation of the heart, if it develops.

SECTION XII

DISEASES OF THE LIVER AND GALL-BLADDER

SIZE, LOCATION AND EXAMINATION OF THE LIVER

The liver is relatively large at birth, making up from 4% to 4.5% of the body weight, while in the adult it makes up only 2.5%. At birth the liver fills about two fifths of the abdominal cavity. The left lobe is relatively slightly larger in comparison with the right at birth and during early childhood than in late childhood and adult life. This fact, however, is of no practical importance. The best position for the child when the size of the liver is being determined is lying on its back. It helps a little if the thighs are partially flexed and the knees drawn up. It helps far more, however, to attract the child's attention in some way so that it relaxes the abdominal muscles. A very good plan is for the examiner to pretend that he is trying to feel what the child ate at the last meal. It goes without saying, or should, that the examiner's hands must be warm and his finger nails short.

It is impossible to determine the upper border of the liver, because it is situated so deeply. All that can be determined is the boundary between the liver and the lung. The lower border of the lung being very thin, it is evident that percussion must be very light and that it is a line of flatness, not of dullness, which is determined. The pleximeter finger should be placed parallel to the lung-liver boundary. The upper border of the liver flatness is, on account of the slightly higher position of the diaphragm in infancy, somewhat higher at this age than later. It is at the fifth rib in the right mammillary line, at the seventh in the midaxillary and at the tenth in the scapular line. It gradually descends with the descent of the diaphragm and reaches the adult position at about six years, that is, a position about one rib lower. The large size of the liver and the wide angle of the ribs in infancy more than counterbalance the higher position of the diaphragm, so that the lower border of the liver extends below the costal border. In infancy it extends from one to three centimeters below the costal border in the mammillary line, from two to six centimeters below the tip of the ensiform and passes under the left costal border in the parasternal line. In the median line the lower border may normally extend as low as half way between the ensiform and the navel. The lower border on the extreme right may extend normally in infancy nearly to the crest of the ilium. Its position on the extreme right is very variable, however, in my experience and not of great importance. There are no accurate data as to when the adult relations are attained. The liver is, however, usually not palpable in the mammillary line after three years, although it probably may be felt normally in this position as late as eight years

It is practically impossible to percuss out the lower border of the liver in infancy and early childhood, because of the thinness of its edge. It is, on the other hand, very easy to palpate the lower border, because the abdominal wall is comparatively thin. Palpation, therefore, gives much

more accurate results at this age than percussion. If there is any discrepancy in the results obtained by palpation and percussion, those obtained by palpation should always be accepted. It is important on account of the thinness of the abdominal wall not to palpate too deeply. It is very easy to miss the edge, if too much force is used and the palpation is too deep. The lower border may be determined either by pushing the palpating fingers upward from below or by hooking them slightly over the border from above. I prefer feeling from above. Striking palpation is of very little value at this age, ordinary palpation giving far better results.

It must be remembered that the lower border of the liver may be lower than normal, even if the liver is not enlarged, in emaciated and malnourished infants, as the result of relaxation of the abdominal walls and the ligaments of the liver. It must also be remembered that the liver may be pushed down by large accumulations of fluid in the pleural cavity and by subphrenic abscesses. The left lobe may also be rotated downward when there is an effusion in the pericardium.

Gall-bladder.—The gall-bladder is small at birth. The fundus often does not extend to the margin of the liver and it is usually more embedded in the liver substance than in later life. The capacity of the gall-bladder increases rapidly during the first two years. It is, therefore, extremely difficult to determine whether the gall-bladder is enlarged or not in infancy and early childhood. It is impossible to feel it, unless it is considerably enlarged. As a matter of fact, it very seldom is enlarged at this age.

Enlargement of the Liver.—The liver is frequently enlarged, often from very slight causes, in both infancy and childhood, but more often in infancy. Enlargement from fatty infiltration in disturbances of nutrition, especially in infancy, is very common. It is not infrequently much enlarged as the result of passive congestion and sometimes as the result of amyloid degeneration. It is slightly or moderately enlarged in acute infectious, or catarrhal, jaundice. The enlargement is symmetrical in these conditions and usually also when it is due to cirrhosis or when it occurs in connection with the blood diseases. When the enlargement is due to syphilis, except in early infancy, cysts, abscesses and tumors, it is usually irregular. If, however, the syphilitic hepatitis is general, as it often is in early infancy, the enlargement is usually regular. It is also sometimes regular when it is due to a rapidly growing sarcoma. There is usually little or no enlargement in tuberculosis of the liver.

It is very important before making a diagnosis of enlargement of the liver to be certain that the apparent enlargement is really not due to the displacement of the liver. If the apparent enlargement is due to displacement and both the upper and lower borders of the liver can be determined, they are displaced upward or downward to the same degree. If the liver is enlarged, the vertical distance between the borders is increased and they are not changed symmetrically. One of the most common causes of displacement of the liver downward is laxness of the abdominal walls and of the ligaments of the liver, as the result of emaciation and malnutrition. The liver may be pushed downward by a subphrenic abscess or the flatness increased upward from an abscess. It is impossible by physical examination to determine whether the increase in the liver area is due to enlargement of the liver itself or to an abscess. This can only be determined by the symptoms. An effusion in the pleura causes flatness continuous with that of the liver. The diagnosis is usually easy, but in doubtful cases Litten's phenomenon or fluoroscopic examination will show whether there is an effusion above the diaphragm or not. A large tumor

of the right kidney may also be mistaken for an enlargement of the liver. The chief point of difference is that the kidney tumor can be felt coming forward from the loin, while an enlarged liver never extends into the loin.

Diminution in the Size of the Liver.—The size of the liver may be diminished in marked disturbances of nutrition, in the late stage of cirrhosis of the liver and in acute yellow atrophy of the liver. Acute yellow atrophy of the liver is, however, a pathologic curiosity in childhood. Children with cirrhosis usually die before contraction has taken place, and when there is marked malnutrition the liver is more likely to be enlarged from fatty infiltration than to be shrunken.

FATTY LIVER

Enlargement of the liver, whether acute or chronic, in infancy is probably more often due to fatty infiltration than to all other causes combined. It is less common in childhood, the frequency of its occurrence diminishing directly with age. It is most common in disturbances of nutrition, especially rickets, and in diseases of the digestive tract. The enlargement may be slight, moderate or extreme. In some instances the lower border of the liver reaches well below the navel. The enlargement usually develops slowly, but sometimes comes on very rapidly. I have seen the lower border come down as much as an inch in a day. The edge of the liver is rounded and its surface smooth. It feels rather softer than normal. It is not tender. There are no symptoms resulting from this fatty change in the liver. If the infant recovers, the enlargement of the liver disappears, usually rather slowly, but sometimes, especially when the enlargement has been rapid, as quickly as it came. There is no treatment.

AMYLOID DEGENERATION OF THE LIVER

Amyloid degeneration of the liver is most often due in early life to long continued suppurative disease of the bones, usually tuberculous in origin. It may also be the result of chronic suppuration anywhere, of syphilis or of chronic tuberculosis. In my experience, it is much less common now than it was twenty-five or thirty years ago, probably because of the improvement in orthopedic surgery during this period and also because of the more active treatment of syphilis.

The liver is always finally much enlarged, sometimes so much so that it almost fills the abdominal cavity, but the enlargement usually comes on very slowly. The spleen is also enlarged from the same cause and there are like changes in the kidneys and intestines. The liver feels smooth and hard and the edge is rounded. It is not tender nor painful. There is no jaundice, but almost always a very marked, waxy pallor. The urine does not contain bile; the stools do. The amyloid changes in the liver do not produce ascites or edema of the lower extremities. Both may result, however, from the pressure of the large liver on the portal vein and vena cava, or be due to associated amyloid changes in the kidneys. The only subjective symptoms are those due to the size and weight of the liver.

It is possible that, if the cause is removed, the degenerative process may cease to increase and, if the pathologic changes have not been too extensive, enough normal tissue may be left to permit life and growth. There is very little prospect, however, of any regeneration. In general, death occurs in the course of a few years as the result of asthenia, nephritis, the causative condition or some intercurrent disease.

Nothing can be done to influence the amyloid changes in the liver. Removal of the focus of suppuration or the cure of the syphilis, if that is the cause, may limit their extension.

PASSIVE CONGESTION OF THE LIVER

Enlargement of the liver as the result of passive congestion may develop slowly as the result of prolonged interference with the circulation from chronic conditions in the heart, pericardium, lungs and mediastinum or rapidly as the result of acute failure of compensation of the heart. It may also sometimes be due to some new growth at or near the hilus. When the enlargement comes on slowly, the liver is firm and smooth and the edge rounded. It is not tender. There are also evidences of passive congestion in other organs. It is very seldom that children live long enough to have the liver become small from brown atrophy, but it may diminish in size, if the cause of the passive congestion is removed. There is, of course, no jaundice. The urine does not contain bile; the stools do.

When the enlargement develops rapidly, it is almost always due to acute failure of cardiac compensation. The enlargement, especially when it is acute, may be considerable, the lower border sometimes extending well below the navel. There are often no evidences of passive congestion of other organs. The liver is usually tender and sometimes painful. The surface is smooth and firm, the edge somewhat rounded. There is no jaundice or bile in the urine, while the stools contain bile.

The treatment consists primarily in the removal of the cause. It is surprising how rapidly the liver diminishes in size, if acutely enlarged, when compensation is restored. Free catharsis is also sometimes of benefit.

TUBERCULOSIS OF THE LIVER

The liver may be studded, more or less thickly, with miliary tubercles in acute miliary tuberculosis. It may also contain an occasional tuberculous mass, varying in size from that of a pea to that of an English walnut, in chronic diffuse tuberculosis. In neither instance, however, is the liver perceptibly enlarged. There are no symptoms unless one of the tuberculous masses in the chronic diffuse form happens to compress a large vein or duct, when there may be enlargement of a part of the liver or slight jaundice and a little bile in the urine. The involvement of the liver has no affect on the prognosis and there is, of course, no treatment.

SYPHILIS OF THE LIVER

All statistics show that the liver is the seat of election for the lesions of hereditary syphilis. Nevertheless, it is in only a relatively small proportion of cases that the changes in the liver are sufficient to cause enlargement enough to be recognizable clinically. When the infection is severe, however, the enlargement may be considerable. It is always accompanied by enlargement of the spleen. The pathologic process at birth is usually a diffuse, interstitial hepatitis without much induration or contraction of connective tissue. The liver is, therefore, large, smooth and not much increased in density. If cirrhotic changes have developed before birth or develop later, the liver becomes hard and the surface and edge irregular. Icterus is inconstant and usually not marked. The urine does not contain bile, while the stools do. Any symptoms which might possibly be caused by the disease of the liver are so obscured by those of the general dyscrasia that they are unrecognizable. Death

is quite likely to ensue when the liver and spleen are much enlarged at birth or become so soon after, because their enlargement indicates a severe infection.

In other instances there may be many miliary gummata scattered throughout the liver and sometimes, usually in childhood, larger gummata are formed here and there as in the adult. There is usually not much enlargement of the liver in these cases, but, if the larger gummata are on the anterior surface of the liver, they may sometimes be felt.

Lesser degrees of congenital syphilitic hepatitis may not show any symptoms, but later develop the picture of ordinary cirrhosis. Many of these cases of late and slowly developing syphilis of the liver show an unusual or obscure clinical course. They usually develop between the ages of six and thirteen years. Digestive disturbances are likely to be marked, but icterus is not common. There is little to distinguish them from cases of cirrhosis due to other causes, however, and the diagnosis must depend on the Wassermann test.

There is, of course, no special treatment for syphilis of the liver. The general infection is to be treated in the usual way and symptoms met as they arise.

ACUTE YELLOW ATROPHY OF THE LIVER

As far as I know, I have never seen a case of acute yellow atrophy of the liver in a child, although I have several times suspected it. As the children whom I thought might have it all recovered, my suspicions must have been wrong. I am inclined to believe, therefore, that it must be a very rare occurrence in childhood.

CYSTS OF THE LIVER

Hydatid cysts of the liver in children must necessarily be very uncommon in this country and, if they occur, are almost certain to have originated elsewhere. Cysts from other causes, usually congenital, may occur. A cyst may be suspected, if a fluctuating tumor without any evidences of suppuration is detected on the surface of the liver. The chance of a cyst being in this position is, however, most remote.

ABSCESS OF THE LIVER

Suppurative hepatitis in the new-born is almost invariably secondary to infection through the navel. Numerous very small abscesses may be formed, but death almost invariably ensues before there is time for the formation of a large abscess. There may be some enlargement of the liver and perhaps some tenderness over it. Sometimes there is jaundice. Usually there is nothing to prove that there is suppuration in the liver. It can be suspected, however, if there are symptoms of a serious suppurative focus and either a history of an infection at or near the navel or the evidences of such an infection. The only treatment is preventive by proper care to prevent infection of the navel during or after birth. If suppurative hepatitis has developed, nothing can be done.

Abscess of the liver occurs in childhood from the same causes as in adult life; namely, infections in the abdominal cavity which can reach the liver through the portal vein and its branches. As in adults, appendicitis is the most common cause. It is more likely to be due in them than in adults to the breaking down of mesenteric and retroperitoneal lymph nodes. Abscess has also been the result in children of the entrance of round worms into the hepatic duct. It is almost never a sequela of

infection of the colon, unless it is amoebic in origin. The abscess is usually single and in the right lobe. Abscess of the liver is uncommon at any age, and far more uncommon in the child than in the adult.

There is nothing characteristic about the symptoms and physical signs in childhood, except that the child is less likely or less able to locate the seat of its pain accurately.

Diagnosis.—Even though it may be quite evident that there is a pocket of pus in the neighborhood of the liver, it is often very difficult to determine whether it is in the liver, between the liver and the diaphragm or above the diaphragm, that is, whether there is an abscess of the liver, a subphrenic abscess, an empyema or an abscess of the lung. A history of an inflammatory process below the diaphragm is in favor of an abscess of the liver or a *subphrenic abscess*, and of one above it in favor of an empyema or an abscess of the lung. The history is not conclusive, however, as an abscess of the liver may be secondary to such distant infections as otitis media and a subphrenic abscess due to the perforation of the diaphragm by an empyema or an abscess of the lung. Displacement of the heart and mediastinum is very strong evidence in favor of an empyema or an abscess of the lung. Its absence, however, does not rule them out. Loud respiratory and voice sounds, especially if of normal character, below the level of dullness, are in favor of an abscess below the diaphragm. So also are a good respiratory movement on the right side and the persistence of Litten's phenomenon, that is, of the moving shadow visible in the tenth and eleventh spaces during deep respiration, when the light falls on the patient from the front and cross lights are excluded. Fluoroscopic examination with the Roentgen ray, however, gives the best evidence regarding the location of the pus and is usually conclusive. A prominence over the anterior or inferior surfaces of the liver is also almost positive evidence that the abscess is in the liver. It is almost impossible to differentiate between an abscess of the liver and a subphrenic abscess, in many instances, unless with the aid of the fluoroscope. Fortunately, it is unimportant to do so, because the treatment of both conditions is the same.

Prognosis and Treatment.—The treatment is surgical. The chances are considerably against recovery in both subphrenic abscess and abscess of the liver, but not quite so bad in abscess of the liver.

MALIGNANT DISEASE OF THE LIVER

Malignant disease of the liver is very uncommon in early life, but may occur, even in infancy. It is almost always secondary to malignant disease elsewhere, usually in the kidneys or suprarenal capsules, but may in rare instances be primary. It is usually some form of sarcoma. It may start in a single or several foci or be diffusely distributed. When there are one or more foci, the growth of the tumor is relatively slow, but when the process is diffuse, it may be very rapid, even as much as an inch a day. When there are one or more foci, the liver is irregularly enlarged, hard and bunchy. When the process is diffuse, the enlargement is regular, the surface smooth and the edge slightly rounded. The liver is seldom tender.

In general, there are no symptoms except those due to the size and weight of the tumor and those of cachexia. In certain cases, however, as the result of compression of some of the bile ducts or veins, there may be a little jaundice, bile in the urine and evidences of portal stasis.

The prognosis is hopeless. The course is usually a few months, but in the diffuse form it may be only two or three weeks. There is no treatment.

CIRRHOSIS OF THE LIVER

This condition never occurs in infancy, except in connection with congenital malformations of the bile ducts and as a manifestation of syphilis. It is uncommon in childhood, but not as uncommon as is usually supposed. It is seen most frequently between the ages of nine and twelve years. I have never seen a case of Hanot's hypertrophic cirrhosis with chronic icterus in a child, although a number of cases have been reported. Cirrhosis with enlargement of the liver is, however, much more common in childhood than cirrhosis with diminution in its size. The size of the liver depends, of course, on whether the newly formed connective tissue contracts or not. The pathologic process is the same whether the liver is large or small. It is possible that the vitality of the hepatic cell in the child may have something to do with the frequency of cirrhosis with enlargement in childhood, atrophy of the cell occurring less rapidly than in adult life. It is also possible that children may succumb to the disease sooner than adults, before there has been time for atrophic changes to develop.

Etiology.—Excluding the cases of cirrhosis due to congenital syphilis, which have already been considered, alcohol is unquestionably the most common cause of cirrhosis of the liver in childhood. It is not necessary that large amounts be taken. My own experience and that of others shows that very small amounts, if taken regularly by children, may produce cirrhosis. Great care should be exercised, therefore, not to prescribe alcohol over long periods and to be certain that, if it is ordered, it is also stopped. In several cases which I have seen the alcohol was ordered by a physician, who forgot to stop it, and consequently it was given continuously for years.

It is probable that the products of abnormal fermentation or decomposition in the digestive tract are a more frequent cause of cirrhosis in early than in later life. It is also probable that the products of bacterial growth in the intestines or elsewhere are more harmful at this age. The cases which have apparently followed overfeeding are presumably due to this cause. It is unreasonable to suppose that the hepatic lesions so common in the course of the eruptive fevers have anything to do with the etiology of cirrhosis. The acute toxic infections, even if severe, leave but few traces in the liver; at most, small cicatricial areas or scars, which do not deserve the name of cirrhosis. Moreover, if the eruptive diseases were the cause of cirrhosis, it would be a far more common condition than it is. The form of cirrhosis due to adherent pericardium has been considered under adherent pericardium. Cirrhosis in childhood is almost never secondary to lesions of the gall bladder or larger bile ducts.

Symptomatology.—There is nothing characteristic about the symptomatology of cirrhosis of the liver in childhood. There is the same variability in the relative severity and order of development of the symptoms as in adults, depending on the location of the newformed tissue and the pressure which it exerts. My own limited experience leads me to believe that jaundice and enlargement of the abdominal veins is less common and less marked than in adults, and that ascites is a more common and prominent symptom.

Diagnosis.—When the liver is enlarged, as it usually is, only those conditions need be considered in the differential diagnosis in which the

spleen is also enlarged. Malignant disease and fatty infiltration of the liver may thus be excluded. If the enlargement is due to passive congestion, the cause can be found in the chest. If it is due to amyloid degeneration, the cause of the degeneration is obvious. In Banti's disease, the enlargement of the spleen precedes for a long time that of the liver. The course is longer and the anemia more marked. Leukemia is a more acute disease in early life and the blood shows the characteristic changes.

When the liver is not enlarged or smaller than normal, Banti's disease in a very late stage may be considered, but can be excluded as before. It is conceivable that, if there is ascites, especially if it is an early symptom, as in the type of cirrhosis due to adhesions in the pericardium and mediastinum, there may be some confusion between cirrhosis of the liver and tuberculous peritonitis. The spleen is not enlarged in tuberculous peritonitis, however. There is no jaundice and, more than all, the fluid in cirrhosis is a transudate and in tuberculous peritonitis an exudate. There are often also tuberculous masses to be felt in the abdominal cavity in tuberculous peritonitis.

If the diagnosis of cirrhosis of the liver is made, it is important to determine whether it is syphilitic or not by looking for other evidences of syphilis and by the Wassermann test, because in many instances much can be done for the patient by proper treatment, if it is syphilitic. It is of interest also, but not of great practical importance, to determine whether it is secondary to adhesions in the pericardium or mediastinum or not.

Prognosis.—The prognosis is, of course, hopeless, except in syphilitic cirrhosis, in which, if too much injury has not been done, the process may be arrested by proper treatment. The course is usually shorter than in adults.

Treatment.—The most important treatment is preventive. Alcohol should not be given to children, even in small amounts, over long periods of time. Physicians must be sure to stop alcohol, if they have ordered it, when the supposed need for it has passed. The prevention and proper treatment of syphilis, tuberculosis and chronic disturbances of digestion will also diminish the number of cases of cirrhosis. When cirrhosis of the liver has developed, nothing can be done to stop it, unless it is syphilitic. Symptoms should be treated as they arise. The abdomen should be tapped to relieve ascites as often as is necessary. Little or nothing can be expected from Talma's operation.

ACUTE INFECTIOUS (CATARRHAL) JAUNDICE

This very common disease of early and middle childhood has been described under many names and attributed to many causes. The recent work of Jones and Minot (Boston Medical and Surgical Journal, 1923, CLXXXIX, 531) apparently shows quite conclusively that it is not due to plugging of the common duct with mucus or to the swelling of the mucosa as the result of an ascending infection from the duodenum, but to an infection involving the entire biliary tract and the liver parenchyma. This condition differs materially from the specific infectious disease due to the spirochete icterohemorrhagica and must not be confused with it. The etiology of the common catarrhal or infectious jaundice is unknown, but it is probable that the causative organism is not always the same. As it often follows diseases of the respiratory tract, it seems probable that the same organisms may cause both. It may occur in epidemics, usually

small, or may be sporadic. It is possible, however, that some of the apparently sporadic cases may be part of a mild epidemic, as there is certain evidence to show that the incubation period may be several weeks in length.

Symptomatology.—The symptoms not infrequently develop during the latter part of, or soon after, some respiratory infection. In other instances they follow quite quickly chilling or exposure of some sort. In others they come on almost immediately after some marked indiscretion in diet or develop slowly after a considerable period of overindulgence. In still others there is no apparent cause. The onset and severity of the symptoms is so varied that is is very difficult to describe them. It is easier to understand their variability when it is remembered that they are the result of four factors; a general infectious process, a disturbance of the function of the liver cells, the entire or partial exclusion of bile from the intestine and the retention of bile in the circulation. The symptoms must necessarily vary according to the predominence of one or more of these factors in the individual case. As a rule, there are several days or a week or more in which the symptoms are quite indefinite. They are malaise, irritability, loss of appetite, slight headache, coated tongue, epigastric discomfort and often slight fever. More or less marked jaundice then develops, the temperature is a little higher, usually under 102° F. The stools become clay colored and bile appears in the urine. The liver is a little enlarged and often tender. The spleen is also often somewhat enlarged. In other instances the onset is more acute with a higher temperature and the vomiting of mucus and bile. In such cases the jaundice usually appears more quickly. In these cases it is probable that the infection comes directly from the digestive tract. The vomiting of bile is also not uncommon in the cases which come on less acutely.

The duration of the symptoms is very indefinite. The average is probably between one and two weeks. The jaundice may persist for some days or even many weeks after the cessation of other symptoms and long after the appearance of bile in the stools. In fact, jaundice may be present in many instances when the stools have always been normal or even darker than normal in color. The continuous presence of bile in the stools in some cases in which there is marked jaundice and the persistence of jaundice for a considerable time after the reappearance of bile in the stools are conclusive evidence that the jaundice is due to a disturbance of the function of the liver cells as well as to obstruction in the finer bile ducts. The bile usually ceases to appear in the urine before the jaundice has faded. In some cases the urine may contain albumin, cells and casts.

Convalescence is usually slow and in some cases, in which the damage to the liver cells has been considerable, may be prolonged several months. Recurrences are not uncommon. The termination is always in recovery.

The blood shows a considerable reduction in the number of red cells and hemoglobin, which is most marked soon after the height of the jaundice. A loss of 1,000,000 red cells is not infrequent. The blood platelets increase as the jaundice decreases. The white count increases at first, but drops to below normal as the jaundice diminishes, returning to normal in a few days or weeks. The polynuclear neutrophiles are normal or increased at first, but as the total number of white cells diminishes, there is an absolute rise in the number of mononuclear cells, many of which are immature and abnormal.

Diagnosis.—The picture of acute catarrhal jaundice, when the symptoms have developed, is so characteristic that it is almost impossible to

mistake it for anything else. It is probable, however, that it is often overlooked when the more definite symptoms of jaundice, bile in the urine and clay colored stools, are not present and is mistaken for a simple disturbance of the digestion. It is possible also to mistake it, if it occurs in early infancy, as it occasionally does, for a congenital malformation of the bile ducts. The differential diagnosis between these two conditions has been taken up in discussing the latter condition. It is also conceivable that if the patient is seen late, when the only remaining symptom is jaundice, that it may be confused with congenital hemolytic jaundice. The history of the two diseases is absolutely different, however. The spleen is considerably enlarged in congenital hemolytic jaundice and but little, if any, in acute infectious jaundice. There is, moreover, marked fragility of the red cells in congenital hemolytic jaundice and none in acute infectious jaundice. Inflammation of the gall bladder and obstruction of the large bile ducts from gall stones are so uncommon in childhood that they hardly need to be considered. In these conditions there is almost always a history of pain and marked tenderness in the region of the gall bladder, while the gall bladder is usually palpable, none of which conditions are present in acute infectious jaundice.

Treatment.—Children ill with acute catarrhal jaundice should be put to bed and kept there until they are thoroughly convalescent. While theoretically there is no special reason why they should, practically there is no doubt that exposure to cold and overexertion increase the severity of the disease and bring on recurrences during convalescence. The peevishness, irritability and fretfulness of children with this disease must be treated charitably and due allowances made for them, because these symptoms are characteristic of the disease. They are probably due to the presence of bile in the circulation.

When the amount of bile entering the intestines is diminished, the power of digesting and utilizing fat is diminished. The fat in the food should, therefore, be much diminished or entirely cut out. Theoretically, the digestion of the carbohydrates ought not to be impaired, unless the pancreas is also involved. Practically, however, sugars of all sorts increase the symptoms and, except in mild cases, even the starches are poorly taken care of. The diet, therefore, should be made up mainly of easily digestible proteins.

When there is much vomiting, it is advisable to stop food entirely for twenty-four or forty-eight hours. The best foods to give in beginning to feed these severe cases are clear broths, albumen water and whey, remembering, of course, that the nutritive value of clear broths and albumen water is very slight. A little later, fat free milk or skimmed milk may be given. The digestion of the casein may be made easier by boiling or peptonizing or by the addition of 25% of lime water or of two grains of citrate of soda to the ounce of milk. The milk may also be given in the form of junket or plain blanc mange. At this stage it is also often safe to give orange juice and perhaps to give simple gruels or a little simple cereal, such as farina or rice. The next additions to the diet are meat, zwiebach, toast and crackers. Strained green vegetables can usually be given safely before the yolks of eggs or considerable amounts of starch. Great care must be exercised in adding fats and sugars to the diet during convalescence, as it is very easy to bring on a recurrence by even a slight indiscretion. It goes without saying that in mild cases it is not necessary to restrict the diet so much in the begin-

ning and that the liberality of the diet must necessarily depend considerably on the age of the individual child.

It is advisable to give considerable amounts of water, unless the child is vomiting. Alkaline waters are no more efficacious than plain water.

There are no drugs which can influence in any way the pathologic process in the liver. It is useless to prescribe the so-called cholagogues, even calomel, because, in the first place, none of these drugs increase the secretion of bile. In the next place, it is illogical to increase the secretion of bile in this condition and, finally, it is undesirable, even if it is possible, to stimulate an inflamed organ. It is possible that the administration of the bile salts by the mouth may make the digestion of fat easier. The digestion of fat is made still easier, however, if no fat is given to digest. Furthermore, bile salts given by the mouth tend to increase the secretion of bile by the liver, something which it is undesirable to do in this condition.

The administration of bicarbonate of soda in many instances seems to diminish the vomiting. A level teaspoonful should be mixed with six ounces of water and one or two teaspoonfuls of this mixture given every ten or fifteen minutes. In some instances the administration of one or two heaping teaspoonfuls of bicarbonate of soda in a glass of warm water, by causing vomiting, will wash out the stomach and diminish the vomiting.

It is advisable to keep the bowels of children, ill with this disease, a little relaxed. The best drugs for this purpose are mild salines, such as milk of magnesia or phosphate of soda. These often work better if they are given in repeated small doses rather than in a single large dose.

Hot applications to the abdomen, such as a poultice, a hot water bottle or an electric heating pad, not infrequently diminish the abdominal discomfort and sometimes seem to help the vomiting.

It has seemed to me that the appetite has come back more quickly and that convalescence has been somewhat more rapid when the tincture of nux vomica is given after the end of the acute stage. It should be given in doses of one drop for each year of age, three times daily before meals. It should be given diluted with a small amount of water and should not be prescribed in a mixture with syrups. Children will take it without much objection, if they understand that the person who gives it to them intends them to take it. If anemia is marked, it is well to give some form of iron. The saccharated oxide—Eisenzucker—is a good preparation and should be given in doses of from three to five grains, three times daily, after meals.

DISEASES OF THE GALL-BLADDER AND CHOLELITHIASIS

Diseases of the gall-bladder and gall-stones are extremely rare in infancy and childhood, although they may exceptionally occur at any age. They are so uncommon that it is ordinarily unnecessary to consider them in differential diagnosis. It must not be forgotten, however, that they are possibilities. The symptomatology, prognosis and treatment of these conditions is the same in childhood as in later life.

SECTION XIII

DISEASES OF THE SPLEEN

SIZE, LOCATION AND EXAMINATION OF THE SPLEEN

The position of the spleen is the same at all ages. It lies between the ninth and eleventh ribs on the left side. The anterior border does not normally extend beyond the costo-articular line, that is, the line drawn between the left sterno-clavicular articulation and the tip of the eleventh left rib. During the first few years of life the spleen is seldom more than five centimeters long and three centimeters wide, being smaller more often than larger, while its thickness varies between one half a centimeter and one and one half centimeters. The determination of the area of splenic dullness by percussion during the first few years of life must necessarily, on account of its small size, be extremely difficult. Personally, I do not believe that it is possible. The normal spleen is not palpable unless the abdominal wall is unusually thin and lax. It is easily palpable, however, both in infancy and childhood, if enlarged, even if the enlargement is slight. It is wiser, therefore, to trust to palpation than to percussion during infancy and early childhood. It is safe to conclude that if the spleen is palpable, it is enlarged, and that, if it is not palpable, it is normal. It is easier to feel the spleen by curving the fingers over the costal border from above than by pushing up from below. It is also easier to feel it when the child lies on its right side with the legs flexed than when it is lying on its back. The abdominal wall must, of course, be relaxed. It never is, if the child is frightened, examined roughly or with cold fingers. It is also important not to press in too deeply while palpating, because, on account of the thinness of the abdominal wall and the superficial position of the spleen, it is very easy to push it out of the way by deep palpation. It must also not be forgotten that, if the spleen is much enlarged and the physician examines only close to the ribs, it may be missed.

ENLARGEMENT OF THE SPLEEN

Enlargement of the spleen, often considerable, is very common in early life and not infrequently develops, moreover, as the result of apparently very insignificant causes. It is, therefore, of much less importance than in later life. When the spleen begins to enlarge, it is evident first at the costal border in the line of the tenth rib. When the enlargement is very marked, it is usually somewhat more downward than forward. Sometimes the enlargement is so great that the spleen fills the whole of the left half of the abdomen and may even extend beyond the median line in the neighborhood of the navel. It never entirely fills the flank.

Diagnosis.—It is sometimes difficult to distinguish between an enlargement of the spleen and a tumor of the left kidney. An enlarged spleen is always superficial and never entirely fills the flank. An enlarged kidney is deep down, comes from the flank and can always be felt there.

A large spleen bulges the abdomen. A large kidney bulges the flank. An enlarged spleen is somewhat movable laterally. An enlarged kidney is movable, if at all, anteroposteriorly. An enlarged spleen has a definite edge, which is often notched. An enlarged kidney has no edge and, of course, no notch. An enlarged spleen is always smooth, unless syphilitic in origin, while an enlarged kidney is often irregular in shape. It is also sometimes difficult to distinguish between an enlargement of the spleen and a rolled up tuberculous omentum or a mass of tuberculous mesenteric glands. Confusion arises, of course, only when the tuberculous mass happens to be situated in the region of the spleen. The tuberculous mass seldom has the exact shape of the spleen, is not likely to have as smooth a surface and seldom has as distinct an edge. It may or may not be more freely movable. If it is a mass of glands, it evidently originates deeper down.

Acute Enlargement.—Enlargement of the spleen may be either acute or chronic. The spleen may enlarge quickly in any of the acute infections. There is seldom much enlargement, however, except in typhoid fever and malaria. When the enlargement of the spleen is acute, the spleen is soft and the edge rounded. It is usually hard to distinguish the notch. The size may vary from day to day. The enlargement is due chiefly to hyperemia but in part to hyperplasia of the pulp.

In Rickets and Disturbances of Nutrition.—Chronic enlargement of the spleen in infancy is very common in connection with rickets and disturbances of nutrition. All are often associated with anemia. There is, however, no constant relation between the severity of the rickets, the degree of the anemia and the size of the spleen. All possible combinations may be met. The spleen may be only a little or much enlarged. It is hard and smooth, the edge is rounded and the notch distinct. It usually decreases in size rather slowly, but, even if the enlargement is very marked, it finally disappears entirely. The enlargement is due partly to hyperemia, but mostly to hyperplasia of the pulp. There is sometimes a little overgrowth of the interstitial tissue.

In Secondary Anemia.—The spleen is often enlarged in connection with secondary anemia. The size of the spleen does not vary directly with the severity of the anemia or the degree of leucocytosis. All possible combinations may be found. There is also no constant relation between the size of the spleen and that of the liver or of the lymph nodes, both of which are also often enlarged. The enlargement may be slight or very marked. The surface of the spleen is hard and smooth, the edge rounded, the notch distinct. The enlargement usually disappears slowly, but sometimes may diminish very rapidly. I have known a spleen which filled half of the abdomen to disappear entirely in three months. In rare instances, however, the enlargement persists into adult life. The enlargement is due principally to hyperplasia of the pulp, but partly to overgrowth of the interstitial tissue.

In Blood Diseases.—The spleen is also enlarged in connection with leukemia, pseudoleukemia and many of the severe primary anemias, especially if they are of the aplastic type. The pathologic condition responsible for the enlargement varies with the type of blood disease in the individual instance.

From Chronic Passive Congestion.—Chronic enlargement of the spleen occurs in connection with enlargement of the liver in chronic passive congestion, resulting from diseases of the heart and lungs. The enlargement is relatively not so great in most cases as is that of the liver. It is

relatively not as common or as marked as under similar conditions in adults.

In Syphilis.—The spleen is frequently enlarged, especially in infancy, in congenital syphilis. The liver is also always enlarged. Enlargement of the spleen without enlargement of the liver does not justify a diagnosis of syphilis. There are usually other evidences of syphilis present. The enlargement is due almost entirely to the overgrowth of interstitial tissue, but sometimes to gummata of various sizes. The spleen is hard. The surface may be either smooth or irregular; the edge may be sharp, rounded or irregular.

Amyloid.—Amyloid degeneration of the spleen occurs from the same causes and under the same conditions as amyloid degeneration of the liver. The spleen is hard and smooth and the edge is rounded. The notch is usually distinct.

In Malaria.—The spleen is constantly enlarged in chronic malarial infections. The enlargement may be very great. The spleen is hard, the surface smooth, the edge fairly sharp, the notch distinct. In chronic cases the diminution in the size of the spleen is usually rather slow. The enlargement may persist for a long time after the cure of the malarial infection.

In Tuberculosis.—There may be miliary tubercles in the spleen in acute miliary tuberculosis, and foci of tuberculous tissue, varying in size from that of a pea to that of a marble, in chronic diffuse tuberculosis in the spleen as well as in the liver. In my experience, however, enlargement of the spleen as the result of tuberculous infection, is very uncommon and never marked.

From Malignant Disease.—Enlargement of the spleen from malignant disease is a pathologic curiosity. I have never seen an instance.

There are also two special types of splenomegaly of obscure origin but with fairly characteristic symptomatology, which are worthy of separate description. These two diseases are usually spoken of as Banti's disease and Gaucher's disease. Another condition, in which the spleen is enlarged, which is described by various authors under diseases of the spleen, of the liver and of the blood, is familial, chronic hemolytic jaundice, or congenital acholuric jaundice. I shall take it up under diseases of the blood.

BANTI'S DISEASE

The onset of Banti's disease is rarely before the tenth year and usually later. It is really a disease of adolescence or early adult life rather than of childhood. The course is chronic and may be divided on the symptomatology into three stages.

Symptomatology.—The first, or preascitic stage, lasts several years. It is characterized by gradually increasing weakness and pallor, which are usually finally associated with digestive disturbances and abdominal pain. The large, smooth, hard spleen is often discovered because of these latter symptoms. There is a tendency to hemorrhages into the skin and mucous membranes. Epistaxis is not uncommon. Anemia of the chlorotic type gradually develops, but is never extreme. There is a slight or moderate leukopenia. There is an increase in the urobilin of the stools and urine, which points to increased blood destruction as the cause of the anemia. The resistance of the blood cells is normal and the blood shows few or no evidences of an increase in the activity of the bone marrow.

The second, or intermediate stage, is short, lasting only a few months. The disturbances of digestion are more marked and the symptoms due to the anemia increased. The urine is scanty and high colored and contains a more marked excess of urobilin. The liver also increases in size.

The third stage is characterized by the addition of the signs and symptoms of cirrhosis of the liver, especially painless ascites and slight jaundice, both of which may be intermittent. The liver may diminish in size as time goes on. The anemia increases, as does the tendency to hemorrhage. Emaciation becomes more marked and death occurs in a few years, either as the result of hemorrhage or from some intercurrent disease.

Etiology and Pathology.—The etiology is unknown. It is not unreasonable to suppose that some substance, either itself a toxin or capable of being changed by the splenic metabolism into a splenotoxin, is brought to the spleen through the splenic artery, because the earliest changes are found in the neighborhood of the follicular arteries. This substance is probably hematogenous rather than hepatogenous in origin, because the pathologic changes in the spleen precede those in the liver. The improvement that follows splenectomy, if it is done reasonably early, also indicates that the changes in the spleen are responsible for those in the liver.

The enlargement of the spleen is the result of a chronic inflammatory process. The result of this is an increase in the functional activity of the spleen, which causes increased hemolysis and, in consequence, anemia. There is nothing distinctive in the pathologic changes in the spleen. There is an increase of fibrous tissue in the capsule and reticulum, involving both the pulp and the follicles. The follicles may be hyperplastic in the early stages, but in the later are small and scarce. There are usually evidences of increased blood destruction. The changes in the liver are those of ordinary periportal cirrhosis.

Diagnosis.—If tempted to make a diagnosis of Banti's disease, it is wise to remember that there are many causes for enlargement of the spleen, that enlargement of the spleen is accompanied by change in the size of the liver and anemia in many other conditions beside Banti's disease, that Banti's disease is very rare, that it occurs only in late childhood and that, therefore, this diagnosis is probably wrong. If the enlargement of the spleen is due to any of the severe primary anemias or leukemia, the duration of the disease is shorter and the blood shows the characteristic changes of the disease. The causes of the enlargement are evident in chronic passive congestion and amyloid degeneration. The history is different in malaria and plasmodia can usually be found in the blood. Enlargement of the spleen from congenital syphilis rarely develops as late, the liver is usually larger, other signs of syphilis are almost always present and the blood shows a positive Wassermann test. In congenital acholuric jaundice, jaundice is present from or soon after birth, the condition is familial, the general health is but little impaired, there is no ascites, and the resistance of the red cells is diminished. Gaucher's disease begins earlier, is familial, the impairment of the health is less, the skin is brownish, there is sometimes a peculiar discoloration of the conjunctivae and the spleen is much larger.

Treatment.—There is no medical treatment which has any effect on the course of the disease. Medical treatment can be only symptomatic and palliative. The removal of the spleen, however, is of benefit, even if done late in the course of the disease. When it is done during the first stage, great improvement in the symptoms always results. It is possible

that complete cure may be obtained, but sufficient time has not elapsed to show whether cure ensues or not. The earlier the operation, the better is the prognosis, because in the third stage, when the spleen has become largely fibrotic and cirrhotic changes have occurred in the liver, return to the normal cannot be expected. The mortality from the operation is between 10 and 15%. It is, of course, smaller when the operation is done early.

GAUCHER'S DISEASE

This disease, also known as large-celled splenomegaly, is very rare, less than twenty authentic cases being on record. I have seen several children whom I thought had this disease, but the diagnosis was not proved by autopsy in any of them. It is often familial, but is not hereditary. It begins insidiously in infancy or early childhood, always before the thirteenth year. The course is long, averaging twenty years. Death is due to some intercurrent infection.

Symptomatology.—The first and most prominent feature is an increase in the size of the spleen. The enlargement progresses steadily and finally becomes enormous. It may for a long time be the only evidence of disease, and the discomfort resulting from its weight is often the first symptom noticed. Moderate anemia of the chlorotic type develops after a time. The average number of red cells is about 3,500,000. There is also a definite leukopenia, but no change in the relative proportions of the white cells. After a time there is also a brownish discoloration of the skin, most marked on the face, and a tendency towards submucous and subcutaneous hemorrhages. In some cases there is a peculiar yellowish, wedge-shaped thickening of the conjunctivae on both sides of the corneae. Enlargement of the liver also develops after a time, but is never extreme. Jaundice and ascites are rare and there is no enlargement of the superficial lymph nodes.

Etiology and Pathology.—The etiology of this disease is unknown. The findings at autopsy are, however, pathognomonic, characteristic large vesicular cells with small, eccentric nuclei and slightly granular cytoplasm are found in the spleen, liver, bone marrow and lymph nodes. The enlargement of the spleen and liver is due to these cells, which block the sinuses of the spleen and lymph nodes and are crowded about the liver lobules.

Diagnosis.—Although the diagnosis is plain enough at autopsy, it is very difficult during life, especially in the early stages. A slowly progressive increase in the size of the spleen without any evident cause and with no symptoms, except, perhaps, a slight secondary anemia with leukopenia, should suggest it as a possibility. The presence of a similar condition in another member of the family makes it more probable. A brownish discoloration of the skin is evidence in favor of it. The enlargement of the spleen in proportion to that of the liver is relatively greater, moreover, than in other similar conditions. If the peculiar, wedge-shaped thickening of the conjunctivae is present, the diagnosis of Gaucher's disease is justified.

Treatment.—There is no medical treatment which has any effect on the course of the disease. Splenectomy cannot be expected to cure this disease, because the liver, bone marrow and lymph nodes are also involved. It has been done in a number of cases with an immediate mortality of about 33%. Those that survived have apparently been benefited by the operation, but sufficient time has not elapsed to determine how long the improvement may last or whether recovery may ensue.

SECTION XIV

DISEASES OF THE BLOOD

PHYSIOLOGY AND PATHOLOGY OF THE BLOOD AND BLOOD-FORMING ORGANS IN EARLY LIFE

The Blood and Its Formation in the Embryo

During the first half of fetal life the liver is the chief seat of production of the corpuscles. The bone marrow, which begins to develop in the third month, does not become of much importance in corpuscle formation until late in uterine life. As the productive power of the bone marrow increases, production in the liver diminishes and has practically ceased at birth. The formation of blood corpuscles in the liver and bone marrow is supplemented for a time by production in the spleen and to a limited extent in foci in other places. The activity of the lymphatic glands and other lymphatic tissues is not marked until well after the middle of fetal life.

In accordance with the location of the productive areas and their peculiar characteristics, the first corpuscles are entirely red and are nucleated. As the bone marrow begins to produce, myelocytes, and, a little later, leucocytes, appear in the blood, as well as some platelets. The red corpuscles are basophilic or polychromatophilic. Lymphocytes are for a time relatively few, but during the latter months they predominate. At birth blood production is essentially the same as in the adult, but many of the red corpuscles are basophilic or polychromatophilic. There are a few nucleated red corpuscles and an occasional myeloblast or myelocyte. The marrow of the long bones, as well as that of the bones of the skull, is red at birth. The change toward the fat marrow of the adult begins at about four years, is well marked at seven years, and complete at fourteen years.

The Blood and Its Formation during Childhood

There is much difference of opinion as to whether during childhood the red and white cells originate from a common ancestral cell in the bone marrow or from different cells. For clinical purposes it makes very little difference. In all probability, however, they do not have a common ancestor. The red corpuscles are derived from large nucleated red cells, megaloblasts, through small nucleated cells, normoblasts. During their development the red cells are more or less reticulated and are basophilic or polychromatophilic.

The polynuclear, granular leucocytes are derived primarily from large nongranular mononuclear cells, myeloblasts. These cells gradually become granular and when fully developed are known as myelocytes. These myelocytes pass through several stages in which they become smaller and the nucleus becomes more and more irregular. When they are fully developed, they are the polynuclear, neutrophilic leucocytes. The granulations in the myelocyte, and in all the stages to the completely developed polynuclear cell, may be eosinophilic.

The blood platelets are detached portions of the cytoplasm of another kind of special marrow cell, the giant cell, or megakaryocyte.

The lymphocytes originate in the Malpighian corpuscles of the spleen, the follicles of the lymph nodes and intestinal lymphatic structures, and in isolated groups of lymphoid cells scattered throughout the body. It is possible that some of them may be derived from lymphoid tissue in the bone marrow.

The large mononuclear lymphocytes and transition forms are of many varieties and often are hard for anyone but an expert to distinguish. Normally, many of them are probably endothelial in origin. Others represent steps in the formation of the myelocytes and, perhaps, of the lymphocytes. The large mononuclear cells, seen in acute lymphatic leukemia, are probably derived from the bone marrow.

The blood vessels of the bone marrow are considerably larger in calibre than the capillaries and have a very thin wall, which is made up of endothelial cells and the membranous extensions of their cytoplasm. The cells of the marrow parenchyma are crowded closely against this wall. Under normal conditions this wall acts as an effective barrier against the entrance of immature marrow cells into the circulating blood, but allows mature cells to pass. The polynuclear leucocytes are able to pass through by virtue of their ameboid activity. The continued multiplication of the red corpuscles in a restricted space increases the pressure to such an extent that they are forced through. The nuclei of the immature cells prevent their passage. The megakaryocytes pass wholly or partially through the wall as the result of their ameboid motion and the platelets are broken off in the capillaries. When the barrier is broken down the immature cells can pass freely into the circulating blood.

Blood Destruction

It is variously estimated that from one tenth to one thirtieth of the total blood is destroyed daily. The life of the red cell is probably about twenty-eight days. Fragmentation of the red cells, with the formation of fine dust, containing hemoglobin, takes place in the blood stream and is the most important step in the destruction of the red cell. The cells or their fragments are taken up and destroyed by the phagocytic endothelial cells of the spleen. When blood destruction is excessive, similar cells in the liver, bone marrow and lymph nodes share in the process. The hemoglobin from the destroyed erythrocytes is broken up by the liver and in part excreted as iron-free bile pigment. The split-off iron is largely retained for further use in the formation of new corpuscles. Other tissues may also carry on this function but, under normal conditions, it takes place largely or entirely in the liver.

The white cell dies in the blood stream. The evidences of death are the presence of vacuoles, fragmentation, poorly staining cytoplasm and densely staining, swollen nuclei. The lymphocytes are much more resistant and longer lived than the granular cells.

The life of the blood platelets is about four days.

Blood Regeneration

Under normal conditions, the regeneration of the blood is equal to the destruction of the blood, so that the balance is constantly maintained. Small losses of blood or temporary slight increases in blood destruction are met by an increase in blood regeneration, so that the equilibrium is not disturbed. The balance may be disturbed by loss of blood, either

acute or chronic, or by an increase in blood destruction, that is, by hemolysis. Hemolysis is shown by the presence of degenerative forms of the red and white cells in the blood. Degeneration of the red cells is shown by the presence of shadow forms, paleness, stippling of the shadow forms and crenation. Degeneration of the white cells is shown by fragmentation, poorly staining cytoplasm, swollen and deeply staining nuclei and the presence of vacuoles in the polynuclear cells.

If the balance is disturbed by loss of blood and the demand on the bone marrow is not too great, it may be met by an increase in the output of normal red and white cells. If the demand is greater than can be met in this way, the endothelial barrier is overcome and the unripe cells are thrown into the circulation. The red cells may be irregular in size and shape, reticulated and nucleated, but, if nucleated, they are of normal size. The white cells are younger forms of the polynuclear neutrophiles, that is, forms with less irregular nuclei (Ahrneth's shift to the left) or myelocytes. If the demand is still greater, still younger forms are thrown into the circulation, such as macrocytes, megalocytes, megaloblasts and cells showing the Howell-Jolly bodies and ring forms. There are more myelocytes and various forms of myeloblasts, many of which are of large size. These forms are all spoken of as evidences of regeneration. The bone marrow being red and normally more active in infancy and early childhood than later and there being, therefore, less reserve power in the bone marrow, early regenerative forms come into the circulation much more quickly and in much greater numbers than in later childhood or adult life. Another reason why they are more abundant is probably a tendency of the bone marrow to revert to a younger or to the embryonic condition. If the demand is excessive or continued for a long time the regenerative powers of the bone marrow may finally become exhausted.

When there is disease of the bone marrow, it may conceivably simply destroy certain portions of the marrow. If this happens, the number of cells manufactured is diminished, but those that are manufactured are normally developed. The cells in the blood stream are, therefore, diminished in number but normal in character. On the other hand, the disease may not destroy the bone marrow but disturb its normal activity or break down the endothelial barriers. The number of cells manufactured may then be either normal or diminished, but the cells are incompletely developed, that is, the cells in the circulating blood are young, or regenerative, forms. There may also be an overgrowth of certain parts of the bone marrow and an excessive number of cells produced. In such cases the barrier is also broken down. This is the case in leukemia. The function of the bone marrow in forming red cells may be normal but, on account of lack of iron, the cells may be deficient in hemoglobin. This is the case in the so-called congenital, or alimentary, anemia of early infancy. There may be also an overgrowth of adenoid tissue with an excessive formation of lymphocytes.

The bone marrow and adenoid tissue respond to demands for additional white cells as the result of the chemotactic action of disease processes elsewhere. Leucocytosis of various types results.

The anemias and other diseases of the blood and blood-forming organs are more easily understood, if these simple general facts regarding the formation and destruction of the blood and the mechanism of its replacement are kept in mind. I shall make no attempt to go into the chemistry of the blood, because it is impossible for the practitioner to perform the necessary tests and because it is at present of little clinical importance,

except in relation to certain diseases. What it is necessary to know about it is taken up in discussing these diseases.

THE BLOOD IN INFANCY AND CHILDHOOD

Hemoglobin.—According to Williamson, (Archives of Internal Medicine, 1916, XVIII, 505), 100 c.cm. of blood contains on the first day 23.3 grammes of hemoglobin. The amount at once begins to slowly diminish and during the latter part of the second week averages 21.3 grammes. Somewhere between two weeks and two months it comes down to 18.4 grammes. From the third to the fifth month it is about 13.6 grammes. It gradually diminishes to 12.2 grammes at one year. At two years it is 12.3 grammes, at three years 13.2 grammes, at four and five years 13.5 grammes, from six to ten years 14.2 grammes, and gradually increases to 14.7 grammes at fifteen years.

The percentage of hemoglobin by the ordinary tests corresponds in a general way to the absolute amount. These tests are all, however, quite inaccurate, especially the Tallqvist. They serve well enough, however, for ordinary clinical work, if it is remembered that variations of 10%, or even more, are within the limits of error. The average percentage of hemoglobin on the first day is about 120. It may, however, be as high as 140. The percentage quickly drops, so that at the end of the first week it averages about 105% and at the end of two weeks 90%. The percentage of hemoglobin in jaundiced babies is little, if at all, lower than in babies that are not jaundiced. It rapidly drops during the third week to the minimum of about 60%, after which it gradually rises to about 70% at six months. It remains at about 70% during the rest of the first two years. It then slowly rises, reaching the adult standard at about six years. The normal variations in the percentage of hemoglobin in different children, and at different times in the same child, are very marked. The percentage of hemoglobin averages somewhat higher in boys than in girls.

Specific Gravity.—The specific gravity of the blood varies directly with the percentage of hemoglobin. It is, however, of no practical importance clinically, as no one ever tests the specific gravity.

Red Corpuscles.—Lucas, (American Journal of Diseases of Children, 1921, XXII, 525), found that the average number of red corpuscles on the first day of life was 5,500,000 and that it remained at this point for several days. Most observers, however, have found a larger number, 6,000,000 to 7,500,000. Counts as high as 8,000,000 have been found. Lucas also found that the number had dropped to 4,500,000 corpuscles at two weeks. The large number of red cells and the high percentage of hemoglobin during the early days of life are probably due to a combination of loss of fluid and starvation. In my experience, the number of red cells at two weeks and during infancy is larger than 4,500,000. It ranges between 5,500,000 and 6,000,000. It then gradually diminishes during early childhood, reaching the adult standard at approximately six years. Lucas found nucleated red corpuscles in 52% of all cases on the first day and in only 5% on the second day. On the first day they were equal to 1% of the white cells and on the second day to ½% of the white cells. During the first few days there is often considerable variation in the size of the red cells. They not infrequently show basophilic granules and lose their hemoglobin easily. Nucleated cells are normally not present after the first six days. They appear with less provocation during infancy

than later and, as already noted, variations in the size and shape of the red cells and in their staining qualities develop much more quickly in infants and in young children than in older children and adults.

White Corpuscles.—There is a marked increase in the number of leucocytes during the first few days, the number sometimes being as high as 36,000. This increase is almost entirely in the polynuclear neutrophiles. The explanation of this polynuclear leucocytosis at birth is uncertain. The number of white corpuscles quickly drops within a few days or, at most, a week to from 12,000 to 14,000, where it remains during the first six months. The proportion of polynuclear neutrophiles also rapidly diminishes, so that at the end of a week the percentage of polynuclear neutrophiles and lymphocytes is the same. From this time on the lymphocytes predominate. The normal limits for the white corpuscles during the rest of infancy are between 10,000 and 12,000 per cubic millimeter. The number of leucocytes from this time on is approximately the same as in adults.

The relations of the different forms of white corpuscles throughout infancy are roughly as follows:

Lymphocytes	40% to 50%
Large mononuclear and transition forms	10%
Polynuclear neutrophiles	35% to 45%
Eosinophiles	1% to 5 %
Mast cells	1%

The distinction between lymphocytes and small mononuclear cells is more difficult to make in the infant's blood than in that of older children and adults. The larger mononuclear cells also vary a great deal, not only in the size of the cell as a whole, but also in the size of the nucleus and in the amount of protoplasm. The percentages of the large mononuclear and transition forms and of the eosinophiles remain about the same to middle childhood, while that of the lymphocytes gradually diminishes and that of the polynuclear neutrophiles increases until the adult relations are reached at from five to six years.

The absolute number of the different varieties of white corpuscles is just as important, if not more so, than the relative proportions. Calculating the number of white corpuscles during the first six months as 14,000, the absolute number of the different forms is then,

Lymphocytes	5,600–7,000
Large mononuclear and transition forms	1,400
Polynuclear neutrophiles	4,900–6,300
Eosinophiles	140– 700
Mast cells	140

During the rest of infancy, calculating the number of white corpuscles as 12,000 per cubic millimeter, the number of the different forms of leucocytes is,

Lymphocytes	4,800–6,000
Large mononuclear and transition forms	1,200
Polynuclear neutrophiles	4,200–5,400
Eosinophiles	120– 600
Mast cells	120

During the rest of early childhood the absolute numbers gradually change until the adult figures are attained at five or six years. Unless these normal variations in both the absolute and relative proportions of the white cells are kept in mind, erroneous conclusions are certain to be drawn.

Blood Platelets.—The number of blood platelets during the first week averages between 350,000 and 400,000, although figures as low as 100,000 have been found. Although there are no definite figures, it is probable that the number of blood platelets during the rest of infancy and childhood is practically the same as in adult life, averaging about 300,000.

LEUCOCYTOSIS

There is much confusion in the use of the term leucocytosis. As it is commonly used it signifies an absolute increase in the number of normal white cells in the peripheral circulation with both a relative and an absolute increase in the number of polynuclear neutrophiles. Leucocytosis in this sense occurs in infancy and childhood under the same circumstances as in adults. It is, however, likely to develop more quickly and to be more marked than in later life. One marked difference in infancy is that there is no digestive leucocytosis at this age. Leucocytosis is present in some diseases and not in others. It is of value in differential diagnosis only when one of the diseases in question has a leucocytosis and the other has not. It is of no value when both diseases have or have not a leucocytosis. In diseases in which there normally is a leucocytosis, the degree of the leucocytosis depends on two factors; the severity of the infection and the resistance of the individual. If the infection is slight, and the resistance normal, the leucocytosis is slight. If the infection is severe and the resistance normal, the leucocytosis is marked. If the infection is severe and the resistance is feeble, the leucocytosis is slight or absent.

Leucocytosis is the rule in all conditions in which there is acute inflammation. It is especially marked in pneumonia, diphtheria, inflammations of the upper respiratory tract and their adnexa, erysipelas, scarlet fever and in all suppurative conditions. There is usually no increase in the number of white cells in typhoid fever, measles, German measles, mumps, malaria, influenza, and acute, general, uncomplicated tuberculosis. There may, however, be an increase when the tuberculous process involves the meninges. There is often both a relative and an absolute increase in the number of mononuclear cells in malaria and whooping cough, although there is no increase in the total number of cells. There is also not infrequently an increase in the relative proportion of mononuclear cells in many cases of secondary anemia in early life, and in congenital syphilis. There is nothing constant about the white blood count in diseases of the digestive tract in early life. There is often both a relative and an absolute increase in the number of eosinophiles in asthma, some forms of skin disease and helminthiasis.

ANEMIA

It is very doubtful if *primary pernicious anemia* of either the usual or the aplastic type ever occurs in infancy and childhood. The diagnosis of primary pernicious anemia is sometimes made, however, because of unfamiliarity with the normal peculiarities of the blood in early life, such as the tendency to revert to a younger type of blood with a consequent increase in young forms of both red and white cells and the normal predominence of mononuclear white cells. Physicians are also often ignorant of the severity of some of the secondary anemias in early life and of the marked changes which they produce in the blood. When the bone marrow is overwhelmed by toxemia, as sometimes happens in severe secondary anemia due to sepsis, the blood picture often resembles strongly

that of the aplastic type of pernicious anemia. It is very doubtful, however, if this type ever occurs in early life.

It is also very improbable that *chlorosis*, which was formerly so common in young women, ever occurs in early life. The chlorotic type of blood is, however, very common in secondary anemia in infancy and early childhood, because of the normally relatively low percentage of hemoglobin and the relatively high red count. In early life, moreover, secondary anemia is often due to lack of iron, either in the liver at birth or in the food, so that the normally relatively low percentage of hemoglobin in comparison with the number of red corpuscles is exaggerated. On account of ignorance of these normal conditions, the diagnosis of chlorosis is, therefore, often erroneously made.

Secondary anemia is very common in early life and varies greatly in severity. Several different types may be recognized. The blood picture differs from that in later life because of the normal peculiarities of the blood at this time, which have already been mentioned. Another peculiarity of secondary anemia in early life, especially in infancy, is the tendency to enlargement of the spleen. This may enlarge in any but the mildest cases. The enlargement is not directly due to the anemia, but is simply another manifestation of the cause of the anemia. Ignorance of this tendency to enlargement of the spleen and of the peculiarities of the blood in early life has led to much confusion, not only in nomenclature but also in the diagnosis of secondary anemia. A striking example is the so-called anemia infantum pseudoleukemica of von Jaksch.

Secondary Anemia

Secondary anemia is very common in infancy and childhood. Its frequency is greatest in infancy and gradually diminishes with increasing age. In early infancy it is most often due to an insufficient supply of iron in the liver at birth. In middle infancy it may be due to an insufficient supply of iron in the food. It may also be due, as in later infancy, to disturbances of nutrition and to an insufficiency of air and sunlight. Infants and young children become anemic as the result of an insufficient supply of air and sunlight and of proper food much more quickly and more seriously than older children and adults. Anemia may also be the result of toxic absorption from local foci of infection, as, for example, diseased adenoids and tonsils. Adenoids and enlarged tonsils also indirectly cause anemia by interference with the respiration. Overexertion and fatigue and lack of proper rest and sleep may also be the cause of anemia in childhood.

It is important not to take it for granted, as do the laity, that pallor of the skin indicates anemia. The color of the skin is largely due to the size of the capillaries in it and their nearness to the surface. Children with light or red hair are very likely to be pale. Many of the southern races are pale or have an olive complexion. It is possible, however, to judge fairly accurately from the color of the lips and nails as to whether a child is anemic or not. The lips and nails furnish a far better guide than the conjunctivae. It is often impossible, however, to convince parents that their children are not anemic without a test of the hemoglobin, which they can see for themselves.

The characteristics of the blood in secondary anemia in infancy and during the first half of childhood are bound to differ from those in secondary anemia in older children and adults, because of the normal peculiarities of the blood at this age. When there is a proportionate diminution in

both the hemoglobin and the number of red corpuscles, there is apparently a much greater reduction in the percentage of hemoglobin. If, however, the percentage of hemoglobin is compared with that normal for the given age, and the number of red corpuscles with that normal for the age, the diminution in both is usually parallel. In congenital anemia and in alimentary anemia from lack of iron in the food, the hemoglobin is, of course, relatively lower than the number of red cells, even when both are compared with the normal for the age. If the physician is in the habit of examining the blood of older children and adults and is not acquainted with the normal blood of early life, he is almost certain to draw mistaken conclusions. After six years, however, the relations of the hemoglobin and red corpuscles in secondary anemia are the same as in adult life. Before this time all anemias are apparently of the chlorotic type.

Secondary anemia in early life, except when it is due to sepsis, is due to a disturbance or insufficiency of blood formation rather than to increased blood destruction. There are, therefore, few evidences of blood destruction in the blood, while evidences of blood regeneration are very common. The degree of these evidences depends on the severity of the anemia. They are always more marked than in the adult, because the marrow of the long bones is red and more active than in later life. There is, therefore, less reserve power. The younger the child, the greater are the evidences of regeneration and the earlier the forms which come into the circulation Polychromatophilic red cells are thrown into the blood in very mild cases. Normoblasts are found in very moderate degrees of anemia, while in the severer forms nucleated red cells of all sizes and shapes are quite common. Their significance, of course, is very much less than in later life.

The number of white cells in secondary anemia is independent of the percentage of hemoglobin and the number of red corpuscles. It depends on the cause of the anemia. This may or may not cause an increased demand for white cells. It may have either a positive or a negative chemotactic action on the bone marrow or the adenoid tissue. Hence, there may be an increase or a diminution in the product of either one. If the demand on the bone marrow for polynuclear cells cannot be met, younger cells are thrown into the circulation. In severe cases, the demand may be so great that even fetal types are thrown out. If that portion of the bone marrow which produces the white cells is destroyed or its capacity weakened by the toxemia of the disease, the number of cells which it produces is very much diminished.

It is most important, not only to estimate the total number of leucocytes and to determine the percentages of each, but also to determine the absolute number of each variety. It is impossible to understand the anemias unless this is done. For example, the total white count may be high and the percentage of lymphocytes considerably increased, while the percentage of polynuclear neutrophiles is considerably reduced. Nevertheless, the absolute number of polynuclear neutrophiles may be normal, showing that the bone marrow is doing its work satisfactorily.

The number of blood platelets is also independent of the numbers of red and white corpuscles, because they are produced independently by special cells in the bone marrow. They may be either normal or diminished. They are usually normal in mild cases and may be either normal or much diminished in severe cases, according to whether the part of the bone marrow which produces them is or is not involved.

The different types of secondary anemia and the variations in its severity and prognosis cannot be understood unless the principles of blood formation and destruction are kept constantly in mind, and it is remembered that the red corpuscles, polynuclear neutrophiles and blood platelets have their origin in different parts of the bone marrow and, consequently, may vary independently. It must also be remembered that the lymphocytes originate in adenoid tissue, not in the bone marrow, and that, therefore, their number varies independently of the white cells which have their origin in the bone marrow.

Confusion in the diagnosis of secondary anemia is also almost certain to arise unless it is remembered that the spleen may be enlarged in connection with all types of secondary anemia in early life, except the very mildest. The enlargement of the spleen has no relation to the degree or type of leucocytosis. It has no relation to the severity of the changes in the red corpuscles. The enlargement is not due directly to the anemia, but is due to the same cause as the anemia. The liver is also not infrequently enlarged in secondary anemia. The enlargement of the liver, like that of the spleen, is not due directly to the anemia but to the cause of the anemia. The increase in the size of the liver has no relation to the degree of the anemia, the number of white corpuscles or the size of the spleen, (Boston Medical and Surgical Journal, 1903, CXLVIII, 573). A general enlargement of the peripheral lymph nodes may occur in connection with secondary anemia. Slight general enlargement of the peripheral lymph nodes is not uncommon in connection with secondary anemia in infancy and early childhood and is not a manifestation of the anemia, but simply of a disturbance in nutrition. In some of the very severe cases, however, in which the anemia is apparently due to sepsis, there may be considerable enlargement of the lymph nodes, sometimes general, sometimes localized.

Congenital Anemia

It is conceivable that an infant may be born with defective or incompletely developed blood-forming organs. If so, the blood picture must necessarily vary with the nature of the defect or with the degree of the failure of development and may show slight or marked changes from the normal. In most of the cases to which this term has been applied, however, the blood has been essentially normal, except for an excessive diminution in the percentage of hemoglobin. This diminution is presumably due to an insufficient store of iron in the liver at birth, so that, at any rate, in most instances the so-called congenital anemia is merely a variety of alimentary anemia. The insufficient supply of iron in the liver at birth may be due to anemia in the mother, an insufficient supply of iron in her diet or to some disturbance of the iron metabolism. It is especially likely to occur in premature infants, in whom there was not time for the accumulation of a normal supply, and in twins, between whom the supply had to be divided. The blood picture is essentially the same as in ordinary alimentary anemia, as are also the prognosis and treatment.

Alimentary Anemia

Infants, under normal conditions, are born with a store of iron in the liver. There are no accurate data as to how large this store is. It is well known that the amount of iron in human milk, as well as in cows' milk, is insufficient to meet the needs of an infant for iron. It is also usually stated that the iron stored in the liver is sufficient to make up

this deficiency in the milk for six or eight months. There are no accurate data, however, as to what the need of an infant for iron really is, how much of the iron from the normal breaking down of heomglobin is retained and used again, or as to the utilization of iron. No one really knows, therefore, how much iron a baby should have in the liver at birth or how long it ought to last. Much has been said and written about the need of babies for iron and the methods of giving it to them. Data as to the real needs, the amount of iron really given in the foods recommended, and the utilization of this iron are conspicuous by their absence. It is probably not far from the truth to say that the normal baby under six months of age does not need more than 0.5 mg. of iron, calculated as Fe, daily, not more than 1.5 mg. during the rest of the first year and 2.0 mg. during the second year. The need is probably less rather than more.

In both congenital and alimentary anemia the cause of the trouble is an insufficient supply of iron from which to form hemoglobin. The bone marrow is competent, at any rate in the beginning, although it may be weakened in time as the result of the general disturbance of nutrition from an insufficiency of hemoglobin. The blood, therefore, in most instances shows simply a deficiency in hemoglobin, the red cells, white cells and platelets being normal, except that the red cells are pale. The blood is distinctly of the chlorotic type. In severe and long cases the number of red cells may be diminished as the result of depression of the bone marrow. It is very seldom, however, that the diminution in the number of red corpuscles is sufficient to necessitate the throwing of young forms into the circulation. The depression of the bone marrow is almost never sufficient to affect the production of the white cells and platelets.

Symptomatology.—The chief manifestation of alimentary anemia is pallor. There may be some disturbance of nutrition from the lack of hemoglobin in the blood and the consequent interference with its oxygenating powers. There may be loss of appetite and some enfeeblement of muscular power, and, in consequence, some delay in development. In general, however, the symptoms which are usually attributed to the anemia and to the deficiency of iron in the blood are really due to indigestion and malnutrition resulting from other defects in the food.

Treatment.—Congenital and alimentary anemia being due to an insufficiency of iron either in the liver or in the food, it is evident that treatment must consist in the administration of enough iron in a utilizable form to make up the deficiency. In order to even guess at the amount of the deficiency, it is necessary to know how much iron the infant is getting in its food, and, if the attempt is to be made to make up this deficit by a change in the food, it is necessary to know the iron content of the food to be given. Unfortunately, there is very little accurate knowledge as to the iron content of the various foods which babies and young children take. Most of those who have written about the prevention and treatment of alimentary anemia by diet have taken a great deal for granted and dealt in general principles instead of details. Careful analysis of many of their statements shows that they are simply random guesses and grossly inaccurate. Milk is a food notoriously low in its iron content, which, with modern and more accurate methods of analysis, has been shown to be lower even than was formerly supposed. A pint of cows' milk contains .08 mg. of iron, calculated as Fe, and a quart 0.17 mg. The iron in milk is very far from being sufficient, therefore, to meet the needs of an infant during the latter part of the first and the second year. Iron must, therefore, be given in some other way in order to make up the

deficit after the store of iron in the liver is exhausted. An ounce of beef juice contains 0.2 mg. of iron. The value of this iron is, however, probably not as great as it would seem to be, because the iron contained in hemoglobin and its derivatives is poorly absorbed. The yolk of an egg contains 1.4 mg. and three tablespoonsful of oatmeal 0.3 mg. The iron content of the vegetables is not as great as would seem from their color, because the color of green vegetables is due to chlorophyll and that of carrots to carotin, not to any form of iron. A level tablespoonful of cooked, strained spinach contains 0.59 mg. of iron; a level tablespoonful of string beans, 0.3 mg.; a level tablespoonful of carrots, 0.15 mg. A level tablespoonful of prune pulp contains 0.7 mg. of iron.

It is evident, therefore, that the amount of iron which the baby gets in the ordinary serving of the green vegetables, even of spinach, is entirely insufficient to meet its needs. If it is necessary to give iron in the food, more of it can be easily given in the form of beef juice, prune pulp and yolk of egg than in the green vegetables. Moreover, the green vegetables are much more likely to disturb the digestion and to upset the bowels than are beef juice and the grated yolk of hard-boiled egg. If the ordinary diet of a baby does not contain a sufficient amount of iron or prune pulp and yolk of egg do not agree with it, it is far more reasonable to give the baby some preparation of iron than to take the chance of upsetting its digestion by giving green vegetables. There is nothing in the argument that the iron in green vegetables and other foods, being organic, is better utilized than inorganic iron, because it has been conclusively proved that organic and inorganic iron are equally well utilized. It has also been shown that more iron is absorbed when considerable amounts are given than when small amounts are given, although the small amounts are infinitely larger than can possibly be absorbed and utilized. It makes very little difference what form of iron is used. The saccharated oxide of iron—Eisenzucker—in doses of from five to ten grains daily is a good preparation.

Anemia from Hemorrhage

In early life anemia from hemorrhage is almost always due to an acute loss of blood rather than to repeated hemorrhages over a long period. If one third of the blood is lost suddenly, death ensues, unless at least part of it is immediately replaced. Although the data as to the blood volume in early life are incomplete, it is probably not far wrong to estimate it at about 11% of the body weight. Two thirds of the blood, however, may be lost in twenty-four hours without causing death. Reduction of the total amount of hemoglobin below one quarter of the normal amount also means death.

Immediately after an acute hemorrhage there is, of course, no change in the percentage of hemoglobin and the number of red corpuscles. As soon, however, as the blood becomes diluted to make up the normal volume, the percentage of hemoglobin and the number of red corpuscles diminishes. If the patient survives and the hemorrhage is severe, young forms of red cells are thrown into the circulation, the youth of the cells depending on the severity of the blood loss. There is a marked polynuclear leucocytosis and also an increase in the number of blood platelets, the maximum being reached in a few days. The blood volume is restored first, while the restoration of the red cells is more rapid than that of the hemoglobin.

Treatment.—The treatment necessarily depends on the amount of blood lost. If a relatively small amount has been lost, extra liquid, time and a high protein diet, especially of meats, are all that is necessary, although it may also be advisable to give some form of iron. If the hemorrhage has been larger, physiologic salt solution may be given subcutaneously, intraperitoneally or intravenously. These injections do nothing, however, except restore the blood volume. Their action is, moreover, only temporary, as the water is quickly eliminated. If the hemorrhage has been large, that is approximately one third of the blood volume, transfusion should be done at once. If the hemorrhage has been slower, but approximately two thirds of the blood lost in twenty-four hours, it is wiser to do a transfusion. In children as in adults transfusion should be done only after proper grouping. There is less danger in infancy when the blood is not grouped. If transfusion is impossible, a 6% solution of gum acacia may be used. This is better than salt solution because it remains longer in the circulation.

When the anemia is due to repeated small losses of blood, the blood picture is no different from that in other forms of secondary anemia, the blood changes depending on the amount of blood lost and the response of the blood-forming organs. The most important point in the treatment of cases of this sort is the removal of the cause. When the cause is removed, recovery gradually takes place. It may be hastened by giving a rather high protein diet, preferably in the form of meat, and by the administration of iron. Repeated small transfusions may be of advantage in severe cases of long duration.

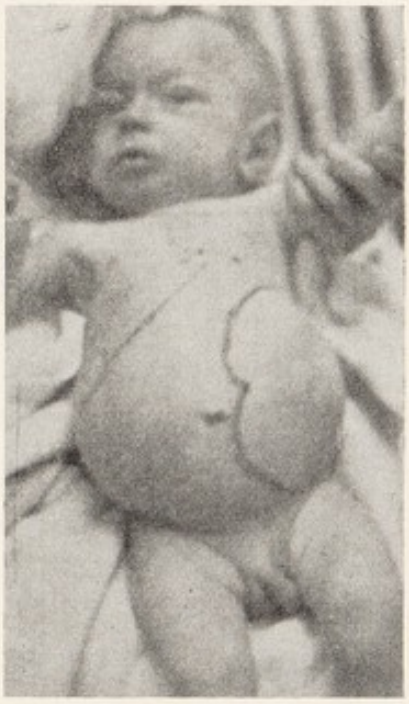

Fig. 139.—Moderate secondary anemia with splenic tumor.

Mild And Moderate Anemia

In this, the usual form, there is a diminution in both the percentage of hemoglobin and the number of red corpuscles. The diminution in the two is usually parallel and, in consequence, during infancy and early childhood the anemia is of the chlorotic type. The functions of the bone marrow are but little affected and the product fairly well completed. There are young forms in the circulation, but usually not very young. There is some variation in the size and shape of the red cells and frequently polychromatophilia and reticulation. Normoblasts are occasionally seen, but seldom megaloblasts. There may or may not be a polynuclear leucocytosis. If there is, most of the polynuclear cells are fully developed. In some cases there may be an occasional myelocyte or myeloblast. In rare instances there may be a diminution in the white cells produced in the marrow and a relative or absolute increase in the lymphocytes. The blood platelets are normal or diminished. There is no tendency to hemorrhage and no change in the coagulation or bleeding times, or in the fragility of the red cells. The spleen is usually not enlarged in the mildest cases, but may be much or little enlarged in the others. The liver is also sometimes enlarged. There is, as already mentioned, no constant relation between the severity of the anemia and the size of the liver and spleen.

This form is the one usually met in connection with rickets and other disturbances of nutrition and digestion. It is also often associated with chronic diseases of the upper respiratory tract and its adnexa. It is the form which occurs as the result of improper hygienic surroundings and lack of air and sunlight, and secondary to diseases of the kidneys and heart. It is not a serious condition, if the cause can be removed.

Treatment.—The treatment consists primarily in the removal of the cause. When this is removed, recovery takes place. Proper food, with perhaps an excess of animal protein, fresh air and sunlight help. Iron also helps. Almost any form will do. The saccharated oxide—Eisenzucker—, Blaud's mass and the tartrate of iron and potash are good preparations. They should be given in doses of at least five grains a day for babies, from ten to fifteen grains for young children and from fifteen to thirty grains for older children. It is seldom necessary to give iron subcutaneously or to resort to transfusions.

Severe Anemia

Secondary anemia of a severe type is more common in infancy and early childhood than in late childhood, but may occur at any age. The most serious cases are often mistaken for primary pernicious anemia or even for leukemia. In infancy the blood picture is more likely to suggest that of ordinary pernicious anemia and in childhood that of the aplastic form.

Fig. 140.—Severe secondary anemia with splenic tumor.

Etiology.—The etiology of severe secondary anemia is in general the same as that of the mild form. The severest cases, however, are almost certainly due to toxemia of some sort, which disturbs or depresses the functions of the bone marrow. The source of the toxemia is probably most often some hidden focus of pus. This is most likely to be in or about the throat or about the teeth. In some instances the source of the toxemia is probably in the intestinal tract.

Symptomatology.—It is extremely difficult to describe the symptomatology and blood picture in severe secondary anemia, because they are never the same in any two cases. There is, of course, almost always more or less pallor, which may be yellowish. In some of the most severe cases, in which the blood shows marked depression of the functions of the bone marrow, the skin is brownish, sometimes being so deeply pigmented as to suggest Addison's disease. Hemorrhages into the skin are not uncommon and severe nose bleeds not infrequent in the severe type with depression of the bone marrow and marked diminution in the number of platelets. The weight is usually well retained, but muscular weakness is often marked. There may be dyspnea on exertion and edema of dependent portions in the most severe cases. Loss of appetite and disturbances of digestion are common. The temperature may be normal or subnormal, but in the most severe cases is often irregularly elevated. The pulse is, of course, rapid. In some instances, especially in infancy, the pulse pressure is very high and a pistol shot and double murmur can

be heard in the groins. In other instances there are various cardiac murmurs, the most characteristic being a diastolic murmur having the distribution of that of aortic insufficiency. The spleen and liver may be much, little, or not at all enlarged. Their size has no connection with any special type of blood picture. The blood picture is different in every case, depending on whether the bone marrow is stimulated, and responds to the stimulation by throwing more or less fully developed cells into the circulation, or is depressed and unable to meet the need for new cells. If the bone marrow is depressed, the picture varies materially according to whether it is depressed as a whole or only one function is affected. While all sorts of variations occur, nevertheless, two main types of severe anemia can be recognized to which the other cases are more or less closely related.

In the *first type*, which is seen more often in infancy, the blood shows marked evidences of regeneration and the spleen is often enlarged, sometimes extremely. This form often occurs in connection with rickets and other disturbances of nutrition. It is apparently less often due to chronic toxemia from a purulent focus. The hemoglobin is usually relatively low, very likely in part because of an insufficient supply of iron in the food. The number of red corpuscles is much diminished, sometimes being below 1,000,000. The blood is full of young forms of the red cells and in the most severe cases of very young forms. There is marked variation in the size and shape of the red cells, but no special tendency to large forms. There is marked polychromatophilia. Nucleated cells of all sorts are common and mitotic figures are often seen. In fact, all sorts of young forms are common. These forms are, however, of much less serious import than in later life, because of the anatomical peculiarities of the infant's blood which have already been described.

There is usually, but not always, a considerable increase in the number of white cells, even up to 100,000. In most instances this increase is in the polynuclear neutrophiles, many of which are only partially developed. Myelocytes and myeloblasts are common and frequently are the cause of an erroneous diagnosis of leukemia. Leukemia should not even be considered, however, unless the percentage of myelocytes is over ten. Even if it is, the chances are very much against the disease being leukemia. Both polynuclear and mononuclear eosinophiles are also often considerably increased. Many unusual forms, hard to classify, are often seen. The number of lymphocytes is usually relatively diminished, but absolutely normal. Sometimes it may be both relatively and absolutely increased. The blood platelets are almost never diminished. The coagulation time is not changed. The bleeding time may be increased, often considerably. I know of no data regarding the fragility of the red cells.

It is to these cases, when there happens to be an associated enlargement of the liver and spleen and leucocytosis, that the name of anemia infantum pseudoleukemica of von Jaksch is applied. In my opinion, there is no justification for giving them a special name. They are simply a special variant of severe anemia at this age. All combinations of blood pictures, degrees of leucocytosis and variations in the size of the liver and spleen occur. A special name might as well be applied to any one of them as to this especial combination.

Diagnosis.—It is hard to see how this form of severe anemia can be confused with anything else if its picture is kept in mind. The changes in both the red and the white cells are much greater than in Hodgkin's

disease, which is, moreover, almost unknown in infancy. It ought not to be mistaken for leukemia, if it is remembered that at this age even a considerable percentage of myelocytes does not necessarily mean leukemia. Myelogenous leukemia, moreover, is also almost unknown in infancy.

Prognosis.—The prognosis is always grave, but depends less on the blood picture than on the general condition and nutrition of the infant. Recovery may take place, no matter how bad the condition of the blood. Death may occur, even when the blood picture does not seem as serious, if the general condition and nutrition are poor. The size of the spleen has no relation to the prognosis. I have seen spleens that filled half of the abdomen disappear entirely inside of three months. As a rule, the enlargement of the spleen entirely disappears, but I have known it to persist into adult life. Incidentally, the enlargement of the spleen in these cases is due to a general hyperplasia of all its elements.

Treatment.—The treatment of this class of cases consists in the first place of regulation of the diet and general hygiene. There are no general rules to be followed in the regulation of the diet. It must be suited to the digestive capacity of the individual infant. The baby should be given the maximum amount of fresh air and sunlight. It is possible that the Alpine lamp may do some good. Iron may be given as in the milder cases. It is often better, however, to give it subcutaneously. The aqueous solution of the citrate is a serviceable form. It is nonirritating rnd is never followed by induration and abscess, if the injection is properly given. The injection is, however, sometimes followed by pain lasting from a few minutes to an hour. It always causes pain, if it is given intramuscularly. Vomiting not infrequently immediately follows the injection. Why, I do not know. A glass syringe should be used. The needle must be of platinum or gold, because the solution corrodes the ordinary steel needle. The average dose during infancy is three quarters of a grain every other day. In the most severe cases it may be necessary to give a single large transfusion in order to save life. In some of the cases which do not respond to regulation of the diet and life, and iron subcutaneously, repeated small transfusions may do good. The spleen should not be removed. The operation is quite likely to be fatal and experience shows that recovery often takes place rapidly and that the spleen quickly diminishes in size under medical treatment.

In the *second type*, which occurs more often in childhood and is probably usually due to some form of toxemia, the striking feature in the blood picture are the evidences of depression or inability of the bone marrow to respond to the calls made upon it. The percentage of hemoglobin is usually absolutely diminished, but is often relatively normal when compared with the number of red corpuscles. The number of red corpuscles is much diminished, very frequently being well below 1,000,000. The red cells, however, show very little evidence of regeneration and practically none of degeneration. There is usually more or less marked achromia and not infrequently some variation in the size and shape of the red cells. In many cases, however, the red cells are normal as to their morphology and staining qualities. There are usually no nucleated cells and, if there are, they are few in number and almost always normoblasts. The number of white cells may be normal, moderately increased or much diminished. The largest number in my cases has been 40,800 and the smallest 2600. The percentage of polynuclear neutrophiles is very much diminished, as is also the absolute number. There is, there-

fore, a relatively very high percentage of lymphocytes. The absolute number of lymphocytes is, however, usually little or not at all increased. Myelocytes are usually absent and, if present, there are very few of them. The blood platelets are very much diminished or absent. The bleeding and coagulation times depend on the number of blood platelets. There is a tendency to bleed whenever the number of blood platelets is reduced to one fifth of the normal number of about 300,000, that is, to 60,000 or lower. When they are thus diminished or absent, the bleeding time is prolonged. The coagulation time is usually more or less prolonged and the retraction of the clot is usually not normal (the methods for determining the bleeding and coagulation times are described under hemorrhagic disease of the new-born). The spleen is enlarged in at least three quarters of these cases, the enlargement sometimes being very marked. The liver is also often considerably enlarged. Enlargement of the peripheral lymph nodes is also not uncommon.

Diagnosis.—Cases of this sort are often confused with acute lymphatic leukemia in an aleukemic stage. Pathologic examinations have shown that they are not leukemia. The confusion arises because of the high percentage of mononuclear cells and the failure to calculate the absolute number of the different forms of white cells. If this is done, it is evident that there is a marked diminution in the number of polynuclear neutrophiles, showing a depression of the functions of the bone marrow, and no real increase in the number of lymphocytes. Furthermore, in lymphatic leukemia the changes in the red cells are those of regeneration instead of those of loss of function of the bone marrow. Acute lymphatic leukemia is, moreover, usually a much more acute disease than this form of severe anemia, is more often associated with hemorrhages and almost always with marked enlargement of the peripheral lymph nodes.

This form of severe secondary anemia in childhood may also be confused with aplastic anemia. In fact, the blood picture is essentially the same as in aplastic anemia and is due, as in aplastic anemia, to depression of the productive powers of the bone marrow. It may be that separating them is merely making a distinction without a difference. These cases do not, however, as in the usual adult form of aplastic anemia, follow a stage of stimulation of the bone marrow. Furthermore, in most of them a fairly definite cause for the depression of the bone marrow can be made out and some of them recover after this cause has been removed. It is impossible to distinguish between this severe type of secondary anemia and aplastic anemia by an examination of the blood. Both show marked depression or incompetency of the bone marrow. In both, if the platelets are markedly reduced, there is a tendency to hemorrhage and the bleeding time is prolonged. The coagulation time may be in both either nearly normal or prolonged, but the clot is not normally retractile.

The diagnosis from certain forms of purpura hemorrhagica is extremely difficult and at times almost impossible. Both may be due to intoxication or sepsis. Both show the same tendency to hemorrhage, although this tendency usually appears earlier in purpura hemorrhagica. The bleeding time is prolonged in both. The coagulation time may be normal or increased in both. It is more likely, however, to be normal or only slightly increased in purpura than in severe anemia. Furthermore, the retractability of the clot is usually less in purpura than in anemia. The differences are, however, too slight to be pathognomonic. There is no fragility of the blood cells in either. The chief difference is in the behavior of the red and white cells. In severe anemia both the

red and white cells are diminished in number and there is little evidence of regeneration. In purpura hemorrhagica the red cells show evidences of regeneration and the polynuclear neutrophiles are increased in number, just as they are after an acute hemorrhage. It is only in very late stages of purpura, when the bone marrow has been exhausted by repeated hemorrhages, that the picture of the red and white cells is the same in the two conditions. In both, of course, the number of platelets is markedly diminished.

Prognosis.—The prognosis in this type of severe secondary anemia is always very grave. If the cause can be determined and removed, there is a fair chance for recovery, even if the red cells are below 1,000,000 and there is a leukopenia and a marked diminution in the blood platelets. If the cause cannot be discovered and removed, the chances of recovery are very slight.

Treatment.—The first and most important element in treatment is the discovery of the cause and its removal. General hygienic measures are important. Iron, whether by the mouth or subcutaneously, is not of much value. Next to the removal of the cause transfusion is the most useful method of treatment. If there has been a severe hemorrhage, a large amount of blood should be given, just as after an acute hemorrhage of any sort. If there has not been a severe hemorrhage, a large transfusion may do harm, because it may depress the activity of the bone marrow. It is, therefore, advisable to give repeated small injections at intervals of from four or five days to a week. These probably stimulate the bone marrow and, therefore, have a definite curative action. They likewise control hemorrhage as well as do large injections. If given for hemorrhage which is, as in these cases, due to a deficiency of platelets, they must be repeated every four or five days because the life of the platelets is only about four days. Two or three ounces is probably sufficient for a single injection. If it is desired to increase the number of red cells, it is said that 15 c.cm. of blood per kilo. of body weight increases the red cells by 1,000,000 per c.cm. The life of injected red cells is four weeks or less.

Transfusions may be done by either the citrate or paraffined tube method. The paraffined tube method is preferable, because reactions from it are less frequent and marked. Some platelets are destroyed by the citration and those in the blood may be injured after the injection. Blood may be injected intramuscularly, but the results are not nearly as satisfactory. Citrated blood may also be injected into the peritoneal cavity (Sipperstein, American Journal of Diseases of Children, 1923, XXV, 107 and 202).

Tests for compatability should be done before transfusion is carried out. There are, as is well known, four blood groups. These groups are distinct, even in the new-born, and the relations are the same throughout life. Roughly, about 5% of children belong in Group I, 40% in Group II, 10% in Group III and 45% in Group IV. The reaction between the cells and sera of the various groups is shown in the following table, taken from Lee (British Medical Journal, 1907, II, 684).

It is evident that if the donor and recipient are in the same group, the cells of neither will be agglutinated or hemolyzed. There is relatively little danger when only the recipient's cells are agglutinated by the donor's serum, because of the marked dilution of the donor's serum and the probable presence of some antiagglutinative substance in the recipient's serum. There is much danger, however, if the donor's

cells are agglutinated by the recipient's serum. Group IV donors may thus be used with considerable safety for the transfusion of recipients of any group as their cells are not agglutinated by the serum of any group. In the same way Group I may be the recipient from donors of any group as the serum of Group I does not agglutinate the cells of any group. Karsner, (Journal American Medical Association, 1918, LXX, 769), found in a study of 1000 soldiers that, granting that Group IV is a universal donor, the chances of a successful transfusion without previous testing is 64.9%. Nevertheless, it is always advisable, if possible, to test the bloods and use only members of the same group as donors.

TABLE XXX

Cells of groups	Serums of groups			
	I	II	III	IV
I	−	+*	+	+
II	−	−	+	+
III	−	+	−	+
IV	−	−	−	−

* indicates agglutination and hemolysis.

Lee's method for testing the bloods is one of the simplest and easiest. Sera of Groups II and III must, however, be kept on hand. A platinum loopful of each is placed on a clean slide and a drop of the recipient's blood or of blood mixed with physiologic salt solution or 1% sodium citrate solution added to it. If both drops show agglutination, the blood belongs in Group I; if neither shows agglutination, it is in Group IV. If one shows it and the other does not, the blood belongs in the group in which there is no agglutination. The reading can be made under the microscope or with a hand lens in fifteen minutes or less. If no standard sera are available, blood from the patient may be allowed to clot and the serum taken. One drop of this serum is then mixed with a drop of a one to ten suspension of the donors' blood in 1.5 % citrate solution. If the reaction is negative, it is safe to undertake transfusion. It goes without saying that, if there is time, the Wassermann test should be done on the donor.

Removal of the spleen is contraindicated in these cases.

HEMOLYTIC JAUNDICE

This disease, which is very uncommon, may or may not be familial. It is transmitted through both males and females. Not all of a family are involved. The descendants of the unaffected escape the disease. It is due to increased blood destruction, the cause of which is unknown. The primary fault may conceivably be in the red cells themselves or due to an increase in the activities of the spleen. The improvement which follows splenectomy suggests that the latter may be the cause. The jaundice is due, of course, to the necessity of disposing of the large excess of hemoglobin resulting from the blood destruction. It is not known whether the hemoglobin is broken down in the circulation or in the liver.

Symptomatology.—Jaundice may be present at birth or develop later. It may be slight and noticeable only in the conjunctivæ or fairly marked, but is never very dark. It varies from time to time. It is usually increased if there is indigestion, but never entirely disappears. The jaundice being due to increased blood destruction, not to biliary obstruc-

tion, the stools are not clay colored and there is no bile in the urine. In fact, the stools are often dark because of the greater amount of bile pigment which they contain as the result of the increase in blood destruction. The urine also contains an excess of urobilin. The spleen is much enlarged, hard and firm. It is not known whether the enlargement of the spleen occurs primarily or as the result of the increased work thrown upon it. Pathologically, it shows marked congestion with an increase of the recticulum and hyaline degeneration of the arteries. The liver is also often somewhat enlarged as the result of the increased work which it has to do. Except in the more severe cases, there is little or no disturbance of nutrition and the duration of life is not shortened. Children with this condition are, however, on account of the anemia which accompanies it, usually not as vigorous or as strong as normal. In severe cases the children are semi-invalids and death may occur in later life in an exacerbation.

Blood.—The most characteristic feature of the blood is the fragility of the red cells. Evidences of degeneration of the red cells, such as shadow forms, achromia, fragmentation and stippling in the shadow forms are common. The bone marrow is not involved and, as blood destruction is usually not very active, the number of red cells is usually over 3,000,000 and rarely below 2,000,000 In the more severe cases, however, the marrow is often unable to meet the increased demand without throwing incompleted cells into the circulation. In consequence, the blood shows more or less marked evidences of regeneration in the form of reticulated cells, variation in the size and shape of the red cells, polychromatophilia and nucleated forms. The white cells and platelets are normal. The platelets being normal, there is no change in the bleeding or coagulation time.

Diagnosis.—This disease being so rare, it hardly seems necessary to take up in detail all the conditions for which it may possibly be mistaken, which include all those associated with jaundice, the various forms of anemia and those associated with splenic tumor. There is usually little difficulty in making the diagnosis, if it is remembered that the jaundice usually begins early, is often familial, is persistent and not obstructive. The blood picture is, moreover, different from that in any other condition associated with jaundice in early life. The diagnosis can always be positively made, moreover, by testing the resistance of the blood cells, this being the only condition in early life associated with jaundice, enlargement of the spleen and anemia, in which the fragility of the red cells is increased. Normally, hemolysis begins at about 0.44% salt solution and is completed at from 0.34% to 0.36% salt solution. In this disease it may begin as high as 0.7% and be completed at 0.46% or 0.5% salt solution. This test is not a very difficult one, but requires so much preparation and apparatus that it is hardly practicable for the general practitioner, who may not need to do it oftener than once in ten years or even in a lifetime.

Treatment.—Medicinal treatment with iron and other drugs is of little benefit. Splenectomy, however, cures the disease promptly. The mortality from the operation is very low, unless it is done during an exacerbation. It may not be necessary in the mildest cases, but should be performed in all others.

LEUKEMIA

Leukemia is very uncommon in early life, although congenital cases have been reported as well as cases in infancy. Its frequency increases

directly with age. Types with a predominance of mononuclear, non-granular cells are more common than those with a predominance of granular cells. The course is, as a rule, much more acute than in later life.

Etiology and Pathology.—The etiology of leukemia is unknown. There is no justification for the belief that it may be bacterial in origin. It should be looked on rather as a blood tumor.

It is easier to understand the different varieties of the disease, if it is remembered that the white cells are formed in both the bone marrow and the adenoid tissues and that scattered throughout the body there are foci of cells which may take on or revert to their fetal functions of producing either myeloid or lymphoid cells. The spleen, liver and lymph nodes contain many cells of this type. So also do the intestines. Leukemia should, therefore, be looked on as a systemic disease in which both the myeloid and lymphoid tissues may be involved, the degree of the involvement of each varying in individual cases. Leukemia may be divided, therefore, into two main types, according to whether the myeloid or the lymphoid tissues are exclusively or predominantly involved. In general, both are involved to some extent. When there is an overgrowth in the bone marrow, either of that portion of the marrow which produces the white cells or of lymphoid tissue, those parts of the bone marrow which produce the red cells and platelets are crowded out and the number of red cells and platelets diminished. In response to the need for more red cells in the circulation, younger forms are thrown into it. These enter the blood more easily, moreover, because of the breaking down of the normal barriers as the result of the overgrowth of the tissues which produce the white cells. All sorts of young red cells are, therefore, seen in the blood. As the platelets are reduced, there is an increasing tendency to hemorrhage.

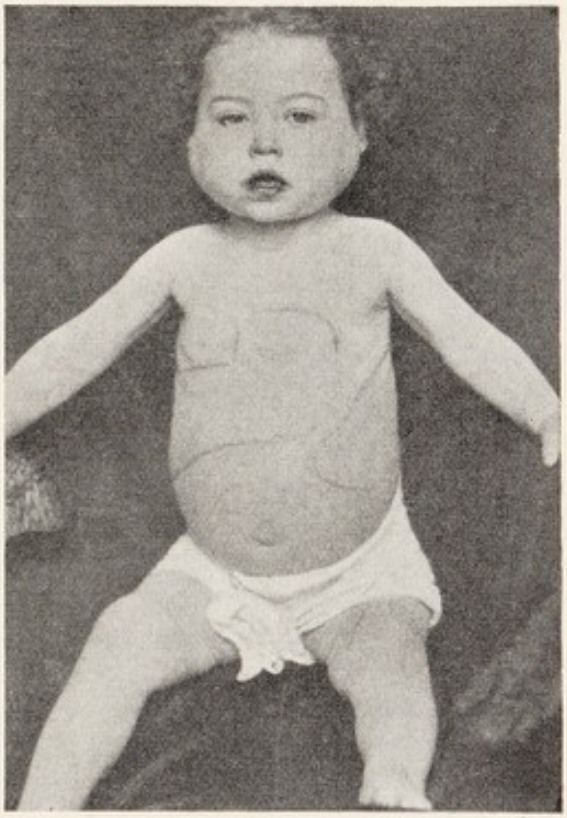

FIG. 141.—Acute lymphatic leukemia.

Symptomatology.—The symptomatology varies considerably according to the type of disease in the individual instance. In the acute type, in which there is a predominance of mononuclear cells, the first symptom may be a severe hemorrhage from some of the mucous membranes, usually of the nose or gums. In other cases, rapid enlargement of the peripheral lymph nodes develops. It is not uncommon to have this enlargement greatest in the lymph nodes of the neck. The lymphoid tissue in and about the parotid glands is sometimes much involved, so that the disease may be at first mistaken for mumps or Mikulicz's disease. There usually is not much enlargement of the liver and spleen in these cases, although there may be occasionally. Marked evidences of severe anemia, such as pallor, dyspnea, edema and disturbances of digestion quickly

develop. Hemorrhages from the mucous membranes continue and purpuric eruptions are not uncommon. Death may occur in a few days from the appearance of the first symptoms. The duration is seldom as long as three months.

In the more chronic type, in which there is a predominance of granular cells of various types, the onset is more gradual. Pallor and the general symptoms of anemia are the symptoms usually first noted, but sometimes the enlargement of the spleen is the first thing that calls attention to the fact that there is something wrong with the child. The enlargement of the spleen may be enormous, so that it fills the whole of the left half of the abdomen and extends even beyond the median line. The spleen is hard and smooth and the notch distinct. The liver is also usually enlarged, but not to the same extent as the spleen. Its surface is hard and smooth, the edge rounded. There is usually some general enlargement of the peripheral lymph nodes, but the enlargement is ordinarily not very great. Hemorrhages are less common, develop later and are seldom serious. The symptoms of the anemia gradually increase. The duration of the illness in childhood is seldom more than a few months. Remissions are not as common as in later life. The end is always in death.

Blood.—The acute cases are usually called *acute lymphatic leukemia*. There is, of course, a considerable increase in the number of white corpuscles, the number varying from 50,000 to 1,000,000. Usually, however, it does not reach 500,000 and not infrequently it is never over 100,000. There is a marked increase, both relative and absolute, in the number of mononuclear cells. In general, while there is a very marked relative diminution in the percentage of polynuclear granular cells, their absolute number is not much diminished. In fact, in many instances it is absolutely increased. The mononuclear cells may be typical lymphocytes or large mononuclear cells, which may originate either in the lymphoid tissues or be early forms of the leucocytic series formed in the bone marrow. No one but an expert in the examination of the blood can distinguish between them. The red cells show, as a rule, only moderate changes, such as achromia and moderate variation in size, shape and staining qualities. Nucleated cells are often absent and never abundant. The blood platelets are likely to be considerably diminished. When they are diminished, the coagulation time is prolonged. There are very few data as to the bleeding time in this condition. The fragility of the red cells is not changed.

In the more chronic cases, ordinarily called *myelogenous leukemia*, there is usually a greater increase in the number of white cells, which are always more than 500,000 and not infrequently over 1,000,000. These cells are almost invariably of the myeloid type, because the pathologic process consists in an overgrowth of the myeloid tissues. Most of the cells are of the younger forms of the myeloid series, both myelocytes and myeloblasts. There is, however, a considerable increase in the number of fully developed forms, the polynuclear neutrophiles, although on account of the large number of abnormal cells they are apparently diminished. Eosinophilic cells, both polynuclear and mononuclear, are not uncommon. There is also an increase in cells with basophilic granulations. The number of lymphocytes is relatively much diminished but absolutely normal. The red corpuscles and platelets are much diminished in number. The red corpuscles show marked regenerative changes. Nucleated cells of all sorts are common. The coagulation time of the

blood is increased as the number of platelets diminishes. There are very few data as to the bleeding time. The fragility of the red cells is unchanged.

Mixed types are not very uncommon and occasion much difficulty in diagnosis. In both types, but more often in the acute, there may temporarily be no increase in the number of white cells and sometimes even a diminution. In these cases, however, there is always a variation from the normal in the relative proportions of the white cells and usually a more marked difference from the normal in the absolute numbers of the different varieties of the white cells.

Diagnosis.—The diagnosis of leukemia is not difficult, if its symptomatology is kept in mind and the blood examined, provided the normal peculiarities of the blood in early life are kept in mind. If they are not, mistakes are certain to be made. I have known, for example, of a diagnosis of acute leukemia being made in a baby with scorbutic hemorrhages and a relative lymphocytosis. The diagnosis between the acute lymphatic type of leukemia and severe secondary anemia with depression of the functions of the bone marrow is sometimes, however, very difficult, especially if the blood is examined in a so-called aleukemic stage of leukemia. In general, however, the increase in the number of white corpuscles in severe anemia is very much less than in leukemia and there is an absolute, as well as a relative, diminution in the number of polynuclear neutrophiles. Acute leukemia may possibly be confused in the beginning with purpura hemorrhagica. In purpura hemorrhagica, however, the leucocytes show simply the changes common after hemorrhage.

The blood pictures are so different in leukemia and Hodgkin's disease that confusion is hardly possible, if the blood is examined. The more chronic type of leukemia, with an increase in the myeloid cells, is sometimes confused with that type of secondary anemia which is often spoken of as anemia infantum pseudoleukemica of Von Jaksch, although more often the latter is mistaken for leukemia, because the blood contains a number of myelocytes or myeloblasts. The so-called anemia infantum pseudoleukemica of Von Jaksch is a far more common condition in infancy than leukemia. The blood pictures may be for a time quite similar. The percentage of myelocytes is never as high, however, in "anemia infantum pseudoleukemica of Von Jaksch" as in leukemia and the number of platelets is not reduced.

Treatment.—There is no curative treatment for leukemia in early life. Life may sometimes be prolonged temporarily by transfusion, but transfusions do no permanent good. In the more chronic varieties, in which the blood shows changes in the bone marrow, a temporary diminution in the number of white cells may be produced by benzol. Great care must be exercised in using it, however, because of its long-continued action. It must be stopped before the number of white cells has returned to normal. Doses of two or three minims, given in olive oil, three or four times daily, are usually sufficient for children. The size of the spleen and liver can be diminished both with the Roentgen ray and with radium, with a coincident diminution in the number of white cells. The improvement is only temporary, however, and the fatal termination only slightly delayed. The removal of the spleen is not only dangerous, but useless. Medicinal treatment with iron, arsenic and other drugs does no permanent good. The best thing to do for a child with leukemia is to make it as comfortable as possible and not to bother it with treatment any more than is necessary.

CHLOROMA

This disease is essentially a sarcoma of the blood forming organs, which may affect primarily either the lymphatic system or the bone marrow. Practically, it is closely allied to leukemia and, according to whether the lymphatic system or the bone marrow is primarily involved, the blood picture is indistinguishable from that of leukemia of the lymphoid or myeloid type. Clinically, it is characterized by the tendency to form external tumors, especially in and about the skull. Tumors in the orbit, causing exophthalmos, are not uncommon. These tumors are, on section, usually, but not always green in color. Hence, the name. The cause of the green discoloration is unknown.

Chloroma is a very rare disease at any age, but more so in early life. Its should always be considered when tumors of the skull, especially in the orbit, develop. If the blood shows the picture of leukemia, the diagnosis of chloroma is justified, although it cannot be certain without an examination of the tumor. When there are no external tumors, it is impossible to distinguish chloroma from ordinary leukemia. Tumors of the suprarenal capsules also often have metastases in the skull and orbit, which grossly resemble closely those of chloroma. The blood, however, shows no leukemic changes.

There is no cure for chloroma. Treatment can be only symptomatic.

HODGKIN'S DISEASE

Hodgkin's disease is the result of a pathognomonic, pathologic process, involving the lymph nodes and lymphoid tissues. Nothing is known as to its etiology. It almost never occurs in infancy, but becomes increasingly more common throughout childhood. It is a rare disease, however, at any age. It is more common in males than in females.

Symptomatology.—The first symptom is usually enlargement of the lymph nodes in the neck. This enlargement increases more or less rapidly and often becomes extreme. In some instances the other peripheral lymph nodes begin to enlarge at the same time or soon after those in the neck. They also may become very large. In other instances they do not begin to enlarge until some time after those in the neck and never become very large. The enlarged lymph nodes are at first soft, but soon become hard. However much they may be enlarged, they are always discrete. There are no signs of inflammation about them, they are not tender and they never break down.

The internal lymph nodes also become enlarged. Those in the abdomen cause no symptoms, but those about the roots of the lungs and in the mediastinum may cause more or less marked symptoms from pressure on the trachea, bronchi, esophagus and nerves. The spleen may be much, little or not at all enlarged, according to whether there is or is not an overgrowth of lymphoid tissue in it and how great this growth is. Enlargement of the spleen occurs in at least one half of the cases. The liver may be enlarged for the same reason, but the enlargement is not usually very marked. The blood shows the changes of mild or moderate secondary anemia. Toward the end there is not infrequently a moderate polynuclear, neutrophilic leucocytosis, seldom, however, of more than 30,000. Coincident with the development of the anemia, pallor and the other ordinary symptoms of anemia appear. There is no change in the blood platelets and consequently no change in the bleeding or coagulation times and no tendency to hemorrhage. The temperature is often elevated. It may be continuously and moderately elevated or periods of

elevated temperature may alternate with periods of normal temperature. When this happens, the temperature is likely to be high, but markedly irregular, in the febrile periods. The course is usually more rapid in early life than later and may vary between two or three months and one or two years. Death occurs as the result of a general cachexia or of pressure on other organs. In rare instances, remissions with temporary diminution in the size of the tumor masses may occur.

Diagnosis.—The diagnosis is often difficult when the enlargement begins in the cervical lymph nodes and does not become very great. If the enlargement begins after an acute infection in the nasopharynx or fauces, the chances are that it is not Hodgkin's disease. If it does not, tuberculosis of the lymph nodes is more probable than Hodgkin's disease. A negative tuberculin test is strongly in favor of Hodgkin's disease, but a positive test, while counting in favor of tuberculosis, does not exclude Hodgkin's disease. In many instances, however, an early diagnosis is not possible, unless one of the lymph nodes is taken out and examined microscopically. If the enlargement is due to leukemia, the blood picture is characteristic. When there is marked enlargement of the spleen, with or without much enlargement of the peripheral lymph nodes, the appearance is much like that of leukemia. The blood pictures are so different, however, that there is no difficulty in the diagnosis. When the enlargement of the spleen is marked and there is little enlargement of the peripheral lymph nodes, as sometimes happens, some of the other conditions associated with enlargement of the spleen may have to be considered. The symptomatology and course of them all is so different, however, that diagnosis is usually not difficult. The removal and examination of a lymph node will always settle it. When only the lymph nodes are involved, the resemblance to lymphosarcoma is very close. In fact, the two conditions are often confused. The capsules and adjacent tissues are more often involved in lymphosarcoma and symptoms from pressure in the mediastinum are more likely to develop. These are differences only in degree, however, and the diagnosis can be made positively only by microscopic examination.

Treatment.—Removal of the glands in the neck, if there are no evidences of enlargement elsewhere, may delay the progress of the disease somewhat. Various operations may also at times be obligatory to relieve pressure. Arsenic in full doses may perhaps sometimes be of considerable benefit, but never cures. Treatment with the Roentgen rays and radium may reduce the size of the tumors temporarily, but does not stop the progress of the disease. It is useless to remove the spleen. Iron, nux vomica and cod liver oil may help some of the symptoms. Opium and other sedatives should be used freely, if necessary, to relieve discomfort.

PURPURA HEMORRHAGICA

True purpura hemorrhagica is due to an insufficiency of the blood platelets. This insufficiency may be due to the action of toxic substances, such as benzol, or to those produced in certain diseases, such as diphtheria. An insufficiency of the blood platelets may also be due to a depression of the bone marrow activity, as in aplastic anemia, or to disease of the bone marrow, as in leukemia. An insufficiency of the blood platelets may also occur without any apparent cause. In fact, it may be a congenital condition. In such cases, of course, there must be an impairment of the functions of the bone marrow. The term pur-

pura hemorrhagica should be applied only to those cases in which there is no apparent cause for the diminution in the number of blood platelets. True purpura hemorrhagica may be either chronic, acute or intermittent. Although the condition may be congenital, it is unusual in early life and, when it occurs, is more often acute. As a rule, when the diagnosis of purpura hemorrhagica is made in childhood, it is wrong and the hemorrhagic condition is due to some other cause.

Blood.—There is a constant diminution in the number of blood platelets. This is not very great in the mild chronic cases, but is very marked in acute cases and in intermittent cases at the time of the hemorrhage. It is less marked in the latter at other times. The hemoglobin and the red corpuscles show the characteristic posthemorrhagic changes, that is, the hemoglobin is diminished and the red corpuscles show more or less marked evidences of regeneration. The number of white cells is increased the increase being in those forms produced in the bone marrow. There is, however, sometimes a leukopenia. The bleeding time is prolonged, more so in the acute cases. The coagulation time is sometimes normal, but is usually slightly prolonged. It is seldom much prolonged. The clot does not contract normally. The fragility of the red cells is normal or slightly diminished.

There are no characteristic changes in the bone marrow. The megakaryocytes may be either increased or diminished, although usually diminished. The marrow shows in addition the changes due to hemorrhage.

Symptomatology.—The characteristic symptom of purpura hemorrhagica is hemorrhage from the mucous membranes. This occurs most often from the nose, next from the gums. Hemorrhage may also take place from the digestive and genito-urinary tracts and into the internal organs. Purpuric eruptions of all sorts may occur. They may be small and widely distributed or few and large. They are not elevated. There is little or no fever in the chronic cases, but in acute cases the temperature is usually moderately elevated and sometimes high. If the hemorrhage is severe, the appearances and symptoms of secondary anemia develop. If hemorrhages occur into the internal organs, there may or may not be symptoms referable to these organs, according to whether the hemorrhage is a large one or not.

Prognosis.—The chronic form may persist for years and not shorten life. There is always danger, however, of death in an acute exacerbation. The subacute form may last for weeks or months with complete recovery, not only symptomatically, but also in the blood condition. Death is not unusual in an exacerbation in the intermittent form or in an acute attack. The duration in some cases is not more than twenty-four hours.

Diagnosis.—The diagnosis of purpura hemorrhagica depends primarily on whether there is or is not a diminution in the number of platelets. If there is not a diminution in the number of platelets, the disease is not purpura hemorrhagica. When there is a diminution in the number of blood platelets, it is then necessary to determine from the examination of the patient and the further examination of the blood whether the hemorrhagic condition is secondary to some other disease, either general or of the bone marrow, or apparently primary. If it is primary, the diagnosis of purpura hemorrhagica is justifiable. Purpura hemorrhagica is not infrequently confused with other forms of purpura, the symptomatology of which is often quite similar. In none of them, however, is

there any diminution in the number of blood platelets. It is also often confused with severe secondary anemia. The diagnosis between these conditions is taken up under secondary anemia. The chronic form of purpura hemorrhagica is also sometimes confused with hemophilia. In hemophilia, however, the number of blood platelets is normal, although they are physiologically defective. In purpura hemorrhagica their number is diminished, but they are physiologically normal. In hemophilia the coagulation time is much prolonged, while in purpura hemorrhagica it is sometimes normal and very seldom much prolonged. The bleeding time in hemophilia is nearly normal, while it is prolonged in purpura hemorrhagica. Hemorrhages occur in hemophilia only as the result of injury. In purpura hemorrhagica they occur spontaneously. Hemophilia is an hereditary disease transmitted through females and occurring only in males, while purpura hemorrhagica is not hereditary and occurs equally in both sexes.

Treatment.—In the treatment of the chronic form it is important to keep up the general condition of the child and to overcome the secondary anemia. If hemorrhage occurs in any type, it is useless to attempt to stop it by administering drugs internally. Local applications usually do no good, but slight or moderate hemorrhages may often be controlled by mechanical pressure. If the hemorrhage continues or is severe, the only way to stop it is by transfusion. A relatively large amount of blood must be given in order to make up the deficiency in blood platelets. The effect of the transfusion is only temporary, because the platelets injected are destroyed in from three to five days. Repeated transfusions are, therefore, often necessary.

SYMPTOMATIC AND IDIOPATHIC PURPURA

Symptomatic purpura is very common and may be the result of many different causes. In it hemorrhages occur only into the skin, never from mucous membranes or into the internal organs. The blood shows no deficiency in platelets or alteration in the bleeding or coagulation times or in the fragility of the red cells. There may be evidences of secondary anemia, with or without leucocytosis. The hemorrhages are presumably dependent upon changes in the walls of the blood vessels, which are probably not always the same and certainly not always due to the same cause. Purpuric eruptions may occur in the course of many of the infectious diseases. They are said to be common in meningococcus meningitis, but I have almost never seen them in childhood. In these diseases they may also be due to bacterial emboli. Purpura is often due to drugs, among the most common of which are iodin, mercury, arsenic, quinine and the coal tar products. They are also common in chronic nephritis and in marked disturbances of nutrition, especially in infancy. I have never seen neurotic purpura in childhood. The hemorrhages in the skin, which sometimes occur as the result of venous stasis in whooping cough and convulsions, may also be described as purpura.

Diagnosis and Treatment.—Symptomatic purpura ought not to be confused with other hemorrhagic conditions, because there are no characteristic changes in the blood, none of the symptoms of the hemorrhagic diseases and usually a definite cause can be made out. It may possibly be confused in childhood with hemorrhagic insect bites, but in them there is always a central wound in the skin. There is no treatment for purpura itself, although the causative condition may require treatment.

Idiopathic Purpura is not uncommon in childhood and has been described under a great variety of names, according to the location of the hemorrhage and its association with other symptoms. It is presumable that the hemorrhages are due to changes in the blood vessels and that these are focal rather than general. It is also presumable that these changes are toxic in origin and that they are similar or closely related to the changes which occur in angioneurotic edema, localized edema and the erythema group, because all these manifestations may be associated with idiopathic purpura. Hemorrhages from the mucous membranes are very uncommon, but may occur from the digestive tract and in the kidneys.

There is usually a slight elevation of temperature and more or less marked general systemic disturbance. There are, however, no characteristic symptoms outside of those of hemorrhage.

When the hemorrhages occur only in the skin, the condition is spoken of as *purpura simplex*. When there are also pains in and about the joints, the condition is called *purpura rheumatica*, *Schönlein's disease*, or *peliosis rheumatica*. In these cases, however, there are no hemorrhages into the joints. When there is hemorrhage into the intestinal wall, as well as the skin, it is associated with severe abdominal pain and colic and the condition is spoken of as *Henoch's purpura*. The hemorrhage into the intestinal wall may precede that into the skin. When this happens, the diagnosis is often very difficult. The hemorrhage into the intestinal wall may cause a localized stoppage of peristalsis and be the starting point of an intussusception. There may or may not be hemorrhage from the bowels; more often there is not. A similar symptom may be due to angioneurotic edema of the intestines without hemorrhage, as has been described by Osler. In very rare instances, very large ecchymoses may occur suddenly. I have known the whole trunk to be involved in a few hours. When this occurs, it is known as *purpura fulminans*. This form has also sometimes been given the name of Henoch's purpura. In most instances, however, when these large hemorrhages occur, the condition is really not idiopathic purpura but a manifestation of purpura hemorrhagica or of severe secondary anemia, due to sepsis.

Prognosis.—The prognosis of idiopathic purpura, *per se*, is good. The only form which is in any way dangerous is Henoch's purpura and that only because it may cause intussusception.

Diagnosis.—It hardly seems necessary to take up the differential diagnosis from the other forms of purpura which have just been considered. In symptomatic and idiopathic purpura the blood picture is normal, except perhaps for slight manifestations of secondary anemia, while in purpura hemorrhagica and the other blood diseases, it shows characteristic changes. Purpura with pains in and about the joints can be distinguished from acute toxic arthritis (rheumatism) by the presence of hemorrhages into the skin. Purpura with abdominal symptoms ought not to be confused with other acute abdominal conditions, provided there are hemorrhages into the skin. Confusion can only arise when the abdominal symptoms appear first. Even then the diagnosis is not very difficult. Intussusception occurs only in infancy. Purpura with abdominal symptoms is a disease of childhood. Vomiting is constant, early and marked in intussusception; uncommon and seldom severe in abdominal purpura. The stools in intussusception contain no fecal matter, but are composed of blood and mucus, while in abdominal purpura they always contain fecal matter and only occasionally blood and

mucus. No tumor can be felt either through the abdominal wall or by the rectum in abdominal purpura, while a tumor is often palpable in intussusception. When an intussusception results from abdominal purpura, the characteristic symptoms of intussusception develop. When bleeding occurs into the kidney, it may be mistaken for acute nephritis. The presence of purpuric spots on the body should suggest purpura as the cause of the renal hemorrhage. Moreover, in purpura the urine contains only blood, while in acute nephritis there are other evidences of inflammation of the kidneys.

Treatment.—The treatment of idiopathic purpura is entirely symptomatic. There are no drugs which have any effect on the disease. The treatment in abdominal purpura must be primarily for comfort. The food must, of course, be limited to articles which are easily and quickly absorbed.

HEMOPHILIA

Hemophilia is an hereditary disease in which there is a marked tendency to profuse or uncontrollable bleeding following trauma. Spontaneous hemorrhage is very unusual. Although cases, apparently authentic, have been reported in females, for practical purposes it occurs only in males. It is transmitted by females. The tendency to hemorrhage is seldom manifest during the first year, but, when established, is persistent, although it diminishes somewhat during later life. The etiology of hemophilia is entirely unknown. There are no symptoms of the condition except the bleeding. Unless the patient bleeds to death at some time, it has no influence on the general well-being or duration of life.

Blood.—Microscopically, the blood shows nothing abnormal, except, if it is examined after a hemorrhage, the typical posthemorrhagic changes. While the number of platelets is normal, they are, however, defective in that they are deficient in prothrombin. The coagulation time is, therefore, much prolonged. The clot when formed is, however, normal. The bleeding time is normal and the fragility of the red cells unchanged. It is possible, but not certain, that there may also be an excess of antithrombin in the blood. The tissue juices are normal.

Symptomatology.—The only characteristic symptom of hemophilia is persistent bleeding after an injury which would not cause bleeding in a normal individual. The hemorrhage is not infrequently fatal, although death seldom takes place with the first hemorrhage. Hemophiliacs, strangely enough, are not likely to bleed from pricks, although they do from scratches. They are more likely to bleed from wounds of the mucous membranes than from the skin. They do not show purpuric spots, but may have large hematomata. Hemorrhages into the joints are not uncommon, nor are renal hemorrhages. If the hemorrhage is severe, it is often accompanied by fever. The symptoms of secondary anemia develop and vary in intensity according to the severity of the hemorrhage.

Diagnosis.—Hemophilia is seldom mistaken for hemorrhagic disease of the new-born, because the symptoms of hemophilia almost never develop before the second year. Hemorrhagic disease of the new-born, however, is not infrequently mistaken for hemophilia. The bleeding in both conditions is due to a deficiency in prothrombin as the result of a qualitative defect in the blood platelets. Hemophilia is a family disease and occurs only in males, the tendency to bleeding seldom develops

before the second year, but persists throughout life. Hemorrhagic disease of the new-born is a sporadic disease and occurs in males and females alike. The tendency to bleeding is a temporary one in the first two weeks or less of life, and there is no tendency to bleeding in later life. If hemorrhage occurs in the new-born, it is, therefore, almost never due to hemophilia.

Hemophilia is sometimes confused with purpura hemorrhagica. The differential diagnosis between these conditions is taken up under purpura hemorrhagica.

Treatment.—The first essential in the treatment of hemophilia is the prevention of hemorrhages. It is very difficult, however, to prevent children from getting scratched and cut. All operations, especially those involving the mucous membranes, should be avoided if possible. If they must be performed, they should be preceded by a transfusion.

Bleeding vessels should, of course, be tied. Mechanical pressure should be applied. It is useless to use any of the ordinary astringents and styptics because, as Larrabee says, "all they do is to make an impressively nasty mess." Epinephrin solutions in combination with pressure will sometimes control slight bleeding from mucous membranes. Cephalin and other tissue extracts are sometimes of use when applied locally. They are inaffective when injected either intramuscularly or intravenously. Gelatin and the calcium salts are useless, however given, as gelatin has no action in any way and there is no deficiency of the calcium salts. If the hemorrhage is severe, it is inadvisable to attempt to check it by animal sera, but transfusion should be done at once. Large transfusions are usually necessary in order to supply a sufficient number of normal blood platelets. The affect of a transfusion lasts, however, only as long as the injected blood platelets survive, that is, not more than four days. Retransfusion may be necessary.

SECTION XV

DISEASES OF THE KIDNEYS AND BLADDER

LOCATION AND PHYSICAL EXAMINATION OF THE KIDNEYS AND BLADDER

Kidneys.—The kidneys are proportionately much larger at birth and during the first five years of life than in the adult. They become relatively smaller up to puberty, when the adult relation is attained. The kidneys at birth are from 4 to 5 cm. long, 2 to 2.5 cm. wide and 1.2 to 1.5 cm. thick, and weigh between twenty and twenty-five grammes. The kidneys are lobulated at birth, the number of lobules varying between five and twenty-five. The lobulation has usually disappeared by five years. The cortex is much thinner in comparison with the medulla at birth than later. The adult relations are reached at about eight years. Partly because of the relatively short lumbar spine and partly because of their relatively large size, the kidneys are lower in relation to the vertebrae and iliac crests in the infant than in the adult. The right kidney is somewhat lower than the left in about two thirds of infants and young children. The adult relations are attained by middle childhood.

The suprarenal capsules are not only much larger in proportion to the body weight at birth and in early childhood than in the adult, but also much larger when compared with the kidneys. In fact, at birth they are almost as large as the kidneys and closely overlap their upper poles.

It is impossible to percuss out the normal kidney in either infancy or childhood. Although the kidneys are relatively large in infancy and early childhood, it is not possible to palpate them when they are normal, unless the child is very thin, and then only occasionally. If the kidney is palpable, the chances are, therefore, that it is in some way abnormal. The kidneys are said to be more movable in early life than later. Nevertheless, mobility sufficient to justify the term of floating kidney is very uncommon at this age and, when it occurs, it is almost always in late childhood. When the kidney is enlarged, the enlargement is usually first evident in the lumbar region. When the enlargement is greater, there may also be bulging in the side or in the anterolateral portion of the abdomen. The enlarged kidney does not move with respiration, but is usually somewhat movable anteroposteriorly. It is behind the colon.

Bladder.—On account of the small size of the pelvis, the relatively large size of the rectum and the greater obliqueness of the pelvis in infancy, almost the whole of the bladder lies at this age above the pubic crest. The long axis of the bladder at birth is nearly vertical. The horizontal axis of the adult is reached at about four years. When the bladder is distended, practically the whole of the distention is upward into the abdomen. The moderately distended bladder is egg-shaped, with the larger end directed forward and upward. There is no marked fundus. The shape of the fully distended bladder is approximately the same at all ages. The tendency is for the bladder, when distended, to lie close to the anterior abdominal wall. Very little of the anterior surface is covered by peritoneum. When the child begins to stand and walk, the weight of

the urine gradually changes the shape of the bladder. The shape of the pelvis also changes, so that by middle childhood the relations of the bladder are essentially the same as in the adult. The bladder at birth is said to hold from two to four drachms, while at six months its capacity is about one ounce. It is probable that these figures are a little small. It is impossible to give any figures as to its capacity after this time, because of its great distensibility.

When the bladder contains more than a small amount of urine, it gives an area of dullness above the pubes and is palpable in the same position. The area of dullness may extend as high as the navel, or even higher. It never, however, extends into the flanks and the upper border is convex. If the bladder is very full, fluctuation and a fluid wave may often be elicited. There is no change in the area of dullness with change of position. A full bladder may be mistaken for an abdominal tumor, either solid or filled with fluid, or for free fluid in the peritoneal cavity. I have even known it to be mistaken for a spinal meningocele. It is conceivable, but not probable, that a solid abdominal tumor may have the shape and location of a full bladder. It does not fluctuate, however, or give a fluid wave. Tumors containing fluid are almost always lateral instead of median. When there is free fluid in the peritoneal cavity, if the child is lying on its back, the dullness is greater in the flanks than in the median line, the upper border of the dullness is concave and the location changes with change of position. In all doubtful cases the diagnosis can be easily and certainly made by emptying the bladder with a catheter.

THE URINE AND ITS SECRETION IN INFANCY AND CHILDHOOD

The urine should be examined repeatedly during the course of all febrile diseases, especially those likely to be accompanied by inflammation of the kidneys. No child should be discharged as well after any of the eruptive diseases or any infection of the upper respiratory tract or its adnexa before the urine has been examined and found normal. If the urine is followed in this way, many, if not most, attacks of acute nephritis can be recognized early enough to prevent them from becoming serious. The examination of the urine should never be neglected in febrile diseases, especially in infancy and early childhood, for which there is no evident explanation. It is surprising how often under such conditions the cause of the illness will be found to be in the urinary tract. Unfortunately, the examination of the urine is often neglected in early life, because physicians either do not appreciate its importance or do not know how to get a specimen of urine.

Collection of Urine.—It is often possible to get a specimen of the urine by holding the baby over a vessel after it has had a long sleep. Exposure to cold air will often make it pass urine. Cloths wrung out in cold water applied over the bladder will sometimes induce micturition. The urine may sometimes be obtained by placing a small dish or special urinal in the diapers. This method, however, is very unreliable. It is useless to catch the urine in absorbent cotton and then squeeze it out, as the results obtained in this way are entirely unreliable. The best way to get the urine in boys is to attach a test tube to the penis with adhesive plaster. The same method may be used in girl babies. The test tube should be pushed through a hole in a piece of plaster large enough to cover the whole vulva. If desired, the test tube may be fastened into the partially cut-off thumb of a rubber glove and the part of the rubber

glove which is connected with the thumb applied to the vulva with adhesive plaster. The easiest and quickest way to get the urine, and the way which should always be used if it is necessary to know at once whether the urine is normal or not, is to catheterize the baby. Either a soft rubber or a woven catheter may be used. Suitable sizes are numbers 6 and 7 of the American scale and numbers 9 to 11 of the French scale. It is a simple matter to pass a catheter in either boys or girls, and the passage is accompanied by no danger.

The Urine in the New-born.—The first urine is acid, almost always clear and but little colored. During the first four or five days it is usually more or less cloudy from the presence of epithelial cells from the urinary passages and uric acid crystals. The specific gravity averages about 1012. Small amounts of albumin are almost always present, but rarely last longer than ten days. The sediment always contains epithelial cells, various forms of uric acid crystals and now and then hyaline casts. The amount of urine is small. It increases rather rapidly on the fourth day, from one half to one and one half ounces being passed in the first three days and between three and four ounces on the fourth day. It averages between seven and ten ounces in the second week.

The Urine in Infancy and Childhood.—In infancy the odor is slight, the color pale. The urine is usually clear, sometimes slightly opalescent, and not infrequently turbid from mucus. If the turbidity is not cleared by heat, a microscopic examination should always be made, because of the possibility of an infection of the urine with colon bacilli. The reaction is feebly acid. The specific gravity varies from 1.003 to 1.008 during the first six months, and from 1.006 to 1.012 up to two years. It does not contain albumin, and sugar is absent with the ordinary reagents. Sugar is not infrequently found, however, from time to time in the urine of healthy infants during the first two months and may be made to appear in the urine at any time during the first year, if the amount ingested is sufficiently increased. The limits of tolerance for glucose and maltose are higher than that for lactose. The sediment is slight and consists entirely of cells. The amount of urine is relatively large, the infant passing from five to six times as much urine per unit of weight as the adult. It varies between seven and seventeen ounces from the first to the seventh month and between eight and twenty ounces up to two years. These figures are, of course, only approximate, because the amount of urine varies in infancy, as at other periods of life, according to the amount of liquid ingested and the amount of perspiration.

The characteristics of the urine in childhood are essentially the same as in the adult. The quantity, however, is three or four times as large per unit of weight. It is impossible, however, to give anything more than approximate figures as to the amount passed at different ages, because of the variations dependent on the amount of fluid ingested, the amount of sweating, the season of the year and the amount of exercise. In a general way, children pass from eighteen to twenty-seven ounces from two to five years, from twenty to forty ounces between five and eight years and from thirty-two to fifty ounces between eight and fifteen years. There are no definite figures as to the relation between the amount of fluid ingested and the amount of urine passed. In middle childhood, however, when children are in bed, the difference is approximately ten or twelve ounces. The specific gravity is on the whole somewhat lower in childhood than in adult life. It is impossible to give any figures as to the specific gravity at different ages, because of the normal variations

depending on the amount of fluid ingested, the character of the diet and the loss of water through the skin and lungs.

Frequency of Micturition.—Infants when awake pass urine very frequently, sometimes several times an hour. During sleep they retain it much longer, sometimes for several hours. The frequency of micturition gradually diminishes, so that by the time a child is three years old it holds its urine when awake for two or three hours and often for eight or nine hours when asleep. The time at which children gain control of the urinary sphincter, whether by day or night, varies within wide limits. In general, they should control it by day, at any rate, by two years and should not wet the bed after they are three years old.

Suppression and Retention of Urine.—By suppression of urine is meant an arrest of urinary secretion. By retention of urine is meant the inability to empty the bladder. The new-born infant may not pass urine for twenty-four or thirty-six hours after birth. One of the main reasons that it does not pass urine sooner is that it emptied the bladder at birth. If it did not, the cause of the delay may be some malformation of the urinary organs. It is, however, usually not. The delay is more often due to a uric acid infarction. There is no cause for alarm, or even for consideration, under twenty-four hours, if the baby seems all right in other ways. The urinary secretion is scanty in infants and young children, and even in older children when they are vomiting, taking little food or water, or have a severe diarrhea. The scanty secretion under these conditions is sometimes mistaken for suppression. They often go from twelve to twenty-four hours without passing any urine, without there being any trouble with the urinary organs. Suppression of the urine may occur as the result of chilling of the surface of the body or of reflex irritation. In such cases, however, the suppression does not usually last long. It may also occur in acute nephritis. When it does occur in acute nephritis, recovery is possible, even when the suppression has lasted several days. It is most important to distinguish between suppression and retention of the urine. This is usually easily done by physical examination. When there is suppression of the urine, there are no evidences of urine in the bladder. When there is retention of urine, all the evidences of distention of the bladder are present. If there is any doubt, the diagnosis can always be confirmed by passing a catheter.

The treatment of suppression, or rather of delayed secretion, in the new-born consists mainly in pushing water. One or two grains of some alkaline salt, such as the bicarbonate of soda or the acetate or citrate of potash, may be given every hour. When there is suppression of urine in childhood, except in acute nephritis, water should be given freely. Sweet spirits of nitre may be tried, hot applications made over the kidneys and colonic flushings with water at 110° F. given. The treatment of suppression of the urine in the course of acute nephritis is discussed in the treatment of that disease.

Retention of urine, that is, the inability to empty the bladder, is not uncommon and is due to many causes. It is usually the result of reflex irritation from some local inflammation or irritation of or near the external genitals. Phimosis, balanitis, vulvovaginitis, and fissure of the anus are among the common local causes. It may also be due to pain in passing urine, or be hysterical in origin. In rare instances, it is due to mechanical obstruction, as from a stone in the urethra or something tied about the penis. The penis should be carefully investigated whenever there is suppression of the urine in babies or little children. I have

several times seen it caused by a hair being tied or wound around the penis and once or twice by a string. Retention of the urine may also be due to disease of or injury to the spinal cord.

The first thing in the treatment of retention of urine is to determine, if possible, the cause. It is necessary to be sure that there is nothing tied about the penis and no foreign body in the urethra. Hot applications should be made over the bladder. Colonic flushings with water at 110° F. may be tried or the child placed in a hot bath. If these measures are unsatisfactory, the child should be catheterized.

Polyuria.—This term simply means an increase in the amount of urine and is ordinarily used only when the increase is temporary. A persistent increase in the amount of urine is one of the symptoms of diabetes mellitus, diabetes insipidus and chronic interstitial nephritis. In diabetes mellitus, the urine contains sugar; in diabetes insipidus, the kidneys are unable to concentrate the urine; in chronic interstitial nephritis, the urine contains albumin and casts and there are other easily recognizable manifestations of the disease. In temporary polyuria, none of these things are found in the urine.

In childhood, polyuria is most often simply the result of drinking large amounts of liquids. It may in childhood, as at any age, be the result of chilling of the surface of the body, excitement or nervousness. The same drugs cause polyuria in children as in adults, notably caffeine and its salts and digitalis and its congeners. The absorption of edema and collections of fluids in the serous membranes is also almost always accompanied by polyuria.

Dysuria.—Painful micturition may in rare instances be due to the passage of small calculi through the urethra, but is most often due to local irritation at or about the opening of the urethra. In both boys and girls this may be caused by an unusually acid or alkaline urine or be due to lack of cleanliness. In boys it is often the result of phimosis and balanitis or, in the circumcised, to irritation from rough clothing. In girls the local irritation may often be very slight and, hence, easily overlooked. Caruncles are seldom the cause in early life. Gonorrheal urethritis or vulvovaginitis may, of course, be the cause, but, in my experience, they seldom are. Dysuria is also not infrequently due to irritation of the urethra during the passage of strongly acid or alkaline urine. It may also be due to the presence of crystals in the urine, especially those of acid sodium phosphate.

Treatment.—The treatment consists in the first place of the removal of the cause. If the urine is strongly acid or alkaline, the reaction should be modified by the administration of appropriate drugs, the alkaline salts being given for an acid urine, acid sodium phosphate or benzoate of soda for an alkaline urine. Local cleanliness should be enforced and simple ointments, such as boracic acid ointment, applied. Circumcision or slitting of the prepuce may sometimes be necessary. In general, however, the stripping back of the foreskin is all that is necessary. This is often as advisable in girls as in boys. If there is irritation from rough clothing, the underclothes should be changed or cotton, linen or silk sewed inside the drawers.

Hematuria and Hemoglobinuria.—Hematuria means the presence of red blood cells in the urine, while hemoglobinuria means the presence of blood pigment in the urine. *Hematuria* in infancy is most often due to scurvy. It may be the first and for a time the only manifestation of this disease. When hematuria is the result of scurvy, the urine contains no

renal elements. Hematuria in childhood is almost always a manifestation of acute hemorrhagic nephritis. There may be almost no other evidences of inflammation of the kidneys in the beginning, either in the urine or symptomatically. In the first days of life hematuria may be due to uric acid infarctions of the kidneys, sepsis, or hemorrhagic disease of the new-born. In either infancy or childhood it may be due to the irritation of crystals, most often of acid sodium phosphate, or a manifestation of purpura. In boys it may be due to hemophilia. Both malignant disease and tuberculosis of the kidneys may cause the appearance of blood in the urine. They almost never do, however, and, if they do, the amount of blood is almost always small and often can be detected only with a microscope. Blood in the urine may be due, of course, to the presence of calculi anywhere in the urinary tract or to external trauma. It may also be caused, as at any period of life, by certain drugs. While there are many possible causes for hematuria, blood in the urine in infancy usually means scurvy, and in childhood acute hemorrhagic nephritis. The treatment of hematuria consists in the discovery and removal of the cause.

Hemoglobinuria is very rare in early life and is due to the same causes as in adults. It may be one of the symptoms of septic infection of the new-born. I have never seen a case of paroxysmal hemoglobinuria in a child.

Pyuria.—Pus in the urine in infancy and childhood is almost always a manifestation of what is commonly called pyelitis. It is important to remember that the urine normally always contains microscopically a few leucocytes and that they are more numerous in the urine of girls. If the urine is macroscopically clear, and there are no symptoms of disease of the urinary tract, it is usually safe to attach no importance to them. Pus in the urine may, of course, be due to inflammation of any portion of the urinary tract from any cause. It is usually possible to tell from the other characteristics of the urine what part of the tract is involved. Pyuria may be due to the breaking through of an abscess outside of the urinary tract into any part of the tract. In such cases the urine is suddenly filled with pus when there was none before. There are also always other signs of the abscess in addition to the pus in the urine. The cause of the pyuria must always be discovered before rational treatment can be undertaken. Treatment then is primarily the removal of the cause and only secondarily of the pyuria itself.

Albuminuria.—There is, of course, always albumin in the urine whenever there is disease of the kidneys or the urine contains pus or blood. The term albuminuria is, however, usually applied to the presence of albumin alone in the urine without any evidences of disease of the kidneys or any blood or pus. There has been much difference of opinion as to the frequency with which albumin is found alone in the urine in infancy and childhood, as well as to its etiology and significance. Lee and Parmenter found albumin without any other evidences of disease in 5% of the students entering Harvard University. I am inclined to believe, as the result of having examined the urine of every child coming to my office for many years, that albuminuria is much less common in early childhood and in the early teens than it is in the later teens, as found by Lee and Parmenter. I am also inclined to think, from a less extensive experience in the examination of the urine of babies, that albuminuria is more common in them than in children.

It is well known that albuminuria may occur as the result of a high protein diet and of extreme exertion. Albumin is almost invariably

found in the urine after athletic contests and after muscular exertion of any sort. It is also not infrequently found after exposure to cold. In babies it may sometimes be due to the passage of unchanged protein through the intestines into the circulation. Albumin is also often found when the urine is extremely acid and when there is general vasomotor instability.

Whatever the cause, it is reasonably safe to believe that the presence of albumin in the urine of a baby or child, without any other symptoms of disease or any other evidences of disease of the urinary tract in the urine, is of no importance either at the time or for the future. It need cause no anxiety and, in general, requires no treatment. It is probably wise, however, if there is apparently a definite cause for the albumin in a given case, to endeavor to remove the cause.

ORTHOSTATIC ALBUMINURIA

This is the name applied to the condition in which there is albumin in the urine when a child is in the upright position, but not when it is lying down. It is also often known as lordotic or cyclic albuminuria. It is uncommon in young children, but not infrequent after six years. It usually disappears in the late teens but may persist into adult life. It is more common in boys than in girls. It is more common in tall and thin children than in short and fat, but may occur in all types. There are no other evidences of disease of the kidneys, either in the urine or symptomatically. Appropriate tests show no disturbance of the renal functions. The amount of albumin may vary from a trace to 1 or 2%.

Etiology.—On account of the dependence of the albuminuria on the position of the child and the greater frequency of the condition in tall, thin children and in those with lordosis of the lumbar spine, the albuminuria has been attributed to congestion in the kidneys as the result of interference with the venous return through bending of the large veins when the child is in the upright position. Further evidence adduced in favor of this explanation is that, if the child keeps its back straight when in the upright position, the albuminuria does not occur, while, if it is kept in the lordotic position when lying down, there is albumin in the urine. The explanation is, however, probably not quite so simple, because, while if the child stands still there is albuminuria, there is none if it keeps its legs in motion. Furthermore, many children with marked lordosis do not show albumin in the urine. There is considerable evidence, moreover, that these children show vasomotor instability. The albuminuria also apparently varies with the general condition of the child and often disappears after the removal of foci of infection. It may also be made to disappear in certain cases in which the urine is highly acid by changing the reaction of the urine to weakly alkaline. It is probable, therefore, that, while the albuminuria is directly connected with the position of the spine, there is some other cause or causes which make it possible for the lordosis to be effective.

Diagnosis.—When albumin is found in the urine of a child that has no symptoms of disease of the kidneys and the urine shows no other evidences of disease of the urinary tract, this condition should always be thought of, especially if the child is tall and thin and has an exaggerated lumbar lordosis. Several separate specimens of urine should then be examined—one passed immediately after getting out of bed in the morning, another after being up and about, another after lying down for an hour in the middle of the day and another after being up and about again.

The bladder must be emptied, of course, at the beginning of each period. If the urine passed after being up and about contains albumin and that passed after lying down does not, the diagnosis of orthostatic albuminuria is justified. It is interesting to strap the patient to a board when he is up and put a hard pillow under his back when he is lying down and observe the results, but it is not necessary for a diagnosis. If orthostatic albuminuria is suspected and the urine does not contain albumin at the time of the examination, it can often be caused to appear in the urine by putting the child in one of the "provocative" positions, recommended by Saito (American Journal of Diseases of Children, 1921, XXII, 388). In one of these the child stands upright with the arms stretched out in front so that the hands are a little higher than the head. In the other the child kneels with the hands crossed over the pubes and the head held well back. Neither position should be held more than ten minutes. The appearance of albumin in the urine justifies the diagnosis of orthostatic albuminuria. This condition is not infrequently mistaken, either through carelessness or ignorance, for some form of nephritis and children consequently subjected unnecessarily to the discomforts and annoyances incident to the treatment of nephritis, while their parents are needlessly alarmed. I have known, for example, a well child with orthostatic albuminuria to be taken out of school, made a semi-invalid, kept on a restricted diet and sent South every winter, with considerable sacrifice on the part of the parents, simply because its physician was unaware that there was such a thing as orthostatic albuminuria. It must also be remembered that a child with orthostatic albuminuria may have an acute inflammation of the kidneys from which it recovers, while the orthostatic albuminuria persists. A mistake may easily be made when this is the case and treatment kept up indefinitely, unless this possibility is kept in mind.

Prognosis.—Orthostatic albuminuria is an unimportant condition. It does no harm and never leads to nephritis. It is quite likely to disappear as children, getting heavier and becoming more muscular, improve their posture. It usually disappears before twenty, but may persist into adult life.

Treatment.—There is no specific treatment for orthostatic albuminuria. In fact, no treatment is needed. Above all, the protein in the diet should not be cut down, as it so often is. If there is a marked lordosis and the posture is poor, it is a good plan, however, to have the child take exercises to improve the posture. In some instances it is advisable to have them wear a supporting belt, not so much on account of the albuminuria as to improve their digestion and general condition. As there is probably some other factor in the etiology of most cases beside the purely mechanical one of position, it is advisable to get rid of all possible ofoci f infection and to regulate the life and diet so as to get the child into the best possible state of nutrition. When the urine is strongly acid, regulation of the diet by giving more fruit and vegetables or the administration of an alkali, by reducing the acidity of the urine, usually diminishes and often causes the disappearance of the albumin.

NEPHRITIS

Etiology.—Nephritis in childhood is usually either acute or subacute. If chronic, it is almost invariably the result of a previous acute attack. Chronic nephritis is almost never due in childhood to syphilis, alcohol or disturbances of the circulatory system. Acute nephritis is almost

invariably secondary to an infectious process elsewhere. The primary infection is most often located in the nasopharynx or its adnexa. The tonsils are most commonly the seat of the infection, but acute nephritis may follow a simple rhinitis or otitis media. Another occasional cause of acute nephritis is a suppurative process involving the teeth or gums. Acute nephritis may accompany or follow any acute infectious disease, especially scarlet fever or diphtheria. The importance of scarlet fever in the etiology of nephritis has, however, been much overestimated. It may be true, perhaps, that scarlet fever is more often accompanied by nephritis than any other disease, but the number of cases due to scarlet fever is absolutely much smaller than that due to infections of the throat. Pneumonia is seldom the cause of nephritis in childhood, while it may be due to such mild infections as chicken-pox, impetigo and eczema. It is very doubtful whether exposure to cold and chilling of the surface of the body is ever the cause of acute nephritis. When this is apparently the cause, it is probable that the chilling simply lighted up some hidden focus of infection.

The inflammation of the kidneys is almost invariably due to the action of bacterial toxins. In rare instances it may be due to the direct action of bacteria upon the renal tissues, or to the combined action of bacteria and their toxins. It is likely that in some of the cases which occur in connection with diseases of the intestinal tract, especially in infants, the inflammation may be due to the action of endogenous chemical toxins. In other rare instances nephritis may be caused by drugs, such as turpentine, corrosive sublimate, arsenic and potassium chlorate.

Classification.—The classification of the diseases of the kidney in childhood on the basis of the pathologic changes in the kidneys is even more unsatisfactory than it is in adult life. It is impossible to fit the findings in the urine to any special type of pathologic lesions in the kidneys. Furthermore, the lesions in the kidneys unquestionably vary materially from time to time in the same case, according to the duration and progress of the disease. Classification on the findings of the modern tests for kidney functions is also unsatisfactory and unreliable. A classification based on a combination of the symptomatology and the findings in the urine seems, therefore, simpler and more rational. The classification which I am in the habit of using is arranged on this basis. While not entirely satisfactory, it is certainly true that the vast majority of the cases of nephritis in childhood fit fairly well into one of these classes. This classification is as follows:

Acute Degenerative Nephritis
Acute Hemorrhagic Nephritis
Acute Exudative Nephritis
Subacute Nephritis
Chronic Nephritis
Chronic Diffuse { Mild / Severe
Chronic Interstitial
With Infantilism

Tests for Renal Functions.—The important points in the examination of the urine, and those which aid most in the differentiation between the various types of nephritis, are the quantity of the urine, the relation of the quantity to the amount of liquids ingested, the amount of albumin and the findings in the sediment on microscopic examination. The specific gravity is of relatively little importance, because it is dependent

on the amount of liquids ingested, the amount of urine passed, the activity of the skin and bowels, and the diet. It is likewise useless to estimate the amount of urea in the urine, to which so much importance was formerly attached.

The modern tests for renal functions are likewise, in my experience, of relatively little importance in comparison with the findings in the urine and the general appearance of the patient. In the mild cases these tests show, of course, little interference with the functions of the kidneys, while in the very severe they show much. They give little information, however, as to the future progress of the case. Cases which show marked interference with the kidney functions may suddenly clear up, and those with little disturbance run on indefinitely. These tests are of far more value in prognosis in the subacute and chronic than in the acute types, and in them are often of great assistance.

Most of these tests are, moreover, impracticable for the general practitioner. Two of them can, however, be relatively easily carried out. These are the phenolsulphonaphthalein and the specific gravity test to determine whether the kidneys are able to vary the concentration of the urine. They are commonly known as the phthalein and gravity fixation tests. The latter is the more valuable. The height of the non-protein nitrogen of the blood is probably a better indicator of the excretory powers of the kidneys than the phthalein test, but can only be determined when a well equipped laboratory is at hand. In chronic cases it furnishes but little more information than the gravity fixation test.

Phenolsulphonephthalein Test.—In this test one c.cm. of a solution containing six mg. of phenolsulphonephthalein is injected intramuscularly and all the urine passed in the first two hours saved. This is made alkaline with sodic hydrate to bring out the color of the phthalein, diluted to 1000 c.cm., and the intensity of the resulting color compared with that of a standard solution. The result is expressed in per cent. of phthalein excreted in two hours. Normally, the average excretion in children is 75%. If the excretion is below 60%, it is certainly abnormal.

Gravity Fixation Test.—Under normal conditions, the specific gravity of the urine rises when there are more solids to be excreted, provided there is no increase in the intake of liquids. The specific gravity of the urine rises, therefore, after a meal rich in protein and salts. The amount of urine diminishes and its specific gravity rises at night, unless extra liquid is taken during the night, that is, the normal kidneys are able to excrete a urine of increased concentration when there is an increase in the amount of solids to be eliminated. When the kidneys are diseased, however, they lose to a greater or less extent their power to excrete a concentrated urine and can only eliminate the increased solids by excreting more urine of the same low specific gravity after meals while the night urine is increased in quantity and of low specific gravity. The test meals devised by Dr. Lewis W. Hill, which I am in the habit of using are as follows:

Breakfast and Supper.

Cereals, two tablespoonfuls
Bread, one slice
Butter, one half ounce
Apple Sauce, two tablespoonfuls
Milk, six ounces
Water, four ounces
Extra salt, fifteen grains
Caffein sodium benzoate, two grains.

Dinner.

Chopped Meat, two tablespoonfuls
One egg
One potato
Butter, one ounce
Milk, six ounces
Water, four ounces
Extra salt, fifteen grains
Caffein sodium benzoate, two grains.

It is not necessary for the child to eat all the food, but it must take the salt, caffein, milk and water. No extra water is given during the twenty-four hours.

The urine is collected every two hours during the day, that is, from 6 A.M. to 6 P.M. The night urine, that is, that passed between 6 P.M. and 6 A.M. is collected together. The specific gravity of each specimen is then taken and the amounts of the day and night urine compared. Normally, there is a difference of at least eight points between the highest and lowest gravity of the two hour specimens, usually considerably more. The specific gravity of the night urine is almost always over 1.020.

Blood Nitrogen.—The upper normal limit of the non-protein nitrogen of the blood is 30 mg. per 100 c.cm. of blood.

In general, a continuously low phthalein excretion means considerable disturbance of the functions of the kidneys. A high phthalein excretion is of no importance. A low phthalein excretion at a single examination is not necessarily serious. On the other hand, the patient's condition may be serious when the phthalein excretion is normal. Fixation of gravity, unless constant, is not especially serious. If it is constant, it means considerable disturbance of the functions of the kidneys. The test is, therefore, much more valuable in chronic cases. An increase in the non-protein nitrogen of the blood is not necessarily important, but, when there is such an increase, the patient is more likely to develop uremia or convulsions than when the blood nitrogen is normal.

Blood Pressure and Cardiac Hypertrophy.—As a rule there is little increase in the blood pressure in acute nephritis, although at times there may be a considerable increase. It is more often increased in the exudative than in the hemorrhagic type. The blood pressure gives little information as to the severity of the condition. It may be high in cases that are doing well and low in cases that are doing badly. If it is very high, however, the patient should be watched closely for the development of uremia. There is nothing constant about the blood pressure in subacute and chronic nephritis in children. A continuously high blood pressure, however, shows that the kidneys have been considerably damaged. The blood pressure is high in the rare cases of the chronic interstitial type, just as in adults.

There is very seldom enough cardiac hypertrophy in acute nephritis in children to be detected on percussion or to increase the intensity of the second aortic sound. There is no apparent relationship between an increase in the size of the left heart and the severity of the illness. There may be slight enlargement of the left heart and accentuation of the second aortic sound in chronic nephritis, but these signs are never marked, except in the rare cases of the chronic interstitial type.

Acute Degenerative Nephritis

This condition properly does not deserve the name of nephritis, because the pathologic condition is really degenerative and not inflam-

matory. It is limited largely to the cells of the convoluted tubules. It is always present to a greater or less degree in every acute infectious disease. It is often spoken of as febrile albuminuria.

There are no symptoms from the degenerative process in the kidneys, or, if there are, they are so overshadowed by those of the causative disease that they are unrecognizable.

The quantity of urine is not influenced by the process in the kidneys, but depends on the height of the temperature, the amount of liquids ingested and the activity of the skin and bowels. The specific gravity naturally varies with the quantity of the urine. The color may be pale, normal or high. It is never reddish. The amount of albumin varies from the slightest possible to a large trace. The sediment is usually slight, unless there is a considerable amount of urates. It shows a few cells of various sorts and an occasional hyaline or granular cast. There may be also a few red corpuscles and sometimes a slight excess of leucocytes. Tests for renal function show no disturbance of the excretory powers of the kidneys.

This condition has no influence upon the prognosis and requires no treatment. It is advisable, however, as in all acute infectious diseases, to give the patient considerable amounts of water.

Acute Hemorrhagic Nephritis

In this type of nephritis the first symptom noted is usually the passage of red or brown urine. This symptom may appear during the course of the disease to which it is secondary, but not infrequently does not appear until a week, or sometimes more, after the cessation of acute symptoms. It is this type of nephritis which is most often apparently primary and which most often seems to be due to a chill. Constitutional symptoms are usually slight. There is often no elevation of temperature and it is never very high. Edema is unusual and never marked. There is, however, a progressively increasing pallor.

The urine is usually not much diminished in amount. It varies in color from bright red to light red, or from very light to dark brown, according to the amount of fresh or changed blood which it contains. There is nothing constant about the specific gravity. It contains from a trace to 1 or even 2% of albumin. There is usually considerable, and sometimes a large amount of sediment. The sediment is made up almost entirely of red corpuscles, most of which are usually normal, but some of which are changed. There are in addition a few leucocytes and large and small round cells. In many cases there may be for some time no casts in the sediment, but as time goes on they almost always appear. In some cases they may be very numerous. They are hyaline, granular, brown granular and blood, sometimes epithelial. The sediment also contains considerable brown, granular debris.

In occasional instances there may also be a large number of leucocytes in addition to the blood. In some instances the sediment is composed for a day or two very largely of leucocytes with a few red cells, and then for a few days largely of red cells with a few leucocytes, the relation between the two alternating. In such cases it is sometimes very hard to know whether the condition is primarily a pyelitis or primarily nephritis. It almost always turns out to be, however, nephritis, and the pus disappears from the urine.

As a rule, tests for kidney function show but little diminution in the excretory powers of the kidney in this type of nephritis, but there are

exceptions. The blood pressure is usually little, if at all, increased and there are almost never any evidences of hypertrophy of the left heart. Here again, however, there are occasional exceptions.

Children with this type of nephritis are usually not very ill. Most of them recover in a few weeks. Very few die. In a moderate number, however, the disease changes into the subacute type. It very seldom becomes chronic.

Because of the large amount of blood in the urine ann the frequent absence of casts, this type of nephritis is sometimes mistaken for purpura or stone in the kidney or bladder. I have also known it to be mistaken for scurvy. In this type of nephritis, however, there is almost always a history of some acute disease within the past one or two weeks. There is no pain and there are no other evidences of a hemorrhagic condition. Moreover, careful search usually reveals an occasional cast.

Acute Exudative Nephritis

This type of nephritis also develops towards the end of or soon after some acute infectious disease. The first symptom noted is usually edema of the face. Sometimes it appears first in the extremities. Investigation then shows that the child is passing a small amount of urine. The edema often becomes very marked in these cases and sometimes there is ascites. The child is manifestly quite sick. There is often nausea and vomiting, sometimes headache. The temperature is usually somewhat elevated, but may be normal. There is more or less pallor.

The urine is always diminished in amount, sometimes only a few ounces being passed in twenty-four hours. It is in this type of nephritis that suppression of the urine occurs. The specific gravity is always high. The color is normal or high, never red. The urine always contains albumin, usually from ½ to 2%. Not infrequently in the severe cases it becomes almost solid on boiling. The sediment is usually considerable. It is made up largely of casts and cells. The casts are hyaline, granular and epithelial. There are almost never any blood casts. The cells are usually mostly small round, with an occasional large round and caudate. Leucocytes are not uncommon. There are often a few red blood cells, but never many. In occasional cases the sediment is made up very largely of mononuclear cells, with an occasional polynuclear leucocyte and casts of large diameter (Morse, Archives of Pediatrics, 1908, XXV, 497).

The tests for renal function are quite likely to show an interference with the excretory powers of the kidneys, although there is nothing constant about the results. There is always a diminution in the ability to excrete salt. The blood nitrogen is usually increased, in the severe cases considerably. There is more often an increase in the blood pressure than in the hemorrhagic type. This may be considerable in uremic cases. There is seldom any clinical evidence of hypertrophy of the heart.

Children with this type of nephritis are usually quite ill. A small proportion of them die with uremia. If they do not die, they usually recover entirely, as this type seldom runs into the subacute type or becomes chronic.

Subacute Nephritis

The subacute type usually develops from the acute hemorrhagic, but in some instances the symptoms from the beginning are so slight that the disease is apparently always of this type. It is often very difficult to state when the acute hemorrhagic ends and the subacute begins. The

persistence of red corpuscles and casts in the urine after three months, without any marked symptoms clinically, probably justifies the diagnosis of the subacute type. As a rule, the children usually look and feel well, although there is sometimes a little pallor and the children are easily fatigued.

The urine is normal in amount, sometimes slightly increased. The color varies from pale to normal, but occasionally may be very slightly reddish or brownish for a day or two, if there is a temporary exacerbation. There is nothing constant about the specific gravity. The urine contains but little albumin, almost never more than a trace, and frequently none at all for days at a time. The sediment is slight and is made up of red cells, epithelial cells and hyaline and fine granular casts. The amounts and relative proportions of these elements vary from day to day. Not infrequently there is almost no sediment for a few days, then it increases again.

The various tests show little or no interference with the functions of the kidneys. The blood pressure is not elevated and there are almost never any evidences of hypertrophy of the left heart.

Almost all of these cases recover entirely, although it may be two or three years before the urine is permanently clear. Occasionally one becomes chronic.

Chronic Nephritis

Chronic nephritis in childhood is a sequela of acute nephritis and is relatively uncommon. It is almost never of the chronic interstitial type seen so commonly in adults. In very rare instances it is associated with delayed development, to which the term of infantilism is often applied. Chronic nephritis may be divided into three different types, chronic diffuse, either mild or severe, chronic interstitial and chronic with infantilism.

Chronic Diffuse Nephritis.—This form of nephritis may be of so mild a type that the child looks and feels well, or of so severe a type that the child presents all the clinical symptoms of chronic diffuse nephritis, as seen in the adult.

The urine may be normal or greatly diminished in amount. The specific gravity varies with the amount of urine. The color is normal or high. The urine is usually clear. It may contain from a slight trace to 2%, or even more, of albumin. The sediment may be much or little, according to the severity in the individual case. It contains casts and cells of all sorts, but never red cells or blood casts, unless there is an acute exacerbation. In the mild cases fatty cells, fatty casts, compound granule cells and free fat are very uncommon, as also are waxy casts. All of these elements may, however, be very numerous in the severe cases.

The tests for renal function usually show more or less impairment of the functions of the kidney, the amount of the impairment depending upon the severity and the stage of the individual case. It is very surprising how little impairment may be present in some of the milder cases. The blood pressure is usually slightly increased, but never very markedly. In the severe cases there may be some evidences of enlargement of the left heart, but these are never very marked.

Children with this type of nephritis almost invariably eventually die. It is surprising, however, how long many of them may live and how comfortable they may be almost to the end. Recovery occasionally takes place in cases which present all the evidences of a mild or moderately

severe chronic diffuse nephritis. It may be that when recovery takes place there really was some other condition in the kidney. If so, however, it is indistinguishable clinically and on the findings in the urine from chronic diffuse nephritis.

Chronic Interstitial Nephritis.—The symptomatology and urinary findings in the chronic interstitial type of nephritis in early life are exactly the same as in adult life. There is the same increase in the amount of urine and in the relative amount of night urine, the same low specific gravity, the same small amount of albumin and sediment. The functional tests show marked impairment of the excretory powers of the kidneys. The blood pressure is increased and there is hypertrophy of the left heart. Recovery never takes place.

Chronic Nephritis with Infantilism.—This type of chronic nephritis is practically chronic interstitial nephritis, modified by the age of the patient. The urinary findings are essentially the same, the blood pressure is very high and the heart shows considerable hypertrophy of the left side. The characteristic point in the symptomatology is the interference with both the mental and physical development. Children with this condition are often several years behind in development, not only physically, but also mentally. Death is inevitable in these cases.

Treatment of Acute Nephritis

The development of acute nephritis, at any rate of a severe type, is preventable in the vast majority of cases. It is almost a reflection on the professional ability of a physician who has a severe case of nephritis develop in a patient for whom he has been given a fair opportunity to care. In the first place, adenoids and clinically diseased tonsils should be removed before they have a chance to cause trouble. The teeth should be watched and, if decayed, filled or removed. If a child has any of the diseases likely to be accompanied or followed by inflammation of the kidneys, its routine should be regulated along the same lines as if it had nephritis. Children that are kept in bed, given a low protein diet and much water relatively seldom develop trouble in the kidneys. The urine should be examined frequently in every acute disease, no matter how trivial, in order that the first signs of trouble may be detected. If nephritis is recognized early and properly treated, it rarely becomes severe. Furthermore, the urine should always be examined during convalescence. No child should be considered well after any acute disease until the urine has been examined after it has been up and about for a week or more.

I have followed for a long time the principles of the treatment of acute nephritis advanced by von Noorden twenty years ago. They form the basis of the treatment advised below.

There is no drug, serum or vaccine which has any directly curative action on the inflamed kidney. When there is no directly curative treatment, the treatment of every diseased organ whose continued action is necessary for the life of the individual may be divided into two parts; the first comprising those measures which diminish the work which the organ has to do; the second, those which help the organ to do its work. Unfortunately, little or nothing can be done to help the acutely inflamed kidneys to do their work. The treatment of acute nephritis consists, therefore, in diminishing the work which the kidneys have to do.

The kidneys are excretory organs. In a general way they excrete the end products of fats and carbohydrates easily and those of proteins with difficulty. Water may or may not be easily excreted.

The proteins in the food should, therefore, be diminished. It is useless to diminish them below the minimum protein needs of a child, because, if these needs are not supplied, the proteins of the body will be broken down and the same load thrown on the kidneys as if the protein was taken in the food. There are no really reliable data as to the minimum protein needs of children of various ages. Calculating that the minimum protein needs are roughly three fifths of the average protein needs, they are approximately at four years, thirty grammes; at eight years, thirty-five grammes; and at twelve years, forty-five grammes. It makes no difference to the kidneys whether the protein is of animal or vegetable origin. The proteins of meat, fish and eggs are no more harmful than those of milk. The amount of protein in the food, not its source, is important. The diet should be regulated, therefore, so as to contain only enough protein to cover the minimum needs. This is easily done by following any of the tables of food values. It is idle to believe, of course, that variations of a few grammes make any material difference.

The caloric value of the food may be kept up, in spite of its low protein content, by increasing the amount of fat and carbohydrates in the diet. Children of four years need a minimum of 1200 calories daily, those of eight years, one of 1400, and those of twelve years, one of 1600. It is easy to supply these calories with the aid of the various food tables. The table of food values, which I am in the habit of using, is shown in Table XXXI on page 651.

The extractives are eliminated with difficulty by the kidneys. They are most abundant in the protein foods. This is another reason, therefore, for keeping down the protein in the diet. From this point of view, meat and fish are not quite as suitable as milk. Broths and soups made from meat are more dangerous, however, than meat itself, because they contain most of the extractives of the meat. Furthermore, they have but little food value and contain much water, which may of itself be very harmful. No broths or soups should be given, therefore, to children with nephritis.

Other substances which are eliminated with more or less difficulty by the kidneys are phosphates, inorganic sulphates, hippuric acid and other salts. For practical purposes they all may be disregarded, except common salt, which always should be kept very low and, whenever there is any edema, cut out of the diet entirely.

Water is a stimulant to the kidneys. Moreover, when a large amount of water passes through the kidneys, the substances which are eliminated by them are diluted, and hence cause less irritation. When the kidneys are acutely inflamed and congested, as in acute exudative nephritis, they are unable to eliminate water. The water then increases the congestion and irritation, is not eliminated and is retained in the body, this retention being manifested by edema and ascites. In the acute hemorrhagic and subacute forms water is more easily eliminated, but even in these types, if it is forced, edema and ascites develop. During convalescence water is excreted very easily and, because of its diluting power, should be given freely. The water intake should be watched just as carefully, therefore, as that of the protein, extractives and salt. If there is any edema, except possibly a little in the eyelids, the water intake must be kept very low. If there is very marked edema, it is advisable to keep it down to a few ounces daily. It cannot be eliminated and can do nothing but harm. If there is no edema, water may be given cautiously, watching at the same time the urine output and the weight.

TABLE XXXI
TABLE OF FOOD VALUES

	Calories	Grams		
		F.	C.	P.
Whole milk, 1 quart	670	38	43	34
Skimmed milk, 1 quart	400	10	43	35
Fat free milk, 1 quart	320	.....	43	35
Heavy cream, 32%, 1 pint	1580	154	22	14
Gravity cream, 16%, 1 pint	860	77	22	14
Buttermilk, 1 quart	360	5	43	35
Whey, 1 quart	260	5	43	9
Beef juice, 1 ounce	10	*	....	2
Crackers, 1 ounce[1]	120	3	20	3
Bread, 1 slice[2]	75	*	15	3
Zwieback, 1 slice[3]	120	3	20	3
Shredded wheat biscuit	105	*	22	3
Rolled oats (cooked), 1 tablespoonful[4]	35	*	6.5	1.5
Cream of wheat, Ralston and similar cereals (cooked), 1 tablespoonful[4]	40	.....	8.5	1.5
Potato, size of large egg	70	.....	15	2
Macaroni (cooked), 1 tablespoonful[4]	32	*	5	1
Egg: Whole	72	5	....	7
Egg: Yolk	60	5	....	4
Egg: White	12	.....	....	3
Meat / Fish (cooked), 1 ounce[5]	60	3	....	7
Bacon, 1 slice = ½ ounce	90	9	....	1.5
Butter, 1¼ inches cube = 1 ounce	225	24		
Olive oil, 1 tablespoonful	125	14		
American cheese, 1¼ inches cube = 1 ounce	130	10.5	....	8.5
Cream cheese, 1⅛ inches cube = 1 ounce	130	10.5	....	8.5
Sugar: Cane, 1 rounded teaspoonful	25	.....	6	
Sugar: Milk, 1 rounded tablespoonful	60	.....	15	
Green peas / Lima beans (cooked), 1 tablespoonful[4]	40	.....	7	3
Carrots / Squash / Turnip / Beets / Onions (cooked), 1 tablespoonful[4]	30	....	6	1
Orange, medium sized	50	.....	13	
Apple, medium sized	70	.....	17	
Banana	115	1	24	2
Prunes, 4, without sugar	30	.....	7	

Clear soups and broths made without rice or barley have practically no nutritive value.

The nutritive value of the "fodder" vegetables, such as spinach, string beans, asparagus, lettuce, celery, cauliflower, cabbage, egg plant, tomatoes and cucumbers, is so slight that they may be disregarded.

Pears and peaches have about the same value as apples of the same size.

[1] Crackers vary so much in size that they must be weighed to determine how many it takes to weigh an ounce.

[2] Bread, 1 slice four inches square and three eighths thick = 1 ounce.

[3] Zwieback, 1 slice = large slice.

[4] A tablespoonful means as in ordinary serving, not level.

[5] The lean of a lamb chop weighs about an ounce; so does a piece of meat about 1¼ inches cube or a thin slice of beef.

* These foods contain from one quarter to one half gram of fat in each of the quantities given.

If the urine increases with the increase in the water and the weight does not, the water should be further increased until considerable amounts are given. If the amount of urine does not increase or the weight increases, the water should be diminished. The normal difference between the water intake and the urine output in children, other things being equal, is somewhere between ten and fifteen ounces daily. When the kidneys show that they can eliminate water freely, the water should be increased up to several quarts daily.

The work of the kidneys may also be diminished by catharsis. In this way certain of the substances which would otherwise have to be excreted by the kidneys or retained in the body are eliminated. Water may also be disposed of in this way, but not as well as by diaphoresis. The bowels should be made to move several times daily, therefore, in every case of nephritis, and in severe cases with uremia free catharsis should be induced. The best drugs are those which cause large watery stools, such as compound jalap powder, licorice powder or elaterium. Enough must be given to attain the desired result, no matter how much is needed.

Edema is best removed by sweating. This is all that sweating does. It is certain that but little urea is eliminated in this way and there is no proof that toxic substances are excreted by the skin. Sweating should never be used, therefore, unless there is edema. It is far better and safer to induce sweating by the application of heat externally than by the administration of drugs internally. It is very difficult to keep a child in a hot air bath long enough to get good results, as it soon becomes restless and kicks the coverings loose. They object much less to hot packs. The child should be wrapped in a blanket and put in a tub of water between 105° F. and 110° F. and kept there for from ten to fifteen minutes. It should then be taken out, wrapped in a hot, dry blanket and kept surrounded by heaters from one half to two hours. Pilocarpine, the only drug powerful enough to be of any practical use, is a very dangerous remedy and should never be used, therefore, except in an emergency. When there is complete, or almost complete, suppression of urine, as sometimes happens in acute exudative nephritis, it is possible that poultices and hot applications to the loins and hot colonic flushings may relieve the congestion of the kidneys and thus hasten the reëstablishment of their excretory powers. Personally, I am rather skeptical. They should, however, be tried. It is also possible that bleeding may tide a child over threatened death from uremia. I have seen a number of cases in which this apparently happened. On the other hand, however, I have seen as many others, seemingly in just as serious a condition, who were not bled and who likewise recovered. Blackfan (Transactions of the American Pediatric Society, 1923, XXXV, 197) has recently taken advantage of the dehydrating action of magnesium sulphate in the treatment of uremia and has obtained some remarkable results. The cerebral symptoms have been relieved, the blood pressure has come down and the secretion of urine quickly reëstablished. He advises the use of a 2% solution of hydrated magnesium sulphate, injected intravenously at the rate of 10 c.cm. per minute and in amounts of about 15 c.cm. per kg. of body weight. The injections may be repeated at twelve or twenty-four hour intervals. Another procedure which is worthy of trial in such cases, if the intravenous injection of magnesium sulphate does not relieve the symptoms, is Edebohls' operation of splitting the capsule of the kid-

ney. This relieves congestion and gives the kidneys a chance to resume their functions. Like the treatment with magnesium sulphate, it does not cure the acute nephritis, but may tide the patient over the emergency.

There are, as already stated, no drugs which have any direct curative action in nephritis. Nevertheless, there are a number of drugs which are used very commonly as if they had. These may be divided into three classes; digitalis and its congeners, caffein and the preparations of theobromine, and the alkalies.

Digitalis and drugs of its class have no direct action on the kidneys, but increase the flow of urine by strengthening the action of the heart and thus sending more blood through the kidneys. The heart does not need stimulation in acute nephritis, however, and in the acute stage the kidneys are already engorged with blood. Any form of treatment, therefore, which increases the flow of blood to the kidneys at this stage, is not only irrational, but may be harmful. In the later stages it may no longer be harmful, but is unnecessary.

Caffein, theobromine and their preparations have a direct stimulant action on the renal epithelium. Their action on the heart is probably of no importance in this connection. Caffein by its action on the vasomotor center may cause such a contraction of the arterioles of the kidneys as to prevent any benefit from the stimulation of the epithelium. Theobromine and its preparations, such as diuretin, have no action on the vasomotor center and are thus preferable to caffein. In the acute stage the renal epithelium is in no condition to respond to stimulation and, moreover, stimulation may do harm by increasing the inflammation. In the later stages these drugs may be of use, but are usually not needed, if the diet and the intake of water are regulated.

It is almost certain that alkalies have no direct effect on the activity of the renal cells. Their action is probably the same as that of other diffusible bodies which are excreted by the kidneys and which during their excretion increase the flow of urine. The effect of the alkalies on the total nitrogen excretion is a very uncertain one. When the nitrogen of the urine is increased by their use, it is probably usually due chiefly to a flushing out of the tissues. In many cases the relative amount of urea is increased by the administration of alkalies. As the object of treatment at the most acute stage is to spare the kidneys, and as water at this time is irritating to the kidneys, it hardly seems rational to give alkalies at this time to increase the work of the kidneys. Later on, alkalies are often useful. They are, however, but little, if any, more effectual than the water in which they are given.

Children with acute nephritis should be kept in bed. They should be warmly dressed and guarded against exposure and chilling. They should be kept in bed until the albumin has disappeared from the urine and in many instances until the urine is microscopically clear. When they begin to get up, they must be most carefully guarded against exposure, their diet regulated almost as carefully as in the acute stage, water pushed and exercise limited. Their urine should not only be examined every few days for albumin, but the centrifugalized sediment should be examined under the microscope, even if there is no albumin in the urine. No child should be considered to have recovered from nephritis until its urine has been free from albumin and the sediment clear of blood cells and casts for at least three months after the child has been living under normal conditions.

Treatment of Subacute Nephritis

The treatment of subacute nephritis is along the same general lines as that of acute nephritis. The same care is necessary in regard to the diet and the regulation of the water intake. If the children are symptomatically well, it is not necessary to keep them in bed after a few months, but after they are up they must be guarded and watched in the same way as children convalescing from acute nephritis. The same rule must be followed in them also in determining when they are well.

Treatment of Chronic Nephritis

The treatment of chronic nephritis is also along the same general lines. It is not advisable to limit the diet quite so strictly, however, if the limitations prove too irksome, because when a disease is practically certain to be ultimately fatal, it is not fair to disregard the desires of the patient in the same way which would be obligatory, if there was a chance of recovery. The same rule should apply in the regulation of the child's life and amusement. When there is considerable edema or ascites, it is probable that life may be prolonged by splitting the capsules of the kidneys, as advised by Edebohls. I have seen one or two cases, which I thought were chronic diffuse nephritis, which recovered after this operation. I cannot help feeling, however, that the recovery was due rather to a mistake in the diagnosis than to the operation. The treatment of chronic interstitial nephritis in childhood is the same as in adult life. That of chronic nephritis with infantilism is essentially the same as that of chronic interstitial nephritis.

Finally, nephritis in childhood, at any rate in its severe form, is to a considerable extent a preventable disease. The prophylactic treatment is the most important. When nephritis has developed, the treatment is dietetic and hygienic. Drugs are of little use, except for the relief of symptoms. The intravenous injection of magnesium sulphate is the best procedure in the treatment of uremia and suppression of the urine.

PERINEPHRITIS

This disease consists of an inflammation of the cellular tissue surrounding the kidney. It may be secondary to disease of the kidney itself or apparently primary. The primary form is the more common in early life. It is equally frequent in boys and girls. It may end in resolution or go on to suppuration. In all the cases which I have seen it has been very acute, although subacute and chronic forms are described.

Secondary Perinephritis.—In my experience it is, when secondary, most often a complication of pyelitis and usually occurs early in the disease. The symptoms are at first indistinguishable from those of the pyelitis. The only difference is that they are all more marked than is usual in uncomplicated pyelitis. There is more marked tenderness over the kidney and after a few days moderate swelling develops in the region of the kidney. Operation in these cases has shown that the swelling is due to edema, not to pus. I have never known one to go on to suppuration. The treatment is the application of heat or cold over the kidney and the continuation of that for the causative pyelitis. It is wiser not to operate on this type of case. When perinephritis is due to the breaking through of a tuberculous process in the kidney or of an abscess secondary to a renal calculus, there is simply an increase in the symptoms already present, with finally the development of a tumor in the region of

the kidney from the formation of a perinephritic abscess. Operation is, of course, obligatory. Inflammation about the kidneys in connection with tuberculosis and renal calculi, which does not go on to the formation of pus, is unrecognizable.

Primary perinephritis is most often due to trauma, but sometimes apparently results from chilling. In other instances there is no evident cause. The onset is often with a chill, followed by a considerable rise in temperature. The temperature is irregularly high and the child shows all the characteristic symptoms of an acute infection. There is a marked polynuclear leucocytosis. In other instances, however, the onset is less acute. It most instances the child complains of pain, which it more often locates in the abdomen than in the flank. It often holds the back rigidly and sometimes keeps the thigh on the affected side flexed. Motion of the spine, and sometimes of the hip, causes pain. There is usually tenderness on pressure in the loin. In other instances, however, there are for some days no symptoms or signs to suggest in any way where the inflammatory process is located. Even in these cases, however, localizing symptoms develop after a few days. There is almost always some swelling in the region of the kidney, which, if an abscess develops, may become considerable. In the majority of instances, however, the inflammation gradually quiets down without the formation of an abscess. If an abscess forms, it may point in the loin, burrow down along the psoas muscles or break into the peritoneal cavity. None of these things ought to happen, however, because the condition should have been recognized and the abscess opened before it has had time to point or burrow.

Diagnosis.—The only things with which acute perinephritis is likely to be confused are appendicitis and acute osteomyelitis of the spine or hip. Careful study of the location of the tenderness and muscular spasm, and the findings on rectal examination, usually make the diagnosis between perinephritis and appendicitis easy. In osteomyelitis of the spine the tenderness is over the spine, not in the loin, and there is no swelling in the loin. In osteomyelitis of the hip all motions of the hip, as well as those which bring strain on the psoas muscle, are limited, while the tenderness and swelling are about the hip joint, not above it. Roentgenograms of the bones also show evidences of lesions in them. It is conceivable that subacute cases of perinephritis inflammation may resemble more closely diseases of the hip and spine, but the diagnosis should not be difficult along the same lines. I have never seen any of these subacute cases.

Treatment.—Absolute rest in bed is necessary. Heat or cold, according to which gives the more comfort, should be applied over the kidney. The treatment is otherwise entirely symptomatic. If it is believed that the inflammation is purulent, operation should be performed at once. It is better to operate unnecessarily than to let an abscess have a chance to burrow.

MALIGNANT DISEASE OF THE KIDNEY

Benign tumors of the kidney in childhood are so uncommon that it is safe to consider every new growth of the kidney in early life malignant. Malignant disease of the kidney is very uncommon after six years of age. More than half of the cases occur in the first two years. It may even be congenital. It occurs with about equal frequency on each side and is equally common in boys and girls.

Histologically, the tumors are varied in character and usually mixed. In most instances they originate in foci of embryonal tissue and belong in

the class of the adenosarcomata. In other instances they originate in foci of adrenal cells, which were caught and embedded in the kidney. In such cases the tumors belong with the hypernephromata. The tumor may start in any part of the kidney. Its growth is usually quite rapid and it may become large enough to fill the greater part of the abdomen. It is usually hard and nodular, but may be smooth, and sometimes is soft and semifluctuant. Localized peritonitis is quite common and results in the formation of adhesions to the neighboring organs. Metastases are common. In the usual type they occur most often in the liver and lung. In the hypernephromata they occur most often in the flat bones, especially those of the skull. A tumor of the skull in an infant or child, especially if it involves the orbit, is almost always a malignant tumor, secondary to a hypernephroma of the kidney. If it is not that, it is usually a chloroma. Metastases in the liver often grow more rapidly than the original tumor in the kidney and dominate the picture. Metastases in the lungs usually cause few symptoms and frequently are not recognized.

FIG. 142.—Metastatic tumor of lung.

Symptomatology.—The first symptom usually noticed is enlargement of the abdomen. This enlargement is, because of the inability of the average person to see anything that is not pointed out to him, usually considerable when it is first noticed. Questioning, however, generally reveals that the child has been losing color and has not appeared as well as usual for some time. As the tumor increases in size, it causes disturbances of digestion and constipation by pressure on the stomach and intestines. It may also cause edema of the extremities from pressure on the inferior vena cava, and disturbances of the respiration, and sometimes of the cardiac action, by pressure on the diaphragm. Toward the end loss of weight and strength is very rapid. In my experience, pain in the abdomen is unusual, even in those cases in which there is peritoneal inflammation and adhesions. Abdominal discomfort from the pressure and weight of the tumor is, however, present in most cases. Hematuria, either macroscopic or microcsopic, may occur and may be one of the earliest symptoms. In my experience, however, it is uncommon. There is usually no fever, but often a moderate polynuclear leucocytosis.

Prognosis.—The duration of life from the time of the discovery of the tumor varies between two months and two years. The end, unless the tumor is removed, is invariably in death. The chances of recovery are, however, but little better when the tumor is removed. I have known of but one case in which death did not occur as the result of recurrence, either in the other kidney or in the neighborhood of the kidney which had been removed. The chances of recovery under treatment with the Roentgen ray or radium are no better than when the kidney is removed, although temporary improvement is not uncommon.

Diagnosis.—Tumors of the kidney originate in the kidney region and grow in the only directions they can, forward and into the flank. They can, therefore, be grasped between the hands, one in the flank and the other on that side of the abdomen. They can be moved a little, but not much. They do not move with respiration. The intestines are in front of them. Examination with the fluoroscope or a Roentgenogram after a bismuth or barium enema often makes it easier to determine the relations of the tumor and the colon. There is sometimes a little difficulty in distinguishing between a tumor of the right kidney and an enlargement of the right lobe of the liver. The enlargement of the liver is in the position of an enlarged liver, that of the kidney in the position of an enlarged kidney. The edge of an enlarged liver is distinct and superficial. The enlarged kidney does not have an edge and it is deep. The liver moves with respiration, the kidney does not. The intestines are behind the liver and in front of the kidney. There is sometimes difficulty in distinguishing between a tumor of the left kidney and an enlarged spleen. The enlarged spleen is superficial and in the position of a spleen. The surface is smooth, there is a definite rounded edge and often a notch. The enlargement does not extend deeply into the flank. The tumor of the kidney is deep down, it does not have a distinct edge, its surface is usually rough and nodular, there is no notch, it can be felt deep in the flank. The intestines are behind an enlarged spleen and in front of a tumor of the kidney. When the spleen is much enlarged, there are likely to be, moreover, characteristic changes in the blood. It is sometimes very hard to distinguish between masses of tuberculous tissue in the peritoneum or greatly enlarged retroperitoneal glands and a tumor of the kidney. Tuberculous masses, however, almost never extend as deeply into the flank and are usually rather more movable. Masses of retroperitoneal glands, while they originate deep down, do not come from the flank in the same way as do tumors of the kidney. They are nearer the median line and grow more directly forward. It is sometimes difficult to distinguish between a malignant tumor of the kidney and a hydronephrosis, the only differences in the physical signs being that a tumor is usually less regular in outline and a hydronephrosis usually fluctuant. The fever, leucocytosis and other evidences of a purulent focus in the body are sufficient to make the diagnosis of pyonephrosis easy. Malignant disease of the kidney may be confused with congenital cystic disease of the kidneys. In cystic degeneration of the kidneys both kidneys are involved and the enlargement is present at birth, while in

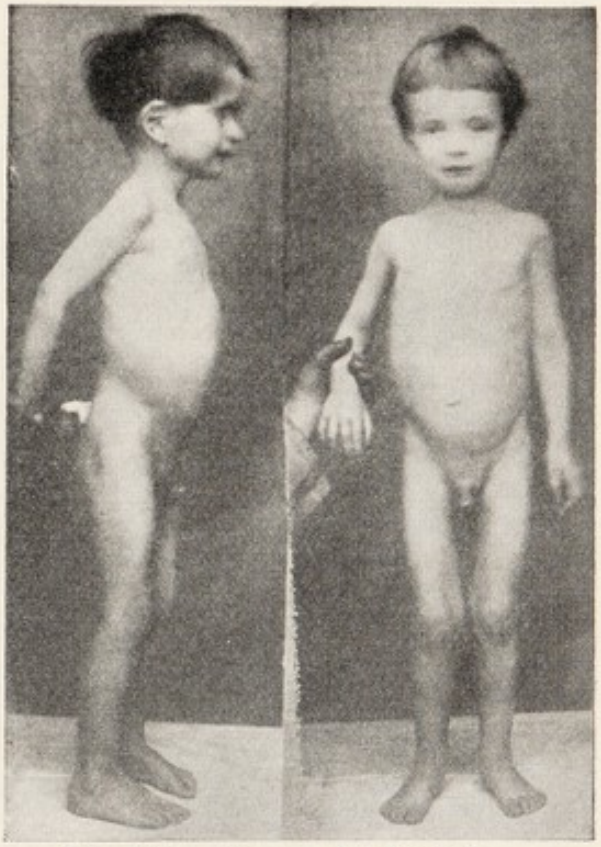

FIG. 143.—Tumor of right kidney.

malignant disease one kidney only is involved and the enlargement develops after birth.

Treatment.—Nothing can be hoped from medicinal treatment. osteopathy, chiropractic or Christian Science in these cases. Treatment with the Roentgen rays and radium may diminish the size of the tumor temporarily, but only delays the fatal termination. The only thing which offers any chance whatever is the removal of the tumor. This must be done early. Even then the tumor almost always recurs at the site of the former tumor or in the other kidney. In an occasional instance, however, there is no recurrence. Every child with malignant disease of the kidney should be given, therefore, the little chance that removal offers. If the operation is delayed, many adhesions are likely to be formed, which render the operation fatal or impossible. Moreover, if the tumor is very large, death is likely to occur from shock. It is useless, of course, to operate if there are evidences of metastases in other organs.

TUBERCULOSIS OF THE KIDNEYS

There are often many miliary tubercles in the kidneys in acute miliary tuberculosis and frequently a few small tuberculous masses in them in chronic diffuse tuberculosis in both infancy and childhood. They cause no symptoms, however, and have no appreciable effect on the course of the disease.

Local tuberculosis of the kidney may also occur in early life. Its frequency increases directly with age, but it is uncommon at any time. I have, however, seen two cases in infants under one year of age. It is usually limited to one side. The infection is seldom ascending. Coming through the blood stream, as it does, the cortex is usually first involved. The disease process usually extends to the pyramids and calices and finally to the pelvis. There are usually no symptoms to draw attention to the urinary tract until the pelvis is involved. When this happens, the urine contains pus and usually caudate and round cells. Macroscopic blood is occasionally present in the urine. It is often present microscopically. There may be pain and tenderness in the region of the kidney. Sometimes, when the kidney has been practically destroyed, and a large tuberculous abscess formed, or when there is perinephritis, there may be a tumor in the lumbar region. Micturition is often increased in frequency and is sometimes painful. These symptoms are present even when there is no secondary involvement of the bladder. As a matter of fact, the bladder is seldom involved in tuberculosis of the kidney in early life.

Diagnosis.—Tuberculosis of the kidney is likely to be mistaken for pyelitis, which is a much more common condition and in which the characteristics of the urine are the same, except that blood is more common in tuberculosis of the kidney than in pyelitis. The symptoms of the two conditions are almost identical. A negative tuberculin test excludes tuberculosis of the kidney. A positive tuberculin test is in favor of it, but does not prove it. The only ways in which a positive diagnosis can be made is by finding tubercle bacilli in the urine or by positive anmial inoculations. Care must be taken, as always when looking for tubercle bacilli in the urine, not to mistake smegma bacilli for them.

The prognosis and treatment are the same as in adults. As in adults, the chances of recovery, even when only one kidney is involved and it is

removed, are poor. Nevertheless, I have known of a case of tuberculosis of the kidney, proved by positive animal inoculations, in an infant to recover entirely without operation.

AMYLOID DISEASE OF THE KIDNEYS

Amyloid disease of the kidneys occurs under the same conditions as amyloid disease of the liver, spleen and other organs. The kidneys are somewhat enlarged, but usually not palpable. The urine is ordinarily increased in amount, low in specific gravity, and contains a small amount of albumin and a few cells, hyaline and granular casts.

The condition is nowadays an uncommon one, but is not infrequently overlooked or forgotten. Recovery is possible, if the cause of the amyloid degeneration can be removed and the degenerative process has not gone too far. The treatment consists in the removal of the cause. There is no treatment for the degenerative process in the kidney

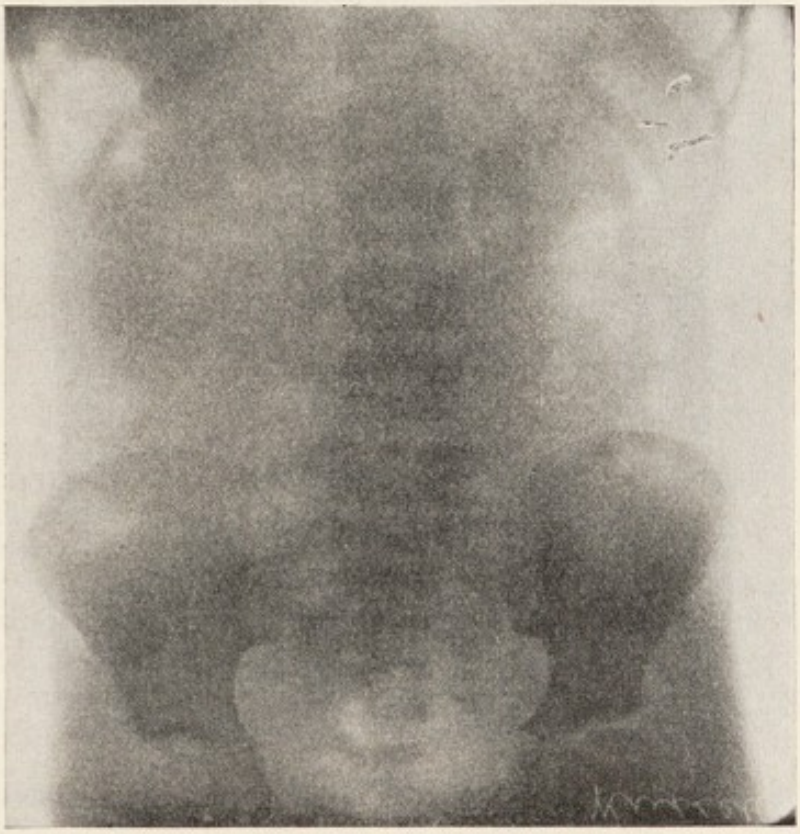

FIG. 144.—Renal calculi.

CALCULI

Renal Calculi.—Uric acid infarctions and small granules of uric acid in the renal pelves are not uncommon in infancy. They may cause pain while passing through the ureters. In infancy the pain which they cause is indistinguishable from that of intestinal colic. Stone in the kidney is rare in childhood. There are usually no symptoms unless the stone attempts to pass through the ureter. The symptoms then are the same as those of renal colic in the adult. It must always be remembered that any severe or intermittent pain in the abdomen or back in a child may be due to a renal calculus, although the chances are very much against it. The differential diagnosis between renal colic and other severe pains is the same in childhood as in adult life. The Roentgen ray may help in

the diagnosis. As in the adult, however, not all stones are revealed by the Roentgen ray. In rare instances a stone in the kidney may cause pyelitis, or even an abscess of the kidney. A stone may become impacted in the ureter and cause hydronephrosis or pyonephrosis, as in the adult.

The treatment of an excess of uric acid in the urine in infants is by giving large amounts of water and the administration of any of the alkaline salts in doses large enough to make the urine slightly alkaline. The treatment of renal colic is the same in childhood as in adult life. If the Roentgen ray shows that there is a stone in the kidney too large to pass through the ureter, it is advisable to operate at once in order to prevent the development of complications.

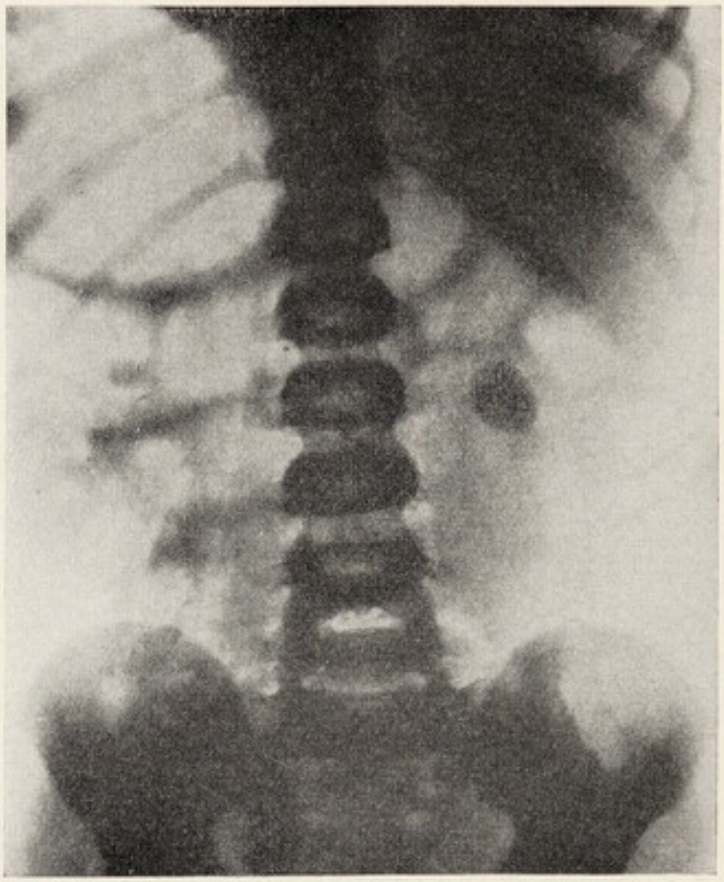

FIG. 145.—Renal calculi.

Vesical Calculi.—These are usually the result of the passage of a small renal calculus into the bladder. They are not very common. They are more common in boys than in girls, because the short, wide urethra of the girl allows the passage of many small calculi, which must necessarily remain in the bladder of the boy. One of the most characteristic symptoms of stone in the bladder in childhood is pain in the lower abdomen on jumping. In many instances there is frequent micturition. Not infrequently there is pain during micturition and sometimes the passage of the urine is interrupted. If there is much straining, there may be prolapse of the rectum. Erections of the penis are not very unusual. If the bladder is inflamed, the urine contains albumin, pus, blood and squamous epithelial cells.

The stone may sometimes be felt in small children by bimanual examination, one finger being introduced into the rectum. The Roentgen

ray reveals many of them. Others can be discovered by cystoscopy. The treatment is, of course, surgical.

HYDRONEPHROSIS AND PYONEPHROSIS

In early life these conditions are simply pathologic curiosities, although they are both occasionally seen. They are due to the same causes as in the adult. An additional cause in male infants is an exaggeration of the normal folds in the posterior urethra which, by increasing the resistance to the passage of urine, brings on dilatation of the bladder and finally of the ureters and kidneys. The symptoms of hydronephrosis and pyonephrosis in early life are the same as in adult life. Children may even have Dietl's crises in intermittent hydronephrosis. The physical signs are also the same as in later life. When a tumor, evidently containing fluid, is found in the flank or anterolateral portion of the abdomen, some form of nephrosis should be considered, therefore, in the differential diagnosis. The treatment of these conditions is, of course, purely surgical and on the same principles as in adults. The prognosis is also the same. Infants with double hydronephrosis from obstruction in the urethra, however, usually die early. In these cases the treatment must be directed, of course, to the removal of the obstruction.

FLOATING KIDNEY

This is a rare condition in childhood and, as in adults, occurs more often in females than in males and more often on the right side than on the left. It is probably a congenital malformation, but, nevertheless, it is seldom discovered before middle childhood. I have seen a number of cases. About one half of them had had no symptoms and were discovered during a routine physical examination. The others had all had pain in the lumbar region and lateral abdomen and several had had fairly marked Dietl's crises. Floating kidney should, therefore, always be thought of as a possible cause of intermittent abdominal pain, especially in girls.

Diagnosis.—A floating kidney may be suspected, if there are pain and discomfort in the flank and anterolateral portion of the abdomen. Children are seldom observant enough to describe dragging or bearing down sensations. It should be strongly suspected, if there are symptoms similar to those of Dietl's crises. The diagnosis of floating kidney can only be made positively, however, by palpating the kidney in an abnormal position. Being able to feel the lower pole of the kidney in late childhood does not justify the diagnosis of floating kidney. It can be felt in most young women and in many girls, especially if they are thin. The displaced kidney has the shape and feel of a kidney, is movable and can be pushed back into position. At times it is palpable and at others it is not. It is difficult to mistake a floating kidney for anything else. Other things may be mistaken, however, for a floating kidney. Among them are mesenteric cysts, tuberculous masses and masses of feces. Mesenteric cysts are movable, but do not have the shape or feel of a kidney and cannot be pushed up into the kidney region. Tuberculous masses and feces almost never have the shape of a kidney. Tuberculous masses are usually little, if at all, movable. Masses of feces can often be dented by pressure and, although often freely movable, cannot be pushed up into the kidney region. They disappear when the intestines are thoroughly cleaned out.

Prognosis and Treatment.—If a floating kidney is discovered by accident in the course of a routine physical examination, no attention

should be paid to it and no treatment instituted. If there have been any symptoms from it, an attempt should be made to keep it in position by a belt. If the belt does not hold it, or there have been severe attacks of pain, it is advisable to attempt to fix it in place. Operations of this sort are, however, often not successful, because the displacement of the kidney, usually being simply one of the manifestations of a general laxity of the abdominal organs, is liable to recur.

PYELITIS

This term is commonly applied to a mild inflammatory condition of the lining of the urinary tract due to bacterial infection, as the result of which the urine contains either or both bacteria and pus. This condition is also sometimes known as pyelocystitis. Other more severe inflammatory conditions of the pelves of the kidneys may occur as the result of calculi, tuberculosis of the kidneys or infection from single or multiple abscesses of the renal tissues They are relatively uncommon, however, and are not what is referred to when pyelitis and pyelocystitis are spoken of in early life. Pyelitis is more common in infancy than in childhood and much more common in girls than in boys. The pathologic changes in these cases are slight. There is some reddening and swelling of the mucous membrane of a part or of the whole of the urinary tract, some desquamation of the epithelium, and sometimes the evidences of degeneration of the lower tubules of the kidney.

Etiology.—In the vast majority of cases the causative organism is one of the colon bacillus group, although many other organisms have been found occasionally. When the infection is secondary to diseases of the intestinal tract, the organism is almost invariably the colon bacillus. So also when the condition is apparently primary. When it is secondary to other infections, especially those of the nose and throat, the organism is not infrequently a streptococcus or some form of staphylococcus.

Infection of the urinary tract can occur theoretically in three ways; through the urethra, through the surrounding tissues by way of the lymphatics and through the blood; ascending, transparietal and hematogenous, or descending. It is probable that it does take place through all these channels. The predominance in girl babies suggests that it is most often ascending through the urethra. When pyelitis occurs in connection with diarrhea in either sex, the infection is probably most often ascending, but sometimes transparietal. When it occurs after infections elsewhere, it is probably almost always hematogenous in origin. When it is apparently primary, the infection may occur in any way, but is probably usually ascending, especially if the patient is a baby girl.

Symptomatology.—In the majority of cases in infancy there is nothing whatever in the symptomatology to call attention to the urinary tract, the symptoms being merely an elevation of temperature and those common to all febrile disturbances in infancy, such as restlessness, drowsiness, fretfulness and indefinite signs of discomfort. In some instances a yellow stain on the napkin first calls attention to the urinary tract. Symptoms of disturbance of the gastro-intestinal tract, sometimes primary, sometimes secondary, are especially common. Anorexia is the rule and is often very marked. Vomiting is not uncommon. The stools are usually abnormal, sometimes as the result of some disease to which the urinary infection is secondary, sometimes as the result of the infection itself.

The temperature is usually very irregular and in no way characteristic. It suggests confined pus more than anything else. In some instances it

suggests an atypical malaria and on this account pyelitis is not infrequently mistaken for malaria. This is especially likely to happen when, as is sometimes the case, there are also chills and sweating. In some

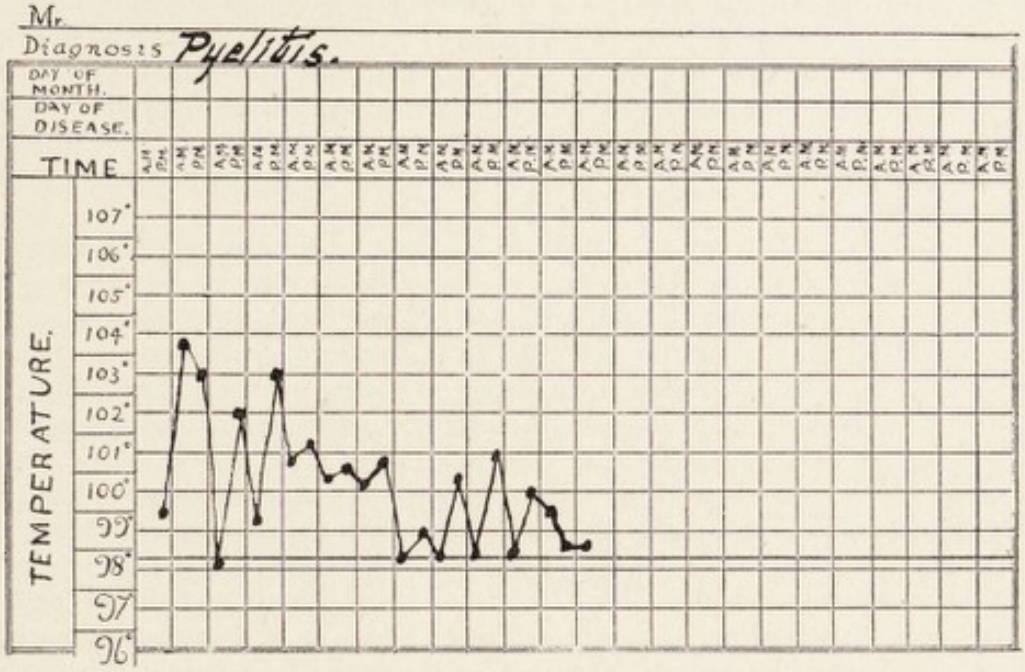

FIG. 146.—Pyelitis in infant.

cases the temperature is more regular, while in others there is little or no fever.

In some instances it is evident that the baby is suffering pain somewhere. It is very seldom, however, that there is anything to suggest that

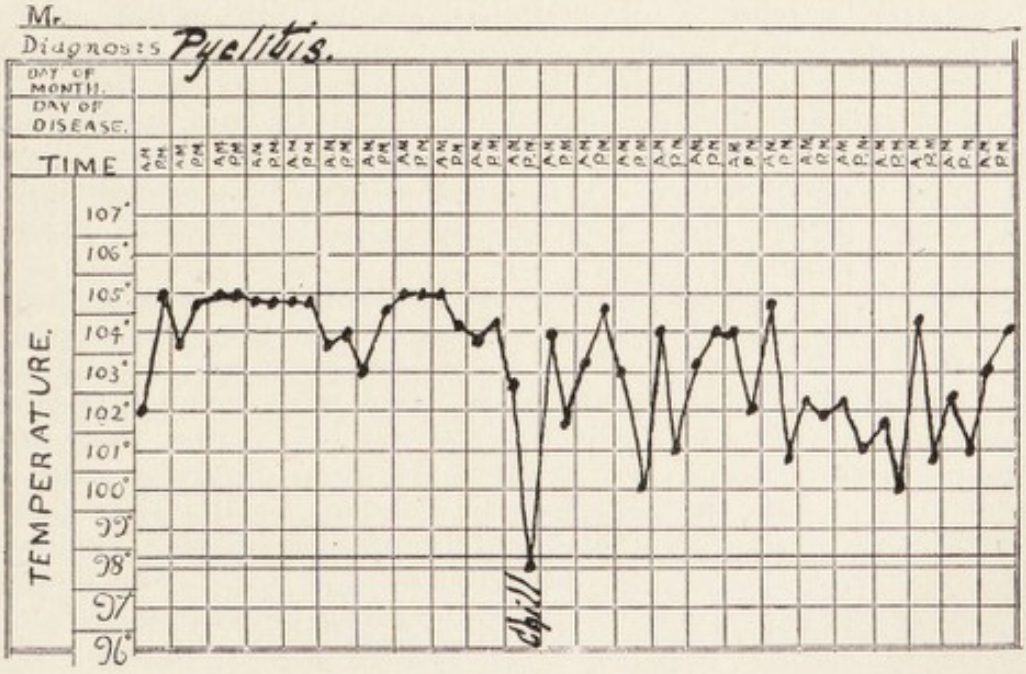

FIG. 147.—Pyelitis in infant.

the location of the pain is in the bladder or kidneys. The pain is often paroxysmal. In certain instances it is apparently due to the temporary blocking of the ureters by masses of pus. At any rate, during the attack of pain there is sometimes swelling and tenderness over the kidneys and

relief follows the passage of considerable amounts of urine containing macroscopic lumps of pus.

Tenderness and enlargement of the kidney are unusual. If they are present, they are sometimes due to a nonsuppurative perinephritic inflammation. Tenderness over the bladder is somewhat more common. Micturition is usually not increased in frequency, sometimes there is partial retention. Painful micturition is uncommon. Loss of weight is usually rapid and marked. Anemia develops rapidly. There is almost always a polynuclear leucocytosis, which may be quite marked, even as high as 50,000.

The symptomatology in childhood is not much more characteristic. There is, however, more often pain in the kidney region, tenderness over the kidneys and noticeable disturbances of micturition. Enuresis, both by day and by night, is sometimes the most marked symptom.

Urine.—In most instances the urine is pale and uniformly cloudy or turbid. The turbidity is due in part to the presence of bacteria and in part to that of large numbers of pus cells, chiefly to the latter. In rare instances the urine is of a peculiar gelatinous appearance. The odor varies from normal to stale or very foul, but is usually normal. The specific gravity is usually not increased. The reaction is almost invariably acid, not infrequently highly so. When the infection is with the Bacillus coli, the acidity is due to the fact that, as it does not decompose urea, it does not develop an alkaline reaction in the urine. The acidity is inimical to the growth of other organisms, so that the Bacillus coli is most often found in pure culture. The urine usually contains less than 0.1% of albumin.

Microscopically, the sediment is composed almost entirely of pus cells, usually single, sometimes in clumps. Caudate, small round and squamous cells, in small numbers, are also present in most cases. Squamous cells are never present in large numbers, as they are in most cases of purulent inflammation, probably because of the absence of the ammoniacal products of the decomposition of urea, which are the cause of the destruction and desquamation of the bladder epithelium in other infections. Hyaline and fine granular casts are occasionally seen, blood or blood elements almost never. The amount of urine is apparently uninfluenced by the disease and varies directly with the amount of liquid ingested.

Bacteriuria.—By bacteriuria is meant a condition characterized by the presence of a very large number of bacteria in the freshly passed urine and the absence of marked symptoms of an inflammatory process in the mucous membrane of the urinary tract. It is most common in infancy and is almost always due to the Bacillus coli. As a rule, there are no symptoms beside malaise and a slight elevation of temperature, but sometimes the symptomatology may be exactly the same as in pyelitis. The urine is uniformly cloudy, having the appearance of a bouillon culture of bacteria. The odor is unpleasant, the reaction acid. This condition is frequently overlooked, because, when no pus is found in the urine, the bacteria are attributed to the age of the urine and decomposition. Whenever the symptoms suggest an infection of the urinary tract and bacteria are found in considerable numbers in the urine, a fresh specimen should always be examined to determine whether they are the cause of the symptoms or simply the result of decomposition.

Prognosis.—The prognosis as to recovery in pyelitis is very good, a fatal termination being very unusual. There are two main types of the

disease. In one, recovery ensues in two or three weeks under any form of treatment. It is likely that recovery would occur in these cases without treatment. The other type drags on for weeks and months in spite of all sorts of treatment. Sometimes it persists more or less intermittently for several years. Exacerbations are common and relapses not rare. Babies and children who have had pyelitis are apparently more likely to have subsequent attacks than children who have never had it. Perinephritis, which seldom goes on to suppuration, is a rare complication.

Diagnosis.—It is evident from the description of the symptomatology that in the majority of cases of pyelitis in infancy, as well as in a considerable number of those in childhood, there is nothing about it to direct attention to the urinary tract. When such symptoms are present, they are usually mild and are easily overlooked. In childhood the sudden onset of enuresis should always suggest the possibility of pyelitis. The symptoms being so indefinite, the urine should be examined, therefore, in all diseased conditions with indefinite symptoms in infancy, especially if febrile, and no physical examination should be considered complete or diagnosis satisfactory in obscure conditions in either infancy or childhood, unless the urine has been examined. As a matter of fact, in the vast majority of cases of pyelitis the diagnosis can only be made by the examination of the urine.

It must be remembered, however, that as one swallow does not make a summer, neither do a few pus cells in the urine of a girl justify the diagnosis of pyelitis. My experience has been that physicians who do not examine the urine as a routine measure constantly miss pyelitis, while those who do are quite likely to make the diagnosis of pyelitis on insufficient grounds. The finding of clumps of pus is strongly suggestive of pyelitis, but does not of itself justify the diagnosis. It must further be remembered that in some instances pus is present only intermittently in the urine. If there is no other evident cause for the symptoms, it is not justifiable, therefore, to exclude pyelitis on a single negative examination.

Treatment.—There are several methods of treatment. This of itself suggests very strongly that none of them is very effective. The only method of treatment which everyone agrees does good is the administration of large amounts of water. Large amounts of water should, therefore, always be given. The patient should be put to bed and kept there, at least as long as there is any fever. The bowels should be kept open. It is not necessary to limit the amount of protein in the diet, as so many physicians believe, because pyelitis is not a disease of the renal tissues, but simply a mild inflammatory condition of the urinary passages. Search should be made for any possible causative condition and treatment for it instituted.

In my experience, pyelitis is more likely to be helped by the *alkaline treatment* than by the others. Consequently, I try it first. It is idle to give a prescribed number of grains of one of the alkalies, as is usually done, and let it go at that. To do any good, sufficient alkali must be given to make the urine alkaline. This can only be determined by having the urine tested repeatedly with litmus paper. I always give mothers and nurses litmus paper and explain its use to them so that they can keep the urine alkaline. It makes no difference what alkali is used. Bicarbonate of soda is cheaper than either the citrate or the acetate of potash and equally effective. Much larger doses are required than are usually given. No matter how much is needed, enough must be given to make the urine alkaline.

Another method of treatment is with hexamethylenamine, which is better than any of the other drugs of its class. Roughly, it may be given in doses of one grain per year of age, three or four times daily. It must never be forgotten that hexamethylenamine is a powerful drug and that it may cause irritation of the urinary tract. The urine should, therefore, be watched constantly for the appearance of microscopic blood when this drug is used. It must also never be forgotten that hexamethylenamine is not itself a urinary antiseptic. It must be broken down and formaldehyde set free before it can do any good. Hexamethylenamine is only broken down when the urine is acid. It is absolutely useless, therefore, to give it, as is so often done, combined with an alkali. If the urine is not strongly acid, acid sodium phosphate or benzoate of soda should be given at the same time. Enough of these drugs should be given to make the urine strongly acid. Ten or fifteen grains a day of either of these drugs is usually sufficient. The dose should be increased, however, until the desired result is obtained. When hexamethylenamine is given, Burnham's test should be used in order to determine whether it is being broken down. This test is performed by adding five drops of a 0.5% solution of phenylhydrazine hydrochloride and a crystal of sodium nitroprusside to 5 c.cm. of urine and shaking. A few drops of a concentrated solution of sodic hydrate is then added. If formaldehyde is present, a blue color results, which slowly passes through blue to brown, red, and finally yellow. If this test is not positive, the hexamethylenamine is doing the patient no good.

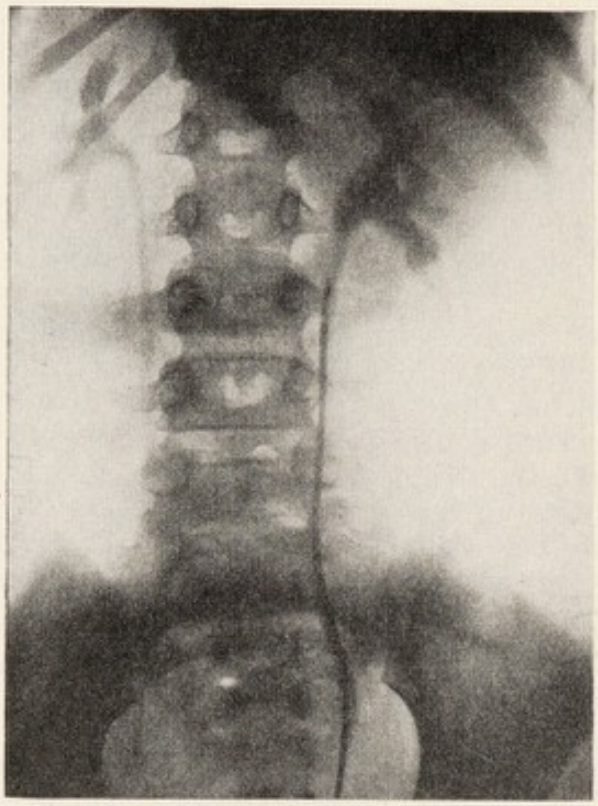

FIG. 148.—Pelves of kidneys in severe chronic pyelitis in child.

Freeman (American Journal of Diseases of Children, 1913, VI, 117) has recommended the use of large doses of hexamethylenamine in obstinate cases and has obtained some striking results by this method of treatment. He recommends doses as large as twenty-five grains a day for a baby of six months, and of from thirty-five to forty-five grains a day for infants between nine and twelve months, with correspondingly large doses for children. When such large doses are used, the urine should be examined several times daily for the presence of blood. If blood is found, the hexamethylenamine should be stopped at once. I have seen some remarkable results from this treatment, but in one instance death occurred within twenty-four hours after beginning it, without any apparent reason. Unfortunately, no autopsy was obtained in this case.

Another method of treatment is by *alternating the reaction of the urine.* It should be made strongly alkaline for three or four days, then acid for three or four days. If it is not normally acid, it can be made so by giving

either acid sodium phosphate or benzoate of soda. This alternation of the reaction should be kept up for several weeks.

Still another method of treatment is with *vaccines*. It is useless to give stock vaccines of colon bacilli, because there are so many strains. If a vaccine is used, it must be autogenous. I have never seen any good results from their use.

If one method of treatment is not successful after about two weeks, it is advisable to try another. When all have been tried, all that can be done is to start over again.

Local treatment is possible, but out of the question for anyone but an expert. It is useless to treat the bladder alone, because the whole urinary tract is involved. To be effectual, therefore, the ureters must be catheterized and the pelves of both kidneys treated, as well as the bladder. In a disease which is so seldom fatal and in which local treatment is so difficult, it is hardly necessary to consider it except in very obstinate and serious cases.

URETHRITIS AND VULVOVAGINITIS

The tendency of most physicians is to think at once that urethritis and vulvovaginitis in infancy and childhood are gonorrheal in origin. Urethritis in male infants is usually not gonorrheal, but is the result of local irritation from the passage of highly acid or alkaline urine or crystals, or the extension into the urethra of a balanitis. The balanitis is usually the result of ammoniacal decomposition of the urine and is more common in the circumcised. In boys urethritis is usually gonorrheal in origin and in them is usually the result of direct contact or of attempted sexual intercourse, often not of their own volition. Urethritis in females, whether infants or children, is uncommon, except in connection with vulvovaginitis. Like urethritis in boys, vulvovaginitis is not always gonorrheal. It may be due to lack of cleanliness, the irritation of rough clothing, or sometimes to pin-worms which have wandered forward. It is impossible to tell from the macroscopic appearance of the discharge whether the condition is gonorrheal or not, although the chances are that it is, if it is profuse and purulent. The diagnosis must always be made by microscopic examination. It must not be forgotten that the discharge may be slight and intermittent in gonorrheal vulvovaginitis. It must also be remembered that the disease is very persistent and that after the first few weeks it may cause no discomfort. On this account no girl should be admitted to a children's hospital without a very careful local examination. If there is any discharge, she should be refused admission unless bacteriologic examination shows that the condition is not gonorrheal, or, if too ill to be sent home, she should be isolated.

Gonorrheal vulvovaginitis in babies and little girls is less often due now to direct sexual contact than in the past, because the superstition that gonorrhea can be cured by direct contact with a virgin of the opposite sex is less prevalent now than it was a number of years ago. In most instances in babies and little girls the disease is contracted by sleeping with the mother or an older sister with the disease. In middle childhood, and not infrequently in early childhood, it is most often acquired from toilet seats, as the child in using them necessarily rubs the external genitals over the front of the seat. In later childhood, gonorrheal vulvovaginitis is most often due to sexual intercourse, the initiative

in these cases usually coming from the patient. In both sexes gonorrhea may sometimes, but rarely, be transmitted by towels or clothing.

Complications are unusual, either in urethritis in boys or vulvovaginitis in girls. The greatest danger is of infection of the eyes. Gonorrheal arthritis is uncommon in early life. Infection of the Fallopian tubes is uncommon in childhood. I have, however, seen several cases of gonorrheal peritonitis.

Treatment.—The treatment of urethritis and vulvovaginitis in infants and children is along the same lines as in adults. I have had little experience with it in recent years and, therefore, shall not attempt to describe it. The most important thing to be remembered, however, in the treatment of vulvovaginitis in little girls is that it must be continued for a long time. Relapses may occur many weeks, or even months, after apparent cure. The child should, therefore, not be allowed to pass from observation until at least a year has elapsed from the last appearance of any discharge.

ENURESIS

This term is applied to the involuntary passage of urine at an age when the bladder should normally be under control. Enuresis may be diurnal or nocturnal. It is most common between the ages of three and seven years. When the involuntary passage of urine is due to some malformation of the urinary organs or to organic disease of the spinal cord, it is not usually spoken of as enuresis. The term is not applied to the dribbling away of urine, but is restricted to the intermittent involuntary passage of reasonably large amounts of urine.

At birth urination is purely a reflex act through a center in the lumbar cord. There is no cerebral control. Control of the lumbar center is gradually acquired by a cerebral center, first when the child is awake, finally during sleep. The time at which cerebral control is established normally varies materially in different children and may be hastened by education.

It is evident that, even after the cerebral control has been acquired, it is not as active as it eventually becomes and, consequently, may be easily overcome during early childhood. The cerebral control is usually established during the day before the baby is two years old, and often earlier, if much attention has been paid to training the baby. It is often established at night by two and one half years. If it is not established by three years, the condition is abnormal.

Etiology.—It is evident that the normal cerebral control of the lumbar center may be overcome, if increased stimuli to reflex contraction of the bladder are sent to the lumbar center as the result of increased irritation or irritability of the peripheral nerves of the genito-urinary organs or the rectum. It is also evident that, if the irritability of the spinal center is increased for any reason, it will respond to lesser stimuli and will be controlled with more difficulty by the cerebral center. It is also evident that anything which interferes with the normal action of the cerebral center allows the lumbar center to take control and results in the reëstablishment of the infantile condition. The following table shows the etiology of enuresis on this basis. It is possible that I may have copied this table from someone else. If so, it was so many years ago that I have forgotten from whom it was borrowed.

TABLE XXXII

Etiology of Enuresis:

- Interference with Cerebral Control
  - Persistence of Infantile Condition
  - Deep Sleep
  - Adenoids
- Increased Irritability of Spinal Centers
  - Heredity
  - Nervousness
  - Malnutrition
  - Anemia
  - Diseases of any sort
- Increased irritation or irritability of peripheral nerves. Results in increased stimuli to reflex contraction. Often associated with weak sphincters and irritability of the bladder muscles.
  - Bladder
    - Urine + (Excess of liquids, Diabetes)
    - Urine − (Acid, Alkaline)
    - Inflammation of
    - Stone
    - Contraction of
  - Urethra
    - Inflammation of
    - Masturbation
    - Caruncles
  - Penis
    - Adherent prepuce
    - Phimosis
    - Balanitis
    - Narrow meatus
  - Vagina, etc.
    - Adherent prepuce
    - Phimosis
    - Balanitis
    - Vaginitis
    - Vulvitis
  - Rectum
    - Polyps
    - Fissures
    - Constipation
    - Pin-worms

It hardly seems necessary to take up these various causes in detail, as it is self-evident what they are and that many of them may be the result of other causes. The most common cause of enuresis, certainly at night, is the persistence of the infantile condition. Disturbances of nutrition are probably the next most common cause, while masturbation and a long prepuce are very seldom responsible.

Prognosis.—The prognosis in general is good. It is very seldom that recovery does not take place before puberty. If enuresis continues after puberty, the outlook is not so good, but even then the habit very seldom persists into adult life. If it continues after puberty, it is most often due to a small bladder. In general, if recovery does not take place before adult life, the trouble is really not enuresis, but is the result of some unrecognized malformation of the urinary organs, disease of the spinal cord, or cerebral defect. The outlook for rapid recovery is better in young children, that is, in those under five or six years, than in older children, because in them the habit has become less firmly established. It is much better, if there is some local cause which can be removed. It also depends considerably on how much trouble the child and its parents are willing to take in carrying out the treatment.

Treatment.—It must always be remembered that nocturnal enuresis is an involuntary act, entirely beyond the control of the child. Children

should never be punished, therefore, for wetting the bed. It sometimes does good, however, to praise them and reward them if they do not. A different attitude should be taken, however, towards wetting the clothes by day. This may be in part involuntary, but is more often largely due to laziness and inattention on the part of the child. If this is the case, it should be punished severely, care being taken that the child has had a reasonable chance to empty its bladder.

The first thing to do in the treatment of enuresis is to search for and remove all causes of irritation of the peripheral nerves. The next thing is to remove adenoids which, if they are large, may interfere with respiration, cause partial suffocation and deaden the cerebral control. Deep sleep cannot be entirely prevented, but it can be controlled to a certain extent by guarding against fatigue. The infantile reflex condition can be to a certain extent overcome and the cerebral control developed by exercises to improve the control of the bladder. The child should be made to pass urine every fifteen minutes for an hour each day and at another time during the day to hold the urine as long as possible. This procedure develops both the muscular coat of the bladder and the sphincters. Finally, measures should be taken to correct the increased irritability of the spinal center, if there is any. It is impossible, of course, to correct heredity, but its influence can be diminished by regulation of the life. The tendency to nervousness can also be diminished by careful regulation of the life to avoid fatigue and excitement. Iron should be given for anemia. Malnutrition can be corrected by careful regulation of the diet and life. It is especially important in this connection to cut out sweets in all forms. In many instances they apparently are the main cause of the enuresis.

In many cases, however, no apparent cause for the enuresis can be found. In such cases the attempt must be made to relieve the condition, as must also be done when an apparent cause for it is found and is being treated. Certain general measures are helpful in all cases. None of them remove the cause and none of them cure the condition, but all together they usually help. No liquids should be given after four or five P.M. The supper should be as dry as possible. Extra water should be given during the early part of the day to make up for the lack of liquid in the latter part. The child should be kept quiet after four or five o'clock in the afternoon, according to its age. It should have no excitement during the late afternoon and evening and should be put to bed early. It should pass urine on going to bed and should be waked up to pass urine early in the evening. It should be waked up to empty the bladder when the parents go to bed and at any time during the night when anyone happens to be awake. It is hardly necessary to put the poles of a battery in the diapers, which will ring an alarm clock when the urine begins to be passed and the poles are connected, as recommended by some German. It is sometimes advisable, however, with older children to set an alarm clock to go off in the early hours of the morning, in order that they may get up and empty the bladder. The child should go to bed with the idea firmly fixed in its mind that it will not wet the bed, and that it will get up and pass urine as soon as it wakes in the morning. Much may be done in this way by suggestion. Furthermore, one of the times when the bed is most often wet is when the child is half-awake in the morning. In this connection, it is often of advantage to praise and reward the child if it does not wet. It is a good plan to have a calendar and to put a gold star on each day when the bed is not wet. The mattress should be hard. It is important to have the bed clothes just right. If they are too heavy, the

child gets too warm and sleeps too deeply. If they are too light, more urine is secreted and, hence, enuresis is favored. The windows should be well open so that the child has plenty of fresh air. It sometimes helps to raise the foot of the bed six inches or more by placing books or blocks under the legs. The object of this is to take the weight of the urine off of the neck of the bladder, which is especially sensitive. Children are more likely to wet the bed when they sleep on the back. If a child sleeps on its back, it is a good plan, therefore, to prevent it from doing so by tying a towel with a knot in the back around the waist or fastening a large spoon on the back with adhesive plaster.

Drugs are used in the treatment of enuresis for two definite objects, namely, to diminish the irritability of the peripheral nerves and to restore the tone of the spinal centers. The best of the drugs to diminish the irritability of the peripheral nerves is belladonna. Either the tincture of belladonna or atropine may be used. Only one dose should be given daily and this after supper. The beginning dose of the tincture of belladonna is between one and three drops. The dose should be increased one drop daily until symptoms of intoxication develop. These are flushing of the face, dryness of the mouth and dilatation of the pupils. When these symptoms appear, the dose should be dropped down two drops and kept there. If belladonna is used in this way, no harm can be done. If there is no relief after two or three weeks on the full dosage, it is useless to continue. Atropine may be used in the same way, beginning with a dose of one five-hundredth of a grain and increasing it by one five-hundredth of a grain at a time. In my experience, belladonna and atropine seldom do any good.

Nux vomica or strychnia are the best drugs to use to restore the tone of the spinal centers. As a rule, it is wiser to use the tincture of nux vomica than strychnia, as the difference between the medicinal and toxic doses is much greater. In general, a drop of the tincture of nux vomica may be given three times daily for each year of age.

Other methods of treatment have been highly recommended. One of these is irritation of the vesicular sphincter and the posterior urethra by means of a bougie-a-boule, the idea being that the discomfort caused during the next urinations will draw the child's attention to the fact that it is passing urine and aid it in gaining control. Faradic electricity may be used over the bladder or in the urethra. I have never seen it do any good. Several authors have reported good results from Cathelin's epidural injections. I have had no experience with them. With the child in the Sims position a needle is introduced in the middle of a line joining the coccygeal cornua and passed directly upward, keeping close to the bone, in order not to perforate the meninges. From five to twenty-five c.cm. of normal salt solution are injected.

SECTION XVI

DISEASES OF THE NERVOUS SYSTEM

THE DEVELOPMENT OF THE NERVOUS SYSTEM

The brain is relatively very large at birth, the relation to the body weight being at birth from 1:8 to 1:10 and in the adult from 1:40 to 1:42. The total weight of the brain increases between three and a half and four times from birth to maturity. The brain grows very rapidly during the first year, weighing about two and a half times as much at the end as at the beginning. Growth is still rapid during the second year, after which it is slow. The adult weight is reached at fifteen years, sometimes even at seven years. The cerebellum is relatively not as large as the cerebrum at birth and increases twice as much in size. There is relatively less gray matter at birth than later. The adult relations are reached at two or three years. The general arrangement of the convolutions and sulci is essentially the same at birth as later, but the sulci are not quite as distinct and continuous before six months. The brain fills the cranial cavity more completely during the first six years than later. The dura is adherent to the skull during infancy. The cranial sutures, while loose at birth, are fairly firmly united at nine months.

The growth of the spinal cord is even more rapid during the first two or three years than that of the brain. The lower end of the cord at birth is at the lower border of the body of the second or at the upper border of the third lumbar vertebra, that is, about one vertebra lower than in the adult. There are no definite data as to when the adult relation is attained.

The cerebral centers are more or less incompletely developed at birth. Those essential for organic life are the best developed and their progress to complete development is more rapid than that of the others. Very little myelinization of the nerve tracts and centers in the cerebral hemispheres is present at birth, although many tracts in the cerebellum and brain stem are partially myelinized and myelinization of the tracts of the spinal cord, except of the pyramidal tracts, is almost complete at birth. Myelinization of the peripheral nerves has hardly begun at birth. Myelinization proceeds very rapidly after birth, most of the sense areas having been covered at three months, and the association fibres at one year. The myelinization of the brain is not entirely completed, however, for many years. Myelinization of the pyramidal tracts and peripheral nerves is almost complete at one year.

Special Senses.—The senses of touch, taste and smell are said to be fully developed at birth. That of touch evidently is, but young infants show very few signs that they taste or smell. They pay no attention to odors and swallow anything that is wet. The eye is anatomically developed at birth. Vision, however, is probably very feeble. A strong light evidently causes discomfort during the first few weeks. The baby does not usually fix its eyes until it is at least six weeks old, and coördination is not well developed until three months or later. Infants hear little

or nothing during the first few days of life, probably because of the swelling of the mucous membrane of the tympanum, and the absence of air in the middle ear. The hearing rapidly improves, however, and in a short time becomes very acute.

EXAMINATION OF THE NERVOUS SYSTEM

On account of these peculiarities of the nervous system at birth and its rapid growth and development during infancy, the findings on physical examination are not only different from those in later childhood and adult life, but vary materially from birth to early childhood. In a general way: the reaction to electrical stimuli is very feeble at birth and for at least two months afterward; the tendon reflexes are feeble at birth, but are exaggerated during most of infancy; the reflexes necessary to organic life are quite active; the special senses are either imperfectly developed or the brain is incompetent to appreciate their messages; inhibition is feeble; mentality, essentially absent at birth, develops very rapidly.

Spasm and Paralysis.—It must not be forgotten that in early infancy, and in emaciated infants, there is a normal hypertonicity of the muscles, most marked in the flexors, which prevents the complete extension of the extremities. It is important not to mistake this normal hypertonicity for permanent contractures. If there is spasm, it is necessary to notice the position of the extremities. It is not safe to assume that there is no spasm because when the child is quiet the extremities are in the normal position. Passive motions of the extremities should always be made to determine whether there is or is not any spasm. If resistance is encountered, it must be determined whether it is due to voluntary opposition or involuntary spasm. It is evident, therefore, that the child's attention must be distracted. It is also important to determine whether the spasm is due to pain or not. It is worth while to remember that the spasm in cerebral paralysis in early infancy is often first shown by opposition to abduction of the thighs.

If the child is unconscious, the presence or absence of paralysis must be determined by the way in which the extremities drop when they are let fall and by the amount of resistance which is encountered on passive motion. When there is paralysis, the extremity drops like a log, whereas, if there is no paralysis, it does not drop in the same way, even when the child is unconscious. If conscious, older children will attempt to make the various motions as directed. Infants, however, will not do this. The power in the arms must be tested by offering them some plaything or showing them their bottle; that in the legs by tickling their feet or pricking them with a pin. It is most important to distinguish failure to use the extremities because of the pain which motion causes from real paralysis.

Reflexes. *Eye Reflexes.*—The reflex closure of the eyelids when some object is brought near the eye is very marked at birth and remains excessive for a month or more. Winking does not develop for about two months. The reaction of the pupil to light is present and active at birth. The reaction of the pupil to accommodation cannot be elicited until the baby is old enough to fix its eyes on an object. It is never present before six weeks and seldom definite enough to be of value before three months.

Abdominal Reflexes.—The abdominal reflexes are very inconstant in infancy and early childhood. They are likely to be feeble during the first month or two and then very lively. They cannot be elicited if the baby is crying or laughing, or if the abdominal wall is held rigidly from pain, fright, or because of cold hands.

Cremasteric Reflex.—This reflex is lively in infancy but much less so during childhood. Neither the abdominal nor the cremasteric reflexes are of much importance, however, unless they are different on the two sides.

Knee-jerks.—The knee-jerk is much more easily determined with the hammer than with the finger. The edge of a hand mirror or of a hair brush makes a very satisfactory substitute for a hammer. The response to tapping the ligamentum patellæ normally varies widely in infancy. It is often very hard to elicit the knee-jerk in infancy because of the baby's failure to relax. It cannot be determined, however, unless the leg is relaxed and on this account great patience is often required. The best method of eliciting the knee-jerk in infancy is to place the hand under the lower part of the thigh, when the baby lies on its back, lifting it a little from the bed. A response can sometimes be obtained, if the angle of the leg on the thigh is varied, the ligament being tapped repeatedly as the knee is moved up and down. The knee-jerk is best elicited in childhood

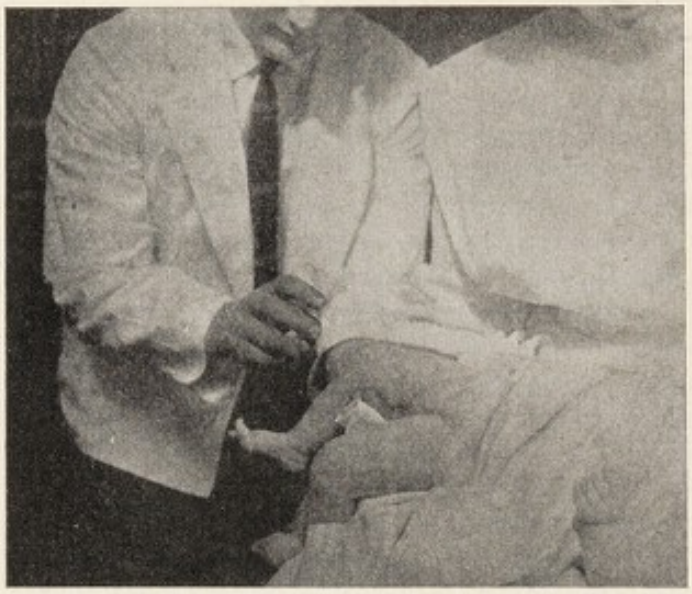

FIG. 149.—Method of eliciting the knee-jerk.

by having the child sit up with the leg hanging down, as in adult life. Reinforcement is of little use, except in older children.

Plantar Reflex.—The plantar reflex is tested by drawing the finger or the handle of the percussion hammer along the inner side of the sole of the foot, beginning at the heel. After infancy the normal reaction is flexion of all the toes. During infancy the response is just as often by extension as by flexion of the toes. Not infrequently in infancy, moreover, the response is like that in *Babinski's phenomenon*, which is the simultaneous extension of the big toe with flexion of the other toes. This phenomenon is of no importance, therefore, in infancy. In childhood, however, it is abnormal and shows some irritation or affection of the pyramidal tract.

Oppenheim's Reflex is the same as the Babinski phenomenon and is obtained by pressure downward along the inner border of the tibia. This is most easily obtained by grasping the edges of the tibia between the thumb and fingers and running down the length of the bone. It has no more value in infancy than the Babinski phenomenon. In childhood it is rather more reliable, because the child does not draw away as the result of being tickled and does not appreciate at all what is being done.

Peroneal Reflex.—The peroneal reflex is tested by striking with the percussion hammer just below and behind the head of the fibula. Normally, there is no reaction. In spasmophilia, however, there is abduction of the foot with a slight elevation of its outer margin.

Ankle and Patellar Clonus.—The significance of the ankle and patellar clonus is much less in infancy and early childhood than later. The ankle clonus is often present in infants ill with any disease and may be elicited in hysterical and neurasthenic children as late as middle childhood without having any pathologic significance.

Trousseau's Symptom.—Pressure on the nerve trunks of an extremity normally causes no reaction. In spasmophilia, however, pressure on the nerve trunks of an extremity not only brings on the typical spasm of tetany in that extremity, but also in the others. This reaction is known as Trousseau's symptom. The pressure is usually made on the large nerve trunks in the upper arm by grasping it firmly just below the axilla.

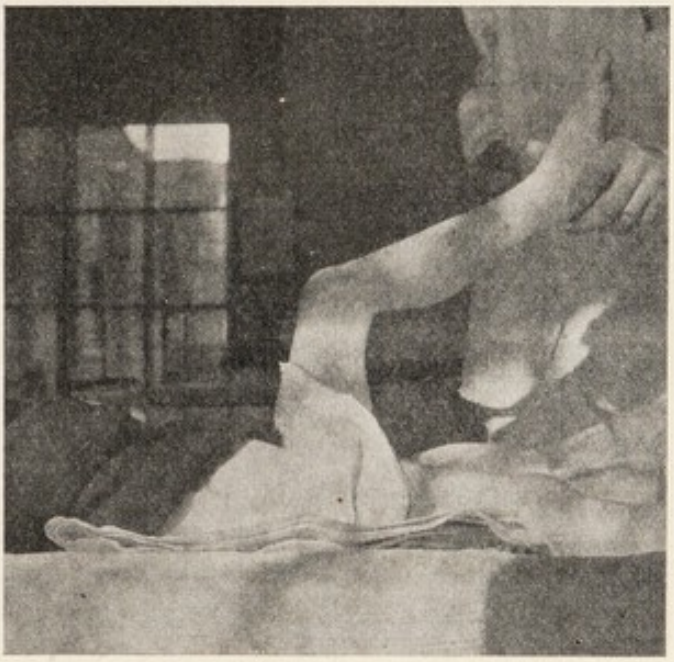

FIG. 150.—Kernig's sign.

It is often necessary to hold it for about two minutes to bring out the reaction. The pressure must be hard enough to cause discomfort and usually causes some blueness of the extremity.

Chvostek's Symptom, or the Facial Phenomenon.—Under normal conditions mechanical irritation of the facial nerve causes no contraction. In spasmophilia, however, irritation of the facial nerve, either by striking it or rubbing something quickly across it, causes contraction of the facial muscles on that side. The reaction is usually especially marked about the mouth.

Kernig's Sign.—Under normal conditions the leg can be extended on the thigh to an angle of 135 degrees or more, when the thigh is at a right angle with the trunk. Kernig's sign consists in the inability to extend the leg on the thigh, when it is at a right angle to the trunk, to as much as 135 degrees. This sign is best tested with the child lying on its back. It makes no difference whether the thigh is flexed to a right angle on the trunk and the attempt then made to extend the leg or the leg extended on the thigh with the thigh extended and the attempt then made to bring the

thigh to a right angle with the trunk. Kernig's sign is an involuntary manifestation and may or may not be accompanied by pain. The phys-

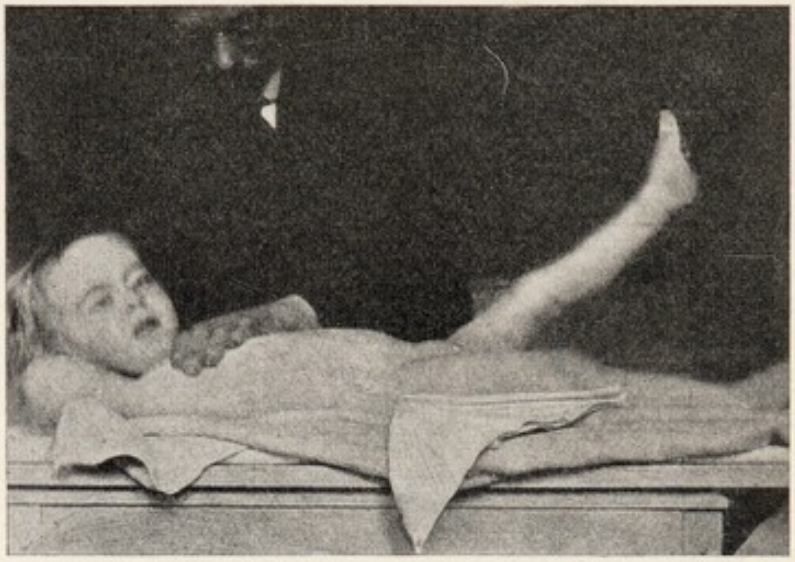

FIG. 151.—Brudzinski's neck sign.

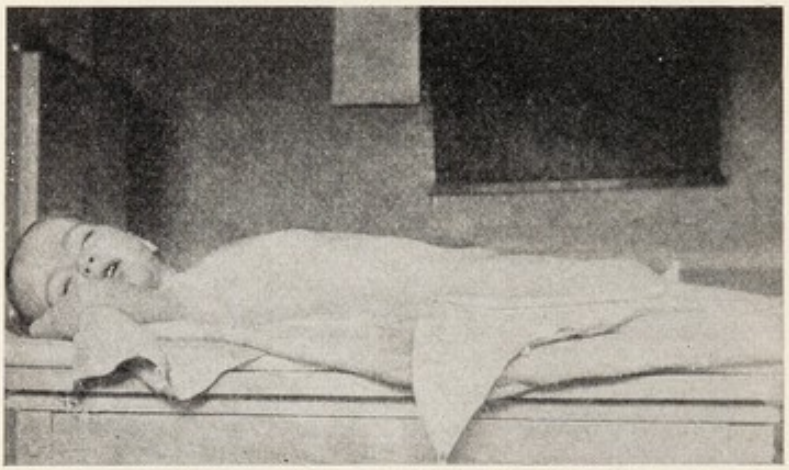

FIG. 152.—Before and after eliciting Brudzinski's neck sign.

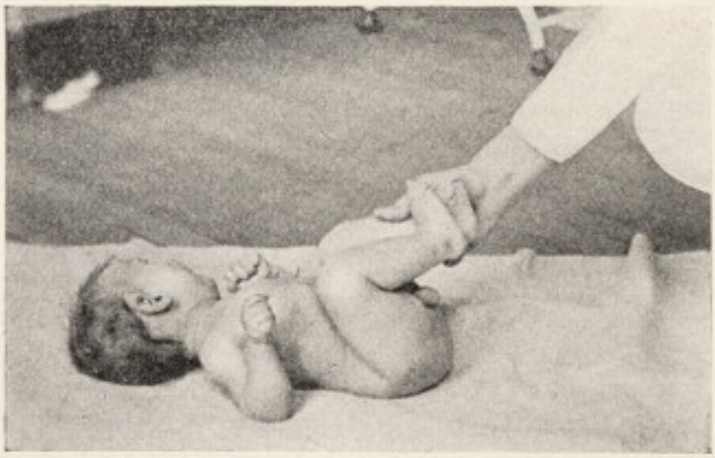

FIG. 153.—Identical contralateral reflex.

iologic hypertonicity of young and emaciated infants may be mistaken for Kernig's sign, if it is not borne in mind. Kernig's sign is strong, but not positive, proof of meningitis in infancy and early childhood.

Brudzinski's Neck Sign.—Under normal conditions flexion of the head forward, when the child is lying on its back, causes no motion of the extremities. Under certain conditions, however, passive flexion of the neck forward, while the child is lying flat on its back, the chest being held stationary, causes flexion of the legs at the hips and knees, but sometimes only at the hips. This sign is known as Brudzinski's neck sign. It is sometimes present on one side and not on the other. It is present in many cases of meningitis and is seldom, if ever, found in any other condition.

Contralateral Reflex.—Under normal conditions passive flexion of one leg causes no motion on the other side. In meningitis, and sometimes in other conditions, passive flexion of one leg causes a concomitant reflex of the leg on the other side—the identical contralateral reflex. Sometimes, however, the motion of the leg is extension instead of flexion—the reciprocal contralateral reflex.

Sensation.—Sensation to touch and pain can be made out even in infancy, although it is not very active during the first few months. Sensation to differences in temperature cannot, of course, be determined until the child is old enough to know hot from cold and to talk. Sensation is in general the same thoroughout childhood as in adult life.

Vasomotor Disturbances.—Superficial vasomotor disturbances of the skin are not at all uncommon in infancy and childhood in many pathologic conditions. Tâches cérébrales, which are obtained by drawing the finger or a pointed object over the skin are, therefore, of no importance in the diagnosis of cerebral diseases.

Electrical Reactions.—The excitability of both motor and sensory nerves and of the muscles, whether to the faradic or the galvanic current, is very feeble during the first two months and the muscular response is sluggish. The adult response is gradually attained, often after a period of hyperexcitability. After the first few months, there is under normal conditions, in early life as in adult life, a quick response to stimulation of both nerve and muscle by both the faradic and galvanic currents. The kathodal closure contraction occurs with weaker currents than the anodal closure contraction. In the *reaction of degeneration*, when it is partial, there is a diminished response to irritation of both nerve and muscle with the faradic current and to irritation of the nerve with the galvanic current, while the response to stimulation of the muscle with the galvanic current is somewhat increased. When the reaction of degeneration is complete, there is no response to stimulation of either nerve or muscle by the faradic current or of the nerve by the galvanic current. The reaction to stimulation of the muscle by the galvanic current is diminished and the contractions are sluggish, and require a stronger current. Furthermore, the anodal closure contraction occurs with a weaker current than the kathodal closure contraction. It must not be forgotten that when atrophy of the muscle has taken place, there is no response to any form of electricity.

It is very difficult to determine the electrical reactions in infants and young children. A reliable examination can only be made by a man specially trained in their use.

Erb's Phenomenon.—This term is applied to a peculiar quantitative reaction of the nerves to the galvanic current, which is characteristic of spasmophilia. The typical law of contraction for this condition has been developed by Thiemich and Mann. For practical purposes, however, the important points to remember are that the appearance of kathodal

opening contractions under 5 ma. is pathognomonic of spasmophilia and that the appearance of anodal opening contractions with less current than that causing anodal closing contractions is strong evidence in favor of it. It is not at all difficult to determine the current sufficient to cause the kathodal opening contraction.

Special Senses.—In infancy the only senses which can be tested are those of touch, sight and hearing. In childhood all the senses can be tested as in adults, but the responses to fine differences are usually less accurate. In young babies sight is tested by moving a bright light back and forth in front of the eyes and noticing whether the eyes follow it. In older babies and young children a toy or the bottle can be used in the same way. A less reliable test is to approach the hand or some object quickly towards the eyes, noticing whether the child winks or not. This test is liable to error because of the current of air produced by the movement of the hand. Hearing is tested in babies and young children by speaking quickly in a loud voice or making a sudden noise near the ears, and noticing whether the child jumps or turns its head.

Lumbar Puncture.—Lumbar puncture should always be performed in infancy and childhood with the child lying on its side on a hard, flat surface. It is very difficult to do it properly when the child is on anything which gives, like a bed, and allows lateral flexion of the spine. The table on which it is placed should not be so wide that the person who holds the baby cannot reach it easily. It is not necessary to anesthetize a baby, but it is wiser to anesthetize a child, unless it is unconscious. It is extremely important to have the lumbar spine flexed so that the spinous processes are separated. It does no good to bend the head forward on the chest and flex the thighs on the body. The spine must be flexed. The skin at the point of puncture should be sterilized with tincture of iodin or with soap and water, followed by alcohol. A line drawn through the crests of the ilia passes through the fourth lumbar spine at all ages. The puncture should be made in the space below or the space above this line. There is but little chance of wounding the cord, if the puncture is made in the second lumbar space, except in babies, as the cord usually does not reach as low as this. It is needless to say that the needle and the hands of the operator should be sterile. Either a needle or a trocar may be used. Many prefer the Quincke needle, which is a needle with a short bevel and a bevelled stylet to fit it. Personally, I prefer an ordinary needle. The needle is best introduced in the median line, being pushed directly forward and a little upward. It may, however, be introduced from a quarter to one half an inch to one side of the median line, being pushed forward, upward and inward. It is easier to go in in the median line. In babies it is about one and one quarter inches into the spinal canal. The distance, of course, increases with age. The mistake in older children is usually not to push the needle in far enough. There is a sudden cessation of resistance when the needle passes into the canal. It is important not to go in too far, because of the danger of wounding some vessel on the anterior wall of the canal and causing bleeding. The admixture of blood makes it impossible to tell whether the fluid is turbid or not and interferes to some extent with the microscopic examination of the fluid. There is no danger of a serious hemorrhage into the spinal canal, even if a vessel is wounded, because the bleeding stops as soon as the needle is withdrawn. A needle about the size of the ordinary antitoxin needle is about right. It is wise to use as large a needle as possible.

If no fluid runs out of the needle or cannula, it is probable that it is not in the canal. It may be plugged with fibrin or, what is more likely, obstructed by one of the nerves of the cauda equina. In rare instances, when there is obstruction high up in the canal, there may be so little fluid in the canal that it does not run out. A stylet should be passed through the needle or cannula to make sure that it is clear, or it should be twisted about a little or pushed in or pulled out a little. If there is still no fluid, it should be withdrawn and introduced in another space.

Under normal conditions the cerebrospinal fluid runs out slowly, drop by drop. When there is an increase in intracranial pressure, it spurts. It may run slowly, if it is purulent and thick. It is safe to let the fluid run out until the fontanelle, if it is open, becomes a little depressed or until the drops begin to come quite slowly, unless in the meantime the patient shows signs of irritation of the nervous system. The intraspinal pressure is normally under 8 mm. of mercury. It is impossible to estimate the intraspinal pressure, however, without considerable practice and the proper apparatus. Fortunately, it is very seldom necessary in ordinary everyday work. After the needle is withdrawn a sterile sponge should be applied with a plaster strap or a piece of cotton fastened on with collodion.

Two sterile test tubes and corks, or better a sterile test tube and a sterile centrifuge tube, in which to collect the fluid, should be ready at the time of the puncture. In tuberculous meningitis the organisms are more likely to be in the latter part of the fluid than in the first. It is also advisable to have a white blood counter at hand, because it is not possible to get an accurate count, if the fluid is taken from a test tube later to be counted.

The normal cerebrospinal fluid is clear, like distilled water, usually contains very few, and never more than ten, cells to the cubic mm., most of which are small mononuclear. It contains no organisms, does not deposit a fibrin clot on standing, gives no reaction to the tests for globulin and does show a trace of glucose with Benedict's or Fehling's solutions.

There is no sediment in the normal cerebrospinal fluid. In tuberculous meningitis there is a white fibrinous cloud suspended from the center. In meningococcus meningitis it is a balloon-like mucoid mass, while in pneumococcus meningitis it is at the bottom of the test tube.

The number of cells should be determined with the ordinary white blood counter.

There are many tests for globulin. Those most commonly used are Noguchi's and Nonne's. Noguchi's test is more accurate than Nonne's. Noguchi's test is performed by adding four parts of a 10% butyric acid in normal salt solution to one part of spinal fluid. The mixture is boiled. As much normal sodic hydrate solution as there was spinal fluid is added and the mixture is boiled again. Normal fluids may give a faint opalescence. Pathologic fluids are likely to give a floculent precipitate, indicating an excess of globulin. In Nonne's test an equal amount of a saturated solution of magnesium sulphate is added to the spinal fluid in a test tube and the mixture compared with a tube containing spinal fluid alone. A white precipitate indicates an excess of globulin. A simpler and, for everyday purposes, thoroughly reliable test is that in which a saturated solution of ammonium sulphate is allowed to run under the cerebrospinal fluid in the same way as the nitric acid in performing the

nitric acid test for albumin in the urine. If there is globulin in the fluid, there is a white zone at the line of contact.

In the normal cerebrospinal fluid there is enough glucose to reduce both Benedict's and Fehling's solutions. The glucose is more often diminished or absent in meningococcus meningitis than in tuberculous meningitis. The variations are so inconstant, however, that the test for glucose is of no practical value.

If there is a definite fibrin cloud, or a definite sediment of any sort, this should be stained and a differential count of the white cells made and bacteria looked for. If there is no cloud or sediment, and an immediate diagnosis is desired, the fluid should be centrifugalized and the sediment examined in the same way.

Puncture of the Lateral Ventricles.—Puncture of the lateral ventricles is not difficult when the anterior fontanelle is open. The needle should be introduced at the lateral angle of the fontanelle, passed downward, slightly forward and inward. It is needless to say that the operation must be performed under aseptic conditions. Trephining is necessary, if the fontanelle is closed.

Cistern Puncture.—Cistern puncture is a difficult operation and should not be attempted except by an expert.

PECULIARITIES OF DISEASE OF THE NERVOUS SYSTEM IN EARLY LIFE

It is well known that in infancy and early childhood the manifestations of disease of the nervous system are more violent than in late childhood and adult life and that apparently slight causes may produce marked and even alarming symptoms. This is especially true of the responses to reflex irritation. It is easy to understand these peculiarities when the anatomical conditions are borne in mind. The brain is relatively very large and grows very rapidly, as does also the spinal cord. A rapidly growing organ is notably unstable and, furthermore, is easily exhausted. The large size of the brain in relation to that of the cranial cavity makes an increase in intracranial pressure easier and more serious. The imperfect development of the cerebral centers, especially of those of inhibition, and of the conducting fibres exaggerates and modifies the responses to all sorts of stimuli. The imperfect development of the spinal centers, together with the lack of cerebral control of these centers, predisposes to eccentric and excessive reflex activity. Disturbances of nutrition, so common in infancy and early childhood, affect especially rapidly growing organs, as are notably the brain and cord at this period Poorly nourished nerve tissue at all ages, moreover, shows its poor nutrition especially by instability and exaggerated response to all sorts of stimuli. Furthermore, minor injuries to the nervous system, especially to the brain, which in the older child and adult produce but little permanent disability, may in the infant and young child, by interfering with or preventing normal development, cause very serious damage and be permanently disabling.

General and Functional Nervous Diseases

CONVULSIONS

Convulsions are most common during the first two years of life, although, except in the first one or two weeks, they are less common in the first six months than during the rest of infancy. They become progressively less common up to seven years, after which they are unusual, except as a manifestation of epilepsy. It must never be forgotten that a

convulsion is merely a symptom, not a disease. This is true, even if there is a series of convulsions.

Etiology.—*Predisposing Causes.*—The most important predisposing cause of convulsions is infancy. The reasons why infancy is such an important cause have already been explained. Another important predisposing cause is heredity. There is no doubt that the children of neurotic parents and belonging to neurotic families are more prone to have convulsions than those coming from better stock. All the diseases and conditions that affect the general nutrition likewise predispose to convulsions, because the brain shares in the general malnutrition and, as is well known, poorly nourished nerve tissue is unduly irritable. Among these conditions are rickets, disturbances of digestion, anemia and debility from any cause. Perhaps the most important of all of this class is spasmophilia, in which there is a diminution in the calcium of the blood and hence an increase in the excitability of the cerebral centers.

Exciting Causes.—These may be divided into three classes: (1) those which cause direct irritation of the cerebral cortex; (2) those that act through reflex irritation; (3) those which produce toxic substances which affect the cerebral centers.

The first class, which cause direct irritation of the cerebral cortex, includes all forms of cerebral disease, such as meningitis, tumors, abscesses, embolism and thrombosis, hemorrhage and its results, and all sorts of cerebral defects. The convulsions due to this class of causes are often spoken of as organic, and those due to the other classes as functional. There is nothing about the convulsions themselves to distinguish the organic from the functional. In fact, it is very probable that many of the so-called functional really have an organic basis. With the organic convulsions, however, are usually associated other evidences of the cerebral lesion which causes them.

The importance of reflex irritation in the etiology of convulsions in early life has, it seems to me, been much exaggerated. I do not go so far as to say that eye-strain and phimosis cannot cause convulsions, but I have never seen any cases in which I thought they were the cause, while I have had many children brought to me for convulsions who were wearing glasses or who had been circumcised to stop them. I have seen various manifestations, such as vomiting, looseness of the bowels, fever and irritability, which I have supposed to be due to reflex irritation from dentition, but I have never seen convulsions which were not better accounted for in some other way. It is possible, nevertheless, that the excitability of the nervous system may be increased by irritation from the teeth and the development of convulsions from some other cause thus made easier. In no one of the children that I have seen in whom the convulsions were attributed to worms have I ever found either worms or their eggs, and no patient whom I have known to have worms has had convulsions which could fairly be attributed to them. I have had one case, however, in which I made a diagnosis of epilepsy after a most careful study, in whom the convulsions ceased after he passed a number of round worms. I have also had another case in which convulsions, which had persisted for some years, ceased after stretching of the sphincter ani by an irregular practitioner. There is no doubt that babies and children often have convulsions after eating indigestible and improper food and that the convulsions cease after this food has been removed from the digestive tract. It seems reasonable to believe that the food is the cause of the convulsions in these instances. It is more difficult to know whether the

food causes the convulsions by acting simply as a foreign body and in this way producing reflex irritation or whether it sets up abnormal decomposition or fermentation in the digestive tract, the products of which, being absorbed, bring on the convulsions through toxic irritation of the cerebral centers. The immediate cessation of the convulsions after emptying of the digestive tract, before sufficient time has elapsed for the elimination of toxic substances from the system, suggests strongly that reflex irritation is more important in these cases than toxic absorption.

The convulsions that occur at the onset or in the course of acute diseases are presumably due to the action of toxic substances in the circulation on the cerebral centers. They may, however, be due in part to high temperature. The convulsions which occur not infrequently in whooping-cough, however, are not all due to the toxemia of the disease. They may also be due to asphyxia, cerebral edema or hemorrhage. Convulsions in the new-born infants of uremic mothers may be due to the transmission of the uremic poison from the mother through the placenta. Convulsions may occur in connection with certain severe types of the status lymphaticus. In these cases, however, the babies are always seriously ill and the convulsions are only one of the manifestations of the condition. I have never seen convulsions which I thought were due to pressure from the thymus and I have never seen single convulsions, occurring at long intervals, which I thought could be attributed to toxemia from the thymus. I have likewise never seen convulsions in infancy or childhood from lead poisoning, although this is always given as one of the causes of convulsions in childhood. It should, nevertheless, be kept in mind as a possible cause.

Pathology.—There is, of course, no permanent pathology for functional convulsions. At the onset of a convulsion there is said to be a cerebral anemia, which is quickly followed by venous hyperemia. When convulsions are organic, the causative lesion is, of course, permanent.

Symptomatology.—It is hardly necessary to describe the symptomatology of a typical convulsion. It would be very hard for even a layman to mistake a convulsion for anything else or anything else for a convulsion. In many instances, however, the manifestations of a convulsion differ materially from those of a typical convulsion. Even in cases where there is no question of epilepsy, a convulsion may be represented simply by an instantaneous loss of consciousness or a very slight stiffening or twitching, or, in a baby, by rolling up the eyes. It is these slight manifestations, I suppose, that the old ladies describe by the name of inward convulsions. It is often difficult, and always unimportant, to distinguish between a tonic and a chlonic stage in the convulsions of early life, or even between tonic and chlonic convulsions. The diagnostic importance of unilateral and localized convulsions is much less in early life than later. The younger the patient, the less important they are. It is also a waste of time to note whether a convulsion begins on one side or on the other, or whether one part is more affected than the others. The reason is that the nervous system of the infant and young child is so irritable that any and all sorts of responses may result from the same stimulus, and many different stimuli produce the same response. There is nothing about the convulsions in spasmophilia to distinguish them from other convulsions. In general, in early life, a convulsion is simply a convulsion and there is nothing about the severity, the distribution, the onset or the character of the convulsion which shows anything as to its cause.

Diagnosis.—When a child has a single convulsion, the only important thing in the diagnosis is to determine its etiology. When a child has had convulsions in the past, unless there are evidences of organic disease of the nervous system, the question always arises as to whether the convulsion is a manifestation of epilepsy. In infancy, epilepsy is the least probable diagnosis. If evidences of spasmophilia are found, epilepsy may be excluded. The character of the convulsions is of no aid in the diagnosis. The repetition of convulsions without apparent cause is in favor of epilepsy. If there has always been an apparent cause for each convulsion, this fact counts against epilepsy. In childhood the character of the convulsion is of more importance. Points in favor of epilepsy are the presence of an aura or of an initial cry, the occurrence of the convulsions at night, and stupor or a desire to sleep after a convulsion. Epilepsy cannot be excluded, however, because there is no aura or initial cry. The presence of an apparent cause, such as fever or indigestion, for each attack is against epilepsy. The repetition of convulsions over a considerable time without any apparent cause is strongly in favor of epilepsy. In general, it is always wise to be very careful about making a positive diagnosis of epilepsy in childhood, because the chances are against this being the right diagnosis.

Prognosis.—Death rarely occurs in or as the result of a single convulsion. This is especially true of the convulsions of spasmophilia. The younger and more feeble the individual, however, the greater is the danger. A single convulsion is most dangerous when it occurs in whooping-cough or in the course of diseases of the larynx or lungs. Moreover, the cerebral congestion may be so great in any convulsion that it may result in a hemorrhage, which will later cause feeblemindedness, epilepsy or spastic paralysis. Death is unusual, even when there is a series of convulsions in rapid succession and lasting many hours. It may, however, occur as the result of exhaustion, even in strong babies or children. Repeated convulsions, reflex in origin, may apparently in time develop a "bad habit of the brain" and lead to epilepsy.

Treatment.—At the onset of a convulsion there is cerebral anemia which is quickly followed by venous hyperemia. This has always developed by the time the physician reaches the patient. The immediate indication is, therefore, to relieve cerebral hyperemia. This may, perhaps, be accomplished by the application of heat or counter irritants to the surface of the body, heat to the feet and cold to the head. At any rate, it affords a rational explanation for the popularity of these household measures. They certainly can do no harm, even if they do no good. It must be remembered, however, that the temperature of the bath should not be over 100° F. It must also be remembered that, if the convulsion is due to the action of a high temperature, a hot bath will do harm and that it is a cold bath to bring down the temperature which is needed. It is also advisable to undress the patient before putting it in the bath, because, if it is undressed slowly, it will usually be out of the convulsion before it is ready for the bath and then the bath will be unnecessary. If desired, a heaping tablespoonful of mustard may be added to six gallons of water. It is not advisable to keep the patient in the mustard bath more than ten minutes, however, for fear of overstimulating the skin. Personally, I have never been able to make up my mind that a mustard bath is any better than a plain water bath. When the child is taken out of the bath, it should be immediately wrapped in a warm blanket and kept there for a time. It is often a good plan to put

an ice-bag on the head. Ether and chloroform, however, are far more effective for the immediate relief of a convulsion than either the warm bath or the mustard bath. A few drops of chloroform usually stop a convulsion at once. Ether is, however, safer in the hands of the laity.

As soon as the emergency allows, the attempt should be made to determine the cause of the convulsion, in order to remove it, if possible. The temperature should be taken at once and in the rectum. A careful history should be taken, special attention being paid as to whether there have been previous convulsions, recent exposure to contagious diseases, indiscretions in diet or manifestations of spasmophilia. A careful and complete physical examination should then be made, including the tests for the increased mechanical excitability of spasmophilia. The urine should be examined, if possible, but at this age uremia is one of the rarest causes of convulsions. If it is the cause, the physical signs of disease of the kidneys—edema, ascites and pallor—are almost always marked, making the diagnosis easy. If no definite cause for the convulsion is found, it is almost always safe and advisable to wash out the lower bowel and to give a cathartic. It is usually not advisable to give an emetic or to wash out the stomach, as these procedures are liable to start up another convulsion, in which food may be inhaled and set up bronchopneumonia.

When the patient does not relax after a convulsion or the convulsions are repeated, it is advisable to give bromides or chloral by the mouth or rectum. My feeling is that the doses of the bromides usually recommended are rather small and those of chloral too large. Bromides are a safe drug and the limit between the physiologic and toxic doses is a wide one. Chloral is a rather dangerous drug and the limit between the physiologic and toxic doses is a narrow one. Chloral is, however, undoubtedly more efficient than the bromides. Five grains of bromide and one grain of chloral by the mouth, or one and one-half times these doses by the rectum, are sufficient, at least as a beginning, for a baby of six months. Ten grains of bromide and two grains of chloral may be given by the mouth to a baby of two years, or fifteen grains of bromide and three grains of chloral by the rectum. If these doses are insufficient, larger doses may then be given. If the convulsions do not yield to bromide and chloral in safe doses, morphine should be given hypodermically. The tolerance of babies and young children for morphine is, however, relatively low and it must, therefore, be used cautiously. It is always wise to begin with a small dose, giving more later, if the small dose is insufficient. A suitable dose for a baby of six months is $\frac{1}{128}$ of a grain, for a baby of two years, $\frac{1}{64}$ of a grain and for a child of five years, $\frac{1}{32}$ of a grain. Oxygen is sometimes useful when the convulsive state is prolonged and cyanosis continuous.

The curative treatment of convulsions due to spasmophilia is taken up under that disease. When children have convulsions from time to time and the diagnosis of epilepsy is not warranted, the general health of the child should be carefully looked after, its diet regulated and its bowels kept open. It should be made to drink plenty of water and all causes of reflex irritation should be sought for and removed.

IDIOPATHIC EPILEPSY

This term is applied to a condition in which there are repeated attacks of loss of consciousness, usually associated with convulsions of greater or less severity, for which there is no apparent cause. When these attacks

are associated with other evidences of an organic lesion of the nervous system, the condition is called symptomatic epilepsy. When there is an apparent cause for the convulsive attacks outside of the nervous system, they are spoken of as reflex convulsions.

Etiology.—Epilepsy is more common in the children of epileptics and alcoholics and in members of neurotic families than in the children of the community at large. This fact is of no importance to the child who has it, however, and of little importance to anyone, while procreation is as unrestricted and alcohol as easily obtainable as at the present time. It is very doubtful if syphilis causes idiopathic epilepsy, although it may symptomatic epilepsy. It is probable that in most instances epilepsy which apparently is the result of convulsions in infancy was epilepsy from the first. I am quite confident, however, that epilepsy is occasionally the result of damage done to the brain in attacks of convulsions due to other causes, whether reflex or otherwise. The convulsions of spasmophilia never lead to epilepsy. It is impossible to know when a child has one or several convulsions, not due to spasmophilia, whether they may lead to epilepsy later or not. It is foolish to believe that things like eye-strain, phimosis, masturbation or even autointoxication from the intestinal tract can be the cause of epilepsy, although it is possible that autointoxication may be an exciting cause for individual attacks. It is still more foolish to believe that there is any special etiologic organism, having its habitant either in the colon or elsewhere, for epilepsy.

Pathology.—It is safe to assume that there are abnormal changes in the cells of the cerebral cortex in epilepsy. It is conceivable that these changes may be either functional or organic. It is certain that at present no one knows what they are. It is probable that until new methods of approach are discovered, no one will know.

Symptomatology.—The manifestations of epilepsy are essentially the same in childhood as in later life. There are, however, some minor differences. An aura or, at any rate, one which children are able to describe, is unusual. An initial cry is also uncommon. Children are also somewhat less likely to want to sleep after an attack than adults. There is, therefore, nothing about many or most epileptic convulsions in early life to distinguish them from convulsions due to other causes. As in adults, however, epileptic convulsions are likely to occur at night, especially in the early morning hours. Intermittent bed wetting should always suggest epilepsy as a possible cause. Minor convulsive manifestations, such as slight local or general twitching and temporary spasms of various sorts, are very common in infancy and of exactly the same significance as are convulsions. Care must be taken, however, not to confuse the characteristic carpopedal spasms of spasmophilia for epileptic attacks. The characteristic single, purposeful and controllable movements of habit spasms are also materially different from the minor convulsive manifestations of epilepsy.

Minor seizures, petit mal, are more common in early life than later and are very varied in character. Some of the most common manifestations are a sudden look of blankness, an instantaneous loss of consciousness, the dropping of some utensil, a fleeting stop in the performance of some action, in speech or attention, and the involuntary passage of a few drops of urine. In most instances, although there is a temporary loss of consciousness, the child does not fall. In others, however, the child may fall, or, if it does not, stop in the street where it is in great danger. Temporary

lapses or "absences" of any sort are most suggestive, therefore, of epilepsy. These minor seizures may be very frequent, sometimes occurring even as many as one hundred times a day. In rare instances temporary attacks of irritability or anger apparently represent a seizure.

In true idiopathic epilepsy in childhood the mentality is, as a rule, little, and in many instances, not at all affected. If it is, the seizures are usually symptoms of some organic cerebral lesion instead of idiopathic epilepsy. Sometimes, however, mental deterioration develops quite rapidly and becomes very marked, even when there are no evidences of any organic cerebral lesion. When there is mental impairment in idiopathic epilepsy, it is quite as likely to be due to the drugs which are being taken as to the disease itself.

Diagnosis.—The diagnosis of idiopathic epilepsy in well marked cases is not difficult. In general, however, I know of no harder problem in the field of pediatrics than to determine whether, when a child has had one or a few convulsions, they are symptoms of epilepsy or not, or, if not, whether they may lead to epilepsy later. When the convulsion is the first one and there is some apparent cause for it, such as a high temperature, the evidences of some acute disease or the history of an indiscretion in diet, it is hardly necessary to consider epilepsy. If evidences of spasmophilia are found, epilepsy may be excluded. If there have been convulsions in the past, the possibility of epilepsy must be considered, even if there have been apparently good causes for the previous convulsions. Again, epilepsy may be almost certainly excluded, if there are any evidences of spasmophilia. When there is a history of convulsions at intervals without apparently good causes for the attacks, the chances are in favor of epilepsy. The longer the period which the convulsions have covered, the greater is the probability that they are epileptic. Nevertheless, in infancy the chances are that they are not. The presence of an aura or of an initial cry is almost certain proof of epilepsy. Their absence does not, however, count much against epilepsy. The occurrence of convulsions at night is in favor of epilepsy. The history of an injury to the head, followed by convulsions, is somewhat in favor of epilepsy. In general, it is impossible to determine immediately whether a child that has had a number of convulsions at intervals has epilepsy or not.

A most thorough examination should be made in all such cases to find out if there are evidences of an organic cerebral disease or sources of reflex irritation. Such an examination should include a Wassermann test on the blood and spinal fluid, a tuberculin test and a lumbar puncture with an examination of the spinal fluid, as well as examinations of the urine, stools and blood. The stools should be studied not only for evidences of indigestion, but also for evidences of parasites. The fundi of the eyes should be examined for evidences of an increase in the cerebral pressure and every possible location for reflex irritation investigated. When all these things have been done, it will sometimes be found that there is a real organic basis for the convulsions, such as syphilis or a cerebral tumor. In others, some possible sources for reflex irritation may be found. In most instances, however, everything is negative and as little or as much is known after the study as before and time alone renders a diagnosis possible.

The same careful and complete examination should be made in those instances in which there are repeated minor seizures as in those with repeated convulsions. The results are usually equally meagre. In general, these minor seizures are fully as likely to eventually turn out to

be epilepsy as are convulsions, unless the convulsions have an aura or an initial cry.

There ought not to be any difficulty in distinguishing between attacks of faintness or hysteria and epilepsy in infancy and childhood. In the first place, infants are very seldom faint and still less often hysterical, while children are not often faint and seldom hysterical. Careful observation should prevent any mistakes in the few instances in which they are faint or hysterical. Some of the chief differences are that in all epileptic seizures loss of consciousness occurs suddenly and is absolute, while in fainting spells it comes on gradually and in hysteria is not absolute. The pupils are dilated in epileptic attacks, but not in fainting spells or hysterical attacks. The involuntary passage of urine and feces is strong evidence in favor of epilepsy. There are always other evidences of hysteria when the attack is hysterical.

Great care should always be exercised not to make the diagnosis of epilepsy in early life on insufficient grounds. It should not be made unless the manifestations are absolutely characteristic. In infancy the chances are that repeated seizures, suggestive of epilepsy, are not due to that disease. In childhood they may be. It is wiser, therefore, in infancy to say nothing about epilepsy until the diagnosis is certain. In childhood it is wiser to mention it as a possibility, saying, however, that the chances are against it.

Prognosis.—When a child has epileptic seizures of any sort, whether great or small, it is impossible to know what the future may bring forth. In general, recovery is more likely to take place in early life than later. It may occur when there have been many attacks, either of grand mal or petit mal. On the other hand, the attacks may increase in frequency and severity. As time goes on attacks of petit mal are likely to be replaced by attacks of grand mal. In a general way, also, the attacks are likely to become less frequent and more severe. I have known babies and children to have many convulsions, in one instance about six hundred, and then be perfectly well. I have known others, who had had only one or two at the time I saw them, to become confirmed epileptics. I have known children to have hundreds of attacks like petit mal and then be entirely well. It is impossible, therefore, in a given case to make any definite prognosis as to what may happen in the future.

Treatment.—The treatment of an epileptic convulsion is the same as that of any other convulsion. It is more necessary, however, to put something between the teeth in order to prevent biting of the tongue. Children who have convulsions, or who have simply lapses of consciousness, are always in danger of injury from falling or from being run over. The question as to how carefully the child must be watched and its normal activities limited must be decided in each individual case according to the circumstances. In general, however, the child should be allowed to live as nearly a normal life as possible. It is important to look after the general health of the child, to regulate its diet carefully and to be sure that its bowels are kept open and that it drinks plenty of water. As far as possible, all causes of reflex irritation should be removed. It is also wise to cut broths and beef juice out of the diet and to limit the protein intake. It is hardly necessary to cut it down to the minimum protein needs. It is usually advisable to give children the choice of meat, fish or egg, once daily. Red meats are no more harmful than white meats or fish. Milk may be taken freely. If there are evidences of either fermentation or decomposition in the intestine, the diet should be changed to

meet them. In some instances the administration of lactic acid organisms or the bacillus acidophilus may help. Treatment of this sort, however, merely diminishes one of the exciting causes of the attacks, but does not cure the disease. The removal of the colon for epilepsy, is, in my opinion, not only irrational, but unjustifiable. The convulsions can be stopped temporarily, of course, by prolonged starvation. The underlying condition is not cured by starvation, however, and the convulsions sooner or later return. This procedure hardly seems justifiable, therefore, especially in the case of children. I have had no experience with the so-called ketogenic diet, in which one gram of protein is given daily per kilo of body weight and the carbohydrates are limited to between ten and fifteen grams daily, enough fat being given to cover the caloric needs and from 25 to 30% more. This method of treatment, however, seems reasonable.

Great discretion must be exercised in the use of the bromides in epilepsy in childhood, as the by-effects of the drug are often worse than the disease. Large doses given continuously not only disturb the digestion and prevent proper physical development, but seriously interfere with the development of the mind. I have seen a number of children in whom the mental impairment and general malnutrition have been attributed to epilepsy, when they were really due to the large doses of the bromides which they had been taking and disappeared promptly when the bromides were omitted. In general, I believe that it is wiser not to give bromides to children, unless the convulsions are quite frequent and severe. If it is necessary to give the bromides, it makes no difference which salt is used. One is as good as another. When they are used, the dose given must be large enough to diminish or control the attacks and yet must be kept as low as is possible and still accomplish the object aimed at.

Phenobarbital—luminal—apparently does not have the same depressing action on the mental and physical development as do the bromides, while it does have a restraining action on the disease. It has seemed to me, therefore, to be better than the bromides in the treatment of epilepsy. It does not, however, justify all the claims which have been made for it. When it is used, it should be used in the same way as the bromides, that is, enough given to accomplish the object aimed at, but no more than is absolutely necessary to do this. I usually start with one half grain, three times a day, for a baby, three fourths of a grain for a child of four, and one and one half grains for a child of eight or ten years, increasing the dose as necessary.

CHOREA

Chorea, or St. Vitus' dance, is a functional disease of the nervous system, characterized by irregular, involuntary twitchings of some or all of the voluntary muscles of the body. It is most common between the ages of five and fifteen years, much more common in females than in males, occurs most often in the spring, when children are tired from long attendance at school, and is more common in the children of neurotic families.

Etiology.—Chorea is not due to syphilis. The close clinical relationship between acute articular rheumatism, endocarditis and chorea, taken in connection with the present conception that acute articular rheumatism and acute endocarditis are bacterial in origin, suggests that chorea is also bacterial in origin and, perhaps, caused by the same or a similar organism. Further evidence pointing in the same direction is the fre-

quency with which local foci of infection, notably in the tonsils or in and about the teeth, are found in all of these conditions. Nevertheless, bacteria have seldom been found in the brain in fatal cases, these bacteria have not always been the same and there have always been associated with the chorea in these cases rheumatism, acute endocarditis or disease of the tonsils or the teeth. Furthermore, blood cultures in chorea have only occasionally been positive and the organisms found not always the same, although most often streptococci. In the positive cases, moreover, there has always been some other disease or focus of infection in addition to the chorea. The cerebrospinal fluid in chorea is also almost invariably sterile. Rosenow's experiments on animals, however, suggest, but do not prove, that chorea is due to a streptococcus having peculiar neurotrophic properties. The conclusion seems warranted, therefore, that, although there is much clinical and some experimental evidence pointing towards a bacterial origin for chorea, there is no proof of it.

If the bacterial origin of chorea is accepted, it is very difficult to explain the sudden onset of marked chorea within a few hours after a fright or nervous shock. Nevertheless, chorea does come on in this way not very infrequently and in these cases there are no other evidences of infection. It may be argued, of course, that the fright or nervous shock simply opens the doors to infection from some focus which is not recognizable or that there are two varieties of chorea. Neither explanation seems very satisfactory. The real explanation for the discrepancy is not evident at present, but will probably be very simple when the etiology of chorea is finally determined.

Pathology.—Our knowledge of the pathologic changes in chorea is as indefinite and as inconclusive as that regarding the etiology. It is evident that, as complete recovery occurs, they cannot be serious. It is also evident from the symptomatology that they must be located in the gray matter of the central nervous system. It is probable that the entire motor tract may be involved. It is almost certain that the pathologic changes occur more frequently in the cortex than elsewhere in the brain. It is also presumable from the peculiar distribution of the symptoms and the rapid and complete recovery that the lesions are primarily vascular. This presumption counts in favor of a bacterial origin for the disease.

Symptomatology.—In the average case of chorea the onset is gradual. There is at first a little involuntary twitching of the muscles of the upper extremities and face and the disposition changes. The child soon begins to spill its food or to drop things and has difficulty in writing. It often begins to have a little difficulty in talking. The involuntary twitchings of the muscles of the face and arms increase and those of the legs and trunk become involved. All the symptoms increase for two or three weeks, then remain stationary for two or three weeks, after which they gradually diminish again and recovery is complete in from six to ten weeks from the onset. In other instances the onset is very sudden, usually after a fright or nervous shock, and the symptoms are fully developed in a few hours or days. In other instances the symptoms are very mild, so mild sometimes that the diagnosis is in doubt, and the course only three or four weeks. In others the twitchings may be very severe, so that the child throws itself all over the bed, cannot sleep, and becomes much exhausted. In some of these severe cases the child has to be wrapped up and the bed padded to prevent injury. In the worst cases restraint may be necessary.

The muscles of the hands and fingers, face and tongue are those most often and most severely involved. Inability to keep the tongue still and twitching of the muscles of the tongue are among the most characteristic symptoms. The involuntary twitchings are exaggerated by volitional effort. It is usually said, therefore, that children with chorea cannot keep still when they are told to do so. This is true in the severe, but is not true in the mild cases. They can keep still for a short time, if they try. It is not possible, therefore, to rule out chorea because a child can keep still for a short time. The involuntary twitchings of chorea cease during sleep. It is characteristic of the motions of chorea that they are rapid, purposeless and not like ordinary, voluntary, objective motions.

There is usually some muscular weakness associated with chorea. In some instances it is quite marked. It is usually general, but may be much greater on one side than on the other or limited to one side. Sometimes the muscular weakness is more evident than the twitching. I have seen the diagnosis of infantile paralysis made in such cases, as well as those of multiple neuritis, meningitis and cerebral paralysis. It is especially confusing when both twitching and muscular weakness are almost limited to one side. The condition may then be mistaken for "posthemiplegic chorea."

Speech is not infrequently affected. The difficulty is in using the tongue, not in not knowing how to use it or what to say, that is, the condition is one of dysarthria, not of aphasia.

The mental condition is almost always more or less affected, even in mild cases. The children are usually irritable, snappy and cry easily. Sometimes they are stupid. In rare instances they become almost maniacal, the so-called "chorea insaniens."

The deep reflexes are very variable, sometimes being much exaggerated, sometimes normal and sometimes diminished. They are likely to be diminished in the cases in which muscular weakness is marked. Sensation is normal. The electrical reactions are usually normal.

The temperature is usually normal in chorea, unless there are complications. In severe cases, however, when the children are very active, it is almost always somewhat increased. I have seen it as high as 103° F. without any complications. There is usually some anemia, which is more marked in the severe and long cases. The white count is usually normal, unless there are complications, but may be moderately increased. The cerebrospinal fluid is always clear. The pressure may be somewhat increased. The number of cells is usually normal, but may be as high as thirty in uncomplicated cases. The cells are all mononuclear.

Complications.—The only important complication of chorea is endocarditis, although it is often associated with rheumatism and disease of the tonsils, teeth and gums. As always in early life, the endocarditis may be accompanied by myocarditis and pericarditis. Endocarditis is always present in fatal cases. In fact, there is no disease in which endocarditis is so constantly found at autopsy as chorea. Clinically, it is present in from 25% to 50% of the cases. Certainly 25% of children with chorea are left with an organic heart lesion. It must not be forgotten that cardiac murmurs in the course of chorea may be due to other causes than acute endocarditis. The size and rate of the heart, the character of the heart sounds, the location and character of the murmurs, the intensity of the second pulmonic sound, the temperature and the

white blood count must all be taken into consideration before making a positive diagnosis.

Prognosis.—Death seldom occurs in chorea, except from a complicating heart lesion. It may sometimes occur, however, from exhaustion in the very severe cases as the result of the thrashing about, lack of sleep and inability to take food. Recovery is usually complete in three months. If the symptoms continue more than six months, the chances are that there is a mistake in the diagnosis. Recurrences are common, especially in subsequent springs.

Diagnosis.—Chorea is not often mistaken for anything else, except when muscular weakness is an especially prominent symptom and the characteristic twitchings are overlooked. Many other things, however, are mistaken for chorea. Many children are constantly on the move; they never sit still, but kick their legs, play with something or wriggle about. All the motions which these children make are, however, voluntary, purposeful and normal. The child can keep perfectly still when it tries and it makes no involuntary movements when it is doing something definite. In chorea, however, the motions are involuntary, purposeless and abnormal and are increased by voluntary effort. The child with chorea cannot, moreover, keep still but a short time, no matter how hard it tries.

Habit spasms are very often mistaken for chorea. The motions in habit spasms, as in chorea, are involuntary. They are, however, always limited in number, there often being only one and never more than three or four. In chorea there are always many. Careful observation shows that the motions in habit spasms are like those made for a definite purpose, as, for example, wrinkling the forehead when a hat is uncomfortable. In chorea the motions are purposeless. The motions in habit spasms are never made, moreover, when the muscles are being used for another purpose.

"Post-hemiplegic chorea" should not be mistaken for chorea, because it is a chronic condition, usually dating from birth, and is associated with increased deep reflexes and rigidity of the affected extremities. Other chronic organic diseases of the central nervous system which are sometimes mistaken for chorea are those due to lesions in the lenticular nucleus or its neighborhood, which are often associated with disturbance of speech, involuntary and incoördinate motions and ataxia.

Treatment.—There is no specific treatment for chorea. Stock vaccines and autogenous vaccines, prepared from organisms obtained from either the patient's blood or from the tonsils and teeth, are alike valueless. The sera from animals immunized with cultures obtained from the blood, teeth and tonsils of patients ill with the disease are also useless. I have never seen the auto-serum treatment, recommended by Goodman, do any good. I have, however, seen many very disagreeable reactions from it, although fortunately none that were fatal. I have had no experience with the injection of animal serum into the spinal canal, but do not see why it should do any more good, or as much, as the injection of human serum, as in the auto-serum treatment. Injections of solutions of magnesium sulphate, whether subcutaneously or intravenously, are of very doubtful value. I have done lumbar punctures repeatedly in chorea and have never seen them do any good.

In the first place, it is most important to remember that children with chorea are not responsible for the things which they do nor for their mental condition. They must, therefore, be treated patiently and

should not be punished. The most important elements in the treatment of chorea are rest, food and time. In my opinion every child with chorea, no matter how slight, should be put to bed and kept there until improvement is evident. It should not only be kept quiet physically, but also mentally. It is very important that the persons who are with a child ill with chorea be quiet, even tempered and not excitable. On this account it is often advisable to separate a child with chorea from its family, because its family is very likely to be of the neurotic type. Although the child is kept in bed, it should, of course, at the same time have the greatest possible amount of air and sunlight. Food must be pushed. There are no special indications as to the diet, except that it should be abundant, nutritious and easily digested.

Warm baths often quiet children with chorea. It is a good plan to give the child a warm bath just before it settles down for the night. For many years I had considerable faith in hydrotherapy in the treatment of chorea. I used to have the children wrapped in a sheet wrung out in cold water and then wrapped in a dry blanket outside of the sheet. They were kept wrapped up for about an hour. Owing to frequent changes in the house staff and nursing force during the war, this routine treatment was omitted in the hospital for a year before I found it out and the patients did just as well as before. Since then I am rather doubtful as to its efficacy. Electricity does no good. The children usually like massage, but I have never seen any benefit from it.

In spite of what has been said and written as to the curative action of arsenic in chorea, my own experience makes me believe that it has no such action. It may be of some use as a tonic, but I am sure that it has no specific action in this disease. After having decided for myself that it was useless, I have for many years allowed enthusiastic house officers to try it. Everyone has given it up and come around to my opinion. On the other hand, I have never seen peripheral neuritis or any other bad results from its use. If arsenic is to be used, it should be given in the form of Fowler's solution, beginning with from one to three drops, well diluted with water, after each meal, according to the age of the patient. After a couple of days the dose should be increased one drop at noon, the next day, one drop after supper, and the next, one drop after breakfast. After waiting two days it should be gradually increased again in the same way. These increases should be continued until toxic symptoms appear, such as gastro-intestinal disturbances, puffiness of the eyelids or irritation of the kidneys. The drug should then be stopped until these symptoms have disappeared, then begun again two drops lower and kept there. Most children can take about ten drops of Fowler's solution three times daily without showing toxic symptoms. If there are evidences of rheumatism, the children are made more comfortable by salicylic acid in some form, either as the salicylate of soda or aspirin. It has no influence, however, on the course of the chorea.

If children with chorea are very excitable and restless and sleep poorly, it is often a good plan to give them some sedative. The bromides are the simplest. Antipyrine may also be given. Large doses are usually required to do any good. From three to five grains a day for each year of age, given, of course, in divided doses, are usually necessary. Other drugs which are worthy of trial to relieve the symptoms are veronal and medinal, in doses of from two to five grains, several times daily, as needed. In my experience, however, it is seldom necessary to use any of these drugs, provided the child is kept quiet and given proper care.

In the very severe cases the child must be wrapped in cotton to protect it, and the bed padded. It may have to be restrained. In these cases it is absolutely necessary to quiet a child in order to save its life. In most instances morphine subcutaneously is the most useful drug. It must be given in doses large enough to attain the desired result. In some cases, however, it excites the child instead of quieting it. In such cases chloral given in very large doses, as recommended by Forcheimer, may act when the morphine does not. The beginning dose is five grains every four hours by mouth, or half as much more by the rectum. This dose should be rapidly increased, if necessary, until the child is quiet and sleeping, no matter how much is required. It is a dangerous method of treatment, but dangerous methods of treatment are justified in conditions where the patient is certain to die of the disease, unless it is relieved. The child should be kept asleep for twelve hours and then allowed to wake up. In the few cases in which I have seen this method of treatment used, all the motions had ceased when the patient waked up. It may be necessary in the severe cases to feed the child with a tube. It is useless to try to feed by the rectum.

The question always arises in every case of chorea in which there are evidences of disease of the tonsils or of the teeth whether the tonsils and teeth should be removed at once or operation delayed until after recovery. In my experience it is advisable to remove the teeth at once. I am not so certain about the tonsils. If they are left, there is, of course, the danger of continued absorption and danger of infection of the heart with the development of acute endocarditis. On the other hand, the chorea is almost always made worse by the operation and there is always a possibility of an acute infection of the glands or of the heart as the result of the operation, especially if there are any evidences of acute inflammation in the tonsils. In general, therefore, I think it is advisable not to remove the tonsils until convalescence is well established.

It is very important to look after the general health of a child that has had St. Vitus' dance. Its life must be regulated to avoid nervous strain and it must be carefully guarded against overfatigue. It is especially important to be sure that it is kept in good general condition in the spring.

HABIT SPASMS

Habit spasms are involuntary contractions of single muscles or groups of muscles, which result in the performance of some motion which at some time served a useful purpose. The same motion is made over and over again. A child usually does not make more than one motion, never more than three or four. It is very likely to stop making one motion and pick up another. Careful analysis of these motions shows that they are motions which originated because they were necessary to overcome some sort of discomfort. For example, the shoulder is twisted, as when a suspender is too tight. The neck is turned, as when the collar is rough. The nose is twisted, as when there are crusts in the nostril, or the forehead is wrinkled, as when the hat is too tight. They have kept up, however, until they have passed out of the child's control and have become involuntary. They are more common in boys than in girls and in children that are "run down" or members of neurotic families than in vigorous children or those of normal parents.

Habit spasms are often mistaken for chorea. The differential diagnosis is taken up under this disease.

Treatment.—The first thing to do in the treatment of habit spasms is to search for the cause and, if it is still present, remove it. The next thing to do is to regulate the child's life and diet so as to get it into the best possible general condition. It is impossible, unfortunately, to get it new parents. There are no drugs which have any direct action in stopping the spasms, although drugs like nux vomica and cod liver oil may aid in building up the general condition. It does no good to scold children with habit spasms, but encouragement and praise, and sometime judicious ridicule, may help them to overcome them. The best method of treatment is to have the child stand in front of a mirror and make the motions for from two to five minutes several times daily. The object of this procedure, suggested by Doctor Scripture, is not to make the child ashamed, but to educate him to control the motion. When he has acquired control again, he ceases to make it involuntarily. Children that have had habit spasms are very likely to develop others, however, if for any reason they get below par.

NYSTAGMUS AND SPASMUS NUTANS

Nystagmus, most often lateral, sometimes vertical, and occasionally oblique or mixed, is not very uncommon in infancy. It is often associated with imperfect vision, not infrequently the result of congenital cataracts, or with some congenital defect of the nervous system. In such cases careful examination of the eyes and of the nervous system reveals the cause. It is more often a functional condition and may be associated with spasmus nutans. In these cases the babies are intelligent, there are no other evidences of disease of the nervous system and the fundi are normal. Recovery is almost invariable. Nystagmus in childhood may be a symptom of some abnormality of the eyes or disturbance of vision, but is usually one of the evidences of serious organic disease of the brain.

Under the head of spasmus nutans are included both the lateral and nodding motions of the head occasionally seen in infancy, especially in the first year. These motions are usually associated with nystagmus, which usually, but not always, precedes them. They may or may not compensate and neutralize the motions of the eyes. They are usually, however, not as rapid as those of the eyes. They sometimes alternate with motions of the eyes, one beginning when the other stops, and sometimes stop when the baby is blindfolded. The chances are, therefore, that they are not directly secondary to the nystagmus, but are related to, not dependent upon it. A reasonable explanation is that the root nuclei of the muscles which control the motions of the head are close to the ocular nuclei and that any irritation of one set of nuclei may also affect the other. Impaired nutrition of these nuclei, increasing their irritability, explains the frequent association of both nystagmus and spasmus nutans with rickets and other disturbances of nutrition. Darkness, winter and unhygienic surroundings are, therefore, to be regarded not as the direct causes of nystagmus and spasmus nutans, but as indirect causes by bringing on disturbances of nutrition. Recovery from both the spasmus nutans and nystagmus takes place when the disturbance of nutrition is corrected. The treatment is, therefore, that of rickets and of disturbances of nutrition in general. It is not necessary to give bromide, as is often advised, and, moreover, it does no good.

Spasmus nutans in childhood is almost always simply a variety of habit spasm. In some cases, however, it may be due to some disturbance of vision or to incoördination of the eye muscles.

HYSTERIA

I must confess at once that it is very difficult for me to distinguish clearly and to draw definite lines between spoiled, nervous, neurotic, neurasthenic, psychasthenic and hysterical children. Furthermore, I have never been able to decide which are the most foolish, mothers, physicians to the rich, neurologists or psychiatrists, although I am certain that they are all worse than the children. I am also entirely out of sympathy with the nasty-minded Freudians, whether professional or lay, who attempt to explain all nervous manifestations in early life on a sexual basis.

The Nervous or Neurotic Child.—There are unquestionably a certain number of children who are nervously unstable and overexcitable. These characteristics may be evident even in infancy. Such children are usually the progeny of neurotic parents. Heredity probably has much less to do with their peculiarities, however, than the association with and example of their parents. If the infants and children of such neurotic parents are fortunate enough to be brought up and cared for by sane and calm people, they seldom show any evidences of nervous instability. On the other hand, if the offspring of stable and common sense people fall into the hands of neurotics, they quickly become unstable and excitable. Analysis of the life and routine of the nervous baby almost always shows that it is played with too much, "shown off" to friends both by day and by night, jumped and jolted about in its carriage or somebody's arms, not given a chance to sleep continuously and uninterruptedly and overstimulated in many ways, that is, the nervous baby is almost always an overtired baby. The remedy is obvious.

Analysis of the life and routine of the nervous child usually shows a similar state of affairs. It has too many lessons, too many excitements and social demands, does not have sufficient rest by day and sits up too late at night, that is, it also is overtired. Furthermore, a study of the family life almost always shows that it is constantly being nagged at and that it is not allowed to play and amuse itself in its own way. It is expected to live and act like an adult when it is only a child. In addition, it has the bad example of its parents always before it. The remedy is again obvious. The carrying out of the treatment is not, however, always easy.

The Spoiled Child.—Another type of the so-called nervous child is the spoiled child. In fact, most "nervous" children are simply spoiled children. Not infrequently, however, spoiled children are also overtired children and, as people who spoil their children are quite likely to be neurotic, they are obliged to live with neurotic and excitable people. The spoiled child is nervous because it pays it to be nervous. It gets more that way. The remedy is again obvious and again the treatment is hard to carry out. The parents must be trained first. When they are trained, the training of the child is simple. When the child is trained, the nervousness stops.

The Hysterical Child.—The hysterical child is usually simply a super-spoiled child. In some instances, however, there is no element of spoiling, although there is quite likely to be an element of bad neurotic family life. In occasional instances hysteria occurs in children that are not spoiled and who are fortunate enough to have parents who are sane and blessed with common sense. In such cases suggestion or imitation are usually at the bottom of the trouble.

The most common manifestations of what is ordinarily called hysteria in childhood are, in my experience, the fancied inability to swallow anything but liquid foods and the vomiting of food previously taken, when food which is not liked is offered or forced. Many children have learned to vomit whenever they please. Hysteria seems to me rather a strong term to apply to bad habits in spoiled children. A judicious combination of the cessation of suggestion, starvation and discipline cures these children promptly. The worst case of this sort that I ever saw was cured at once when he was obliged to re-eat the meal which he had just vomited. Before making a diagnosis of hysteria or bad habit it is necessary to be sure, however, that there is not a narrowing of the esophagus, either congenital or acquired, obstruction in the fauces, or an anaphylaxis to certain foods.

The next most common manifestation of hysteria, or foolishness, in children is hyperesthesia. The children scream when they are touched anywhere, but do not notice hard pressure when their attention is distracted. They do not mind the pressure of their clothes, but apparently suffer agonies when touched with the finger. They can sit on a chair without discomfort, but cannot bear the weight of the hand on their buttocks. In many instances the proper application of the hand in that locality results in cure. Hysterical anesthesia is, in my experience, very uncommon in childhood. Paresthesia is more common and usually takes the form of thinking that bugs or insects of various sorts are crawling over the skin. This manifestation is almost always the result of suggestion and is usually easily cured by a little common sense explanation and ignoring the complaint.

I have encountered occasionally a child who thought that it was paralyzed or that it could not talk, and have cured them in the usual ways by surprising them into walking or talking or by suggestion with electricity or some other spectacular treatment. I have seen a few cases of hysterical contractures which I have turned over to the orthopedic surgeons. It is most important, however, before making a diagnosis of hysteria in cases in which there is paralysis or spasm or other symptoms which may also be caused by organic lesions of the nervous system, to be sure that there really is no organic basis for them. In my experience, the diagnosis of hysteria under such conditions is very likely to be wrong and eventually the spasm or paralysis is shown to be due to some serious organic lesion. In fact, I have seen so many mistakes of this sort made that I have learned to look upon "hysteria" as a very serious and not infrequently fatal disease! When there are symptoms like paralysis, spasm, contractures and tremor, the diagnosis of hysteria is justifiable only after a most careful and complete examination and when the manifestations are inconsistent with those of any known organic disease of the nervous system or of any injury to the extremities.

The treatment of real hysteria, which becomes increasingly common as children approach the age of puberty, is along the same lines as in adults.

HEADACHES

It is very difficult to determine when an infant has a headache. The most characteristic symptoms of a pain in the head at this age are wrinkling the forehead, tossing or rolling the head and a whining cry. The most common cause is otitis media. Disease of the ethmoid cells and nasal obstruction also apparently cause headache in infancy. When a pain in

the head in infancy or early childhood is not associated with disease of the upper respiratory tract and its adnexa, it is likely to mean organic disease of the brain or its meninges.

Headaches after early childhood are due to the same causes as in adults. The relative frequency of these causes is, however, quite different. Headache in childhood is almost never due to disease of the kidneys, malaria, syphilis or high blood pressure. It may occur at the onset of any acute disease. It is often due to disease of the upper respiratory tract and its adnexa and nasal obstruction. If it is located in the forehead or occiput, it is almost always due to some error of refraction. Headaches occurring at irregular intervals are almost always due to disturbances of digestion. This is almost invariably the case, if they are accompanied by nausea or vomiting. Headaches are not uncommon in late childhood as the result of overwork and worry at school. Headaches from this cause are usually frontal, are more common in the spring, and in girls than in boys. Many nervous and excitable children have severe headaches, coming on suddenly and almost always located over one eye, as the result of excitement, fatigue or overheating. The true nature of these headaches is often overlooked. Headaches may also occasionally be due to insolation, organic disease of the brain or meninges, or decayed or impacted teeth.

Treatment.—The first element in the treatment of headache consists in the discovery and removal of the cause. A cathartic or an emetic relieve a headache due to an acute disturbance of digestion, but do not help one due to eyestrain. Glasses do not help a headache due to fatigue and worry or one due to constipation. An ice-bag to the head usually diminishes the severity of a headache, whatever its cause. So also do phenacetin, atipyrin, aspirin and the various combinations of the bromides and caffein. They should all be used sparingly in childhood, however, and great care be taken to avoid the establishment of a habit. The best treatment for the headaches due to over excitement and fatigue is to put the child to bed in a quiet, darkened room, put an ice-bag on its head and give it a dose of one of the bromides. One grain for each year of age is about right.

DISTURBANCES OF SLEEP

Sleep.—Many infants and most children are not allowed to sleep enough. Normally, an infant sleeps twenty hours out of the twenty-four during the first month, sixteen hours from one to six months, fifteen hours from six to twelve months, and fourteen hours during the second year. The normal child should sleep from eleven to fourteen hours daily from two to five years, from ten to eleven hours between five and ten years and from nine to ten hours throughout the rest of childhood. To do this the child of two must go to bed at six, that of five at six-thirty, that of ten at seven or seven-thirty and that of fifteen at eight-thirty, or nine at the latest. The daily rest should be kept up at least until two sessions of school prevent it, although children can hardly be expected to sleep after they are three or four years old. Just as in adults, there is considerable variation, however, in the amount of sleep needed by individual children. It is important, too, that the sleep is taken at the proper time. Sleeping late in the morning does not make up for hours lost in the evening. The child should go to bed early enough so that it is not necessary to wake it in the morning.

Delay in Going to Sleep.—This is almost always due to excitement, play having been kept up until the last minute. It is often due to the

pernicious habit which many fathers have of romping with young children just before they go to bed. It is also not infrequently due to overfatigue, and sometimes in older children to worry about their work or incidents which have happened during the day.

Restlessness.—Young children do not normally sleep as quietly as older children and adults. Restlessness may be due to an excess of bed clothes or to a deficiency or excess of fresh or cool air. It may also be due to interference with nasal respiration from adenoids. It is more often due, however, to too hearty a supper, overfatigue or excitement. It may be occasionally due to pin worms, but never to round worms, as is so commonly supposed. The treatment is obviously the removal of the cause.

Sleeplessness.—Sleeplessness is due to the same causes as delay in going to sleep and restless sleep, and should be treated in the same way. It is never right to give sedatives, even for a few nights, with the idea of breaking up the habit. The treatment is the removal of the cause.

Night Terrors—Pavor Nocturnus.—Night terrors are not at all uncommon in early and middle childhood. They are most likely to occur from one to three hours after the child has gone to sleep. They may be divided roughly into two classes. In both the child screams with terror and for a time is not oriented. In one type the child is afraid of some definite thing and can tell what it is. In the other type the child does not know what he was afraid of and, when he comes to, does not appreciate that he was afraid. In neither type does the child remember anything about it in the morning. In the type in which the child is afraid of a definite thing, careful investigation always discloses the cause of its fear. He has really been frightened by something at some time or he has had stories told him which have frightened him. In the type in which the child is not afraid of a definite thing, the trouble is almost invariably an indigestible or too hearty a supper.

Children should not be scolded for having night terrors. If something has frightened them in the past, it must be explained to them that the thing cannot hurt them now and that they are perfectly safe in bed in their own home. If they have been frightened by stories, the type of stories which they are told must be changed. Children should always go to bed happy and with a quiet, easy mind. It is an easy matter to give a light supper suitable for the age. Under no condition should children with night terrors be given sedatives to quiet them.

BAD HABITS

It seems to be easier at all periods of life to pick up bad habits than good. This is as true of babies as of adults. In my opinion too much importance is attached to many of the habits which babies and children have. They are looked upon as immoral or evidences of degeneration, when they are simply foolish.

Thumb Sucking.—It is as natural for a baby to suck its thumb, or anything else that it can get into its mouth, as it is for it to breathe and empty its bladder and rectum. They are all reflex actions necessary to life. If the baby does not suck some kind of a nipple, it dies of starvation. It has not sufficient mental capacity to know that it cannot get milk from sucking other things. To look upon sucking in a baby as a sexual manifestation shows, it seems to me, moral degeneration in those who consider it such rather than in the baby. It is very foolish, however, to allow a baby to suck its thumb or other objects, because it does the

baby no good, may disturb its digestion, makes it fussy when it has nothing to suck and may alter the shape of its mouth. Babies should never be given "pacifiers," because they are also almost always dirty and are likely to cause infection of the mouth and sometimes of the digestive tract.

It is easy to stop a baby from sucking its thumb or fingers by confining or tying down its hands. Cardboard cuffs, which prevent bending the arms at the elbows, always prevent it. They are much better than aluminum mitts, which make the hands sweat and interfere with their

FIG. 154.—Cardboard cuffs.

use. The baby may also hurt itself with them. It is useless to put anything which tastes badly on the hands to prevent sucking. It never does any good. The way to prevent a baby from sucking other things is not to give them to it to suck.

Tongue Sucking.—Tongue sucking is simply a foolish habit which children sometimes acquire, usually as the result of some irritation about the teeth, or because they put the tongue in the space left by some tooth which has come out. It is not a manifestation of sexual perversion. It is more likely, however, to occur in children of neurotic parentage and in those who are overtired. The treatment is to get the child into the

best possible general condition by regulation of its life and diet and to convince it of the foolishness of the habit.

Nail Biting.—Nail biting is another foolish habit, not only of childhood but of adult life. It, again, is more common in children of neurotic parentage and in those who are overtired, either physically or nervously. The first element in the treatment is to get them into the best possible general condition. Children who bite their nails are usually old enough to be reasoned with and to have strength of mind enough to stop, if they will. It is useless to attempt to stop nail biting by putting anything which tastes badly on the fingers. The child will either suck it off or wash it off. In obstinate cases the cardboard cuffs, recommended for thumb sucking, may be used.

Pica.—Babies that are old enough to run about and young children not very infrequently get into the habit of eating curious things, the most common of which are sand, dirt, plaster and the wool which they pick from blankets and their clothes. Some children eat the paint off of their cribs. I have seen several cases of lead poisoning in infants from this cause. Other children are very fond of eating wood and will gnaw the furniture, window sills and stairs. I remember one instance in which a little child had eaten away all of the projecting portion of the treads of the front stairs that was not covered by carpet. Such children are almost always of neurotic parentage or have mothers who are so much interested in other things that they have no time to watch their children properly. They are very likely to have disturbances of digestion, and it is often very hard to determine whether the disturbance of digestion is the cause of the abnormal appetite or the abnormal things which have been eaten the cause of the indigestion.

The treatment of pica consists, of course, in stopping the child from eating the abnormal articles until it gets over its foolish habit. This often requires much attention and watchfulness, more, unfortunately, than many mothers are willing to give. The cardboard cuffs, already mentioned, prevent the child from putting its hands to its mouth and, therefore, from putting things into its mouth. A muzzle of some sort may be used in the most obstinate cases. The diet must also be carefully regulated and the digestion straightened out. Punishment is seldom of any use.

Head Banging.—This is another of the foolish tricks which some children pick up. It is not a sign of "thwarted desire," but is sometimes an evidence of anger or of discomfort in the head. Children are especially likely to bang their heads when they go to bed at night, sometimes on the pillow, sometimes on the head of the bed or wall. I have known a whole family of children to put themselves to sleep in this way. This habit, like all the others, is more common in the children of neurotic parents, especially if they live with them, and in those who are overtired and overstimulated. The treatment consists in preventing them from banging the head until they have got over the habit. In the meantime their home surroundings should be changed as far as possible and they should be put into the best possible general condition.

Pseudomasturbation.—This term is used to describe the habit which a certain number of babies and young children have of playing with the external genitals. It is not proper, in my opinion, to apply the term masturbation to this habit. It is as natural for a little boy to take hold of his penis and a little girl to rub her clitoris as it is for them to rub their noses or pull their ears. Occasionally a child finds that the sensation from playing with the penis or the clitoris is more pleasurable than that

obtained from rubbing the nose or pulling the ear and, consequently, keeps on doing it. That is all there is to it. Children are more likely, of course, to pick up this habit, if there is some local irritation of the external genitals. Little boys usually use their hands. I have seen several, however, that rubbed the penis with their toes. Sometimes they rub up against the furniture or some hard object. Little girls seldom use their hands. They are much more likely to rub their thighs together or to rub up against the furniture or some hard object. I have never seen a little boy have an orgasm, but I have repeatedly seen little girls become flushed and then relax, perspiring.

Although this habit should not be regarded as masturbation, it is advisable to stop it in order to prevent it from leading to masturbation later. It is important to remove all sources of external irritation. The diapers must be comfortable and not too rough. The foreskin should be pulled back and the glans kept clean in both boys and girls. In babies and little children the clothes can usually be so fixed that the child cannot get at the external genitals with the hand. Girls must be watched to prevent them from rubbing up against things. The rubbing of the thighs together can sometimes be prevented by putting a large wad of diaper between the thighs or by putting a shield over the vulva. The best way to prevent it, however, is by the application of a crutch splint applied to the thighs, which keeps them apart. It is useless to scold babies and little children for this habit, although they may sometimes be spoken to pleasantly about it and told not to do it.

MASTURBATION

It is very difficult, as children grow older, to know when pseudomasturbation ceases and masturbation begins. Even though in middle childhood the term masturbation may have to be used to describe the habit which children have of playing with the external genitals, it does not seem to me that it should be looked on in the same way as masturbation after puberty and in the adult, because, although there is, of course, sexual excitement and pleasure, it does not represent a sexual orgasm as it does after puberty. It is a bad habit, but not in the same way as it is later. It must, of course, be put a stop to in order that it may not be continued during adolescence and later life. It does not mean that the child is to be eternally damned or become a useless member of society, as so many people seem to think. It never leads to feeblemindedness or idiocy. If children that masturbate are feebleminded or idiots, they masturbate because they are feebleminded and are not feebleminded because they masturbate. In my experience, this habit has little or no influence on the general condition. It may be that I am a poor observer, but I have never been able by looking at a child to determine, as so many people seem to think they can, whether it masturbated or not. I do not believe that even the most observant parent or nurse can tell by the looks of a child in the morning whether it masturbated in the night or not. They certainly cannot tell by the looks of their friends in the morning whether they had sexual intercourse during the night or not. How can they, therefore, expect to tell whether a child has played with itself in the night or not. I have never seen those sly, furtive and ashamed expressions, which are so often described. If they are seen, I am sure that it is because the children have been scolded and watched so much that they are self conscious, not because of any habit which they may have.

Treatment.—The first and most important thing in the treatment of this so-called masturbation in children is to look upon it from the proper point of view. It must not be considered as a terrible thing, which is certain to ruin the child's life, but must be looked on as simply an unusually bad habit. The child should be told that it must stop doing it in the same way that it is told that it must stop any other bad habit. It must not be spoken of with bated breath and its importance exaggerated. The greatest care must be taken not to increase the habit by talking so much about it that it is constantly suggested to the child. It is perfectly useless to tell a child that this habit will do it a great deal of harm, make it feebleminded or cause it to be damned forever. The child does not appreciate the importance of these things anyway and, furthermore, having some sense, as most children have, knows that these statements are untrue. Scolding and punishment are of little use. Praise and assistance through gaining the child's confidence are of great help. The greatest care must be taken to keep the child's confidence and not to make it a liar as well as a masturbater.

All sources of local irritation about the genitals must be looked for and removed. The prepuce should be pulled back, so that the glans can be cleaned in both sexes. If this is impossible, circumcision may sometimes be necessary, in girls as well as in boys. I have never, however, known circumcision alone either to prevent or to cure masturbation. There is, so far as I know, no evidence to show that masturbation is less common among the Jews than among Christians. I have known a boy to masturbate while the stitches were still in after a circumcision. This same boy a few months later said that he guessed he would stop, and did, and laughed at his parents for thinking they could stop him by circumcising him. It is absolutely wicked to think of amputating the clitoris in little girls, as I have known done.

It is probably necessary in some cases to watch children carefully, and, perhaps, to have someone with them when they go to sleep at night to prevent them from playing with themselves. I am inclined to think, however, that such constant attention more often does harm than good and that it often increases the habit through suggestion. It is far better in most instances to gain the child's confidence and to aid it in building up its own will power. In some extreme cases, however, it is necessary for a time to confine the child at night so that it cannot use its hands. This is often hard to do with strong, active children. It can be done, however, by putting the child in a strait-jacket and tying it down on a Bradford frame.

In many instances the mothers of children who masturbate, especially if they are girls, are neurotic and consequently have a very bad influence on the children. In such cases it is advisable to get some sane, calm person to take charge of the child. It is extremely important, however, not to get a nurse that is as neurotic as the mother, has the wrong point of view, or masturbates herself. It goes without saying, of course, that everything should be done to build up the general condition of these children, provided it is necessary, and that their life should be so regulated that they have plenty of amusement and are comfortably tired at night. It is not justifiable to give hypnotics or sedatives to these children.

THOMSEN'S DISEASE

This disease, also known as *Myotonia Congenita*, is very rare. It is usually hereditary and several members of a family are often affected.

It is said to be somewhat more common in males than in females. The symptoms may develop at any period of life. Not infrequently they appear during the first decade and increase in severity as the muscular development increases, reaching their maximum in the second and third decades. There is considerable doubt as to whether the disease is primarily of the nervous system or of the muscles. The only pathologic lesion which has been found is great hypertrophy of the muscle fibres, with an increase in the nuclei and in the interstitital connective tissue. In some instances there has also been vacuolation of the muscle fibers.

Symptomatology.—The characteristic symptom of Thomsen's disease is rigidity of the muscles when motion is attempted after a period of rest. The rigidity lasts from five to thirty seconds. The muscles then act normally until after another period of rest. The muscular rigidity is more marked when the attempt is made to rise after sitting down for a time. All the muscles may be involved, including those of the tongue and the muscles of speech. The rigidity is increased by excitement, fear and cold. The muscular development is increased. The rigidity persists throughout life, but does not shorten it.

Diagnosis.—It is very easy to recognize this disease, if one knows about it or has seen a case of it before. Otherwise the diagnosis is very difficult. The mechanical excitability of the muscles is much increased. Tapping the muscles with a hammer causes a slow, tonic contraction of the fibres, which does not relax for some time. The response of the nerves to both the faradic and galvanic currents is essentially normal. That of the muscles to direct stimulation with either current is much increased. It is sluggish, but persists much longer than normally. Erb has described a special reaction to the direct galvanic current, similar to the reaction of degeneration, known as the myotonic reaction. This is a wave-like contraction, passing slowly from the kathode to the anode with strong currents.

Treatment.—This disease is influenced but little by treatment. It is said that prolonged rest may help, as may also regular, vigorous exercise. Massage is said to help it a little. Electricity and drugs are useless.

Diseases of the Brain

Acute Encephalitis

This disease, which is commonly called epidemic or lethargic encephalitis, is described better by the term acute encephalitis, because it occurs sporadically as well as in epidemics and lethargic manifestations are often very slight or absent. It is not a new disease, but has been much more common during the last few years. It may occur at any age and is about as common in children as in adults. It is more common in males than in females.

Etiology.—Encephalitis is certainly not a manifestation of influenza. It is doubtful if influenza is even a predisposing cause of encephalitis. It is not identical with poliomyelitis—infantile paralysis. Encephalitis is undoubtedly caused by some form of microörganism. This organism has not yet been positively identified, but probably belongs in the class of the filterable viruses. Encephalitis is unquestionably contagious. The contagiousness is, however, very slight. There is some evidence to show that the contagion is contained in and carried by the secretions of the nasopharynx.

Pathology.—The lesions of encephalitis are confined to the central nervous system and affect the brain far more than the cord, which is usually not involved, unless in its upper portion. The gray matter of the cortex and cerebellum may be involved, but that at the base of the brain and in the pons and medulla is more often and more severely affected. The lesions are usually more severe anteriorly than posteriorly. They may be either nodular or diffuse. They consist of accumulations of cells about the blood vessels and cellular infiltrations in the nerve tissues. It is evident, therefore, that the infection comes through the blood. There are also often microscopic hemorrhages and not infrequently edema of the tissues. The cells are chiefly mononuclear.

Symptomatology and Prognosis.—It is almost impossible to describe the symptomatology of acute encephalitis, because no two cases are exactly alike. They necessarily depend upon and vary with the severity, extent and location of the pathologic lesions. The onset may be either acute or slow. Children either do not notice or do not say anything about the early symptoms, if they are slight, so that the onset is often apparently more acute than it really is. Headache is not uncommon in the beginning. In most instances the child is more or less somnolent and stupid. The somnolence may develop quickly or slowly and may go on to absolute coma. In other instances, however, the child is excitable, nervous and restless. Marked delirium, maniacal and other psychotic manifestations are, however, apparently less common in children than in adults. Convulsions may occur, but are not common. The temperature may be moderately or considerably elevated. In some instances it is normal. There is nothing constant about it. The pulse and respiration usually vary with the temperature. The nuclei of the ocular muscles are often involved, especially in the somnolent cases, resulting in various ocular paralyses, of which ptosis is the most common. Nystagmus is not uncommon. There may be paralysis of other facial muscles and in some instances there is disturbance of deglutition and sometimes of the pulse and respiration. If the cerebellum is involved, or the corpus striatum, there may be marked ataxia and incoördination. Twitchings are not uncommon. They may be fine or coarse, localized or general. Catatonia is also not uncommon. Symptoms of meningeal irritation, such as stiffness of the neck, Kernig's sign, and Brudzinski's neck sign, are uncommon. There is very seldom involvement enough of the cortex to cause paralysis or spasm of the extremities. There is nothing characteristic about the deep reflexes, which are usually normal, but may be either increased or diminished. Sensation is normal, but the child may be too stupid to respond. The fundi usually show nothing abnormal, but changes in the optic discs are seen in an occasional case. These may be quite marked.

The leucocyte count may be normal or moderately increased. It seldom goes above 20,000. The increase is in the polynuclear neutrophiles. Blood cultures are sterile. The urine is normal or shows the changes of acute degenerative nephritis. The spinal fluid is often somewhat increased in amount. It is usually clear, but may be slightly blood tinged. A clot seldom forms in it. There is usually a slight increase in the globulin. The sugar is normal or increased. There is nothing characteristic about the colloidal gold test. The cell count may be normal, but is usually increased. The number of cells is ordinarily not more than 100 or 150, but may be as high as 1500. The cells are usually almost all mononuclear, but may be both mononuclear and polynuclear,

and in rare instances the polynuclears predominate. The spinal fluid shows no organisms and is sterile.

The duration of acute encephalitis is very variable. Abortive cases undoubtedly occur in which the duration is only a few days or a week and the symptoms very slight. It is presumable also that, as in poliomyelitis, many cases are so mild that the true nature of the disease is not suspected. The usual duration of the acute symptoms is from two or three weeks to two or three months. Remissions and exacerbations of the symptoms are not uncommon. The mortality is probably between 15% and 25%. In a certain number of cases, probably at least 25%, marked psychiatric disturbances persist after the child is absolutely well in other ways or similar disturbances develop after a few months or a year in which the child has seemed normal. When these symptoms develop late, it is presumable that they are due to permanent changes in the nervous system produced by the disease. When there is no period of intermission, it is hard to know whether the pathologic process is still active or whether the symptoms are due to lesions caused by it. It is certain that no child can be considered safe from the sequelae of acute encephalitis until it has been without symptoms of any sort for at least a year.

Sequelæ.—*Insomnia* is probably the most common sequela of encephalitis in childhood. The children are unable to sleep during the evening and night and do not drop off until the early morning hours. They then sleep quietly until noon or even later. During the hours in which they cannot sleep they are quite likely to be very restless and noisy. Sleep usually becomes normal, however, after a few weeks or months.

Change in Character and Disposition.—Another common sequela is a complete change in the character and disposition of the children. They become noisy and abusive; sometimes are very talkative, restless and emotional. Others are obscene and sexually uncontrolled. Some are violent and destructive.

Disturbance of Motility.—Choreiform and all sorts of incoordinate movements are not uncommon. Ataxic manifestations, similar to those of lesions of the corpus striatum, are also not infrequent. The Parkinsonian syndrome is not as common as in adults.

Hysterical Reactions, Affective Disorders and Tics of various sorts are occasional sequelae. So also is *Mental Deficiency*, especially when the encephalitis occurs in infancy. In rare instances there are evidences of disturbances of the functions of the pituitary gland. *Fröhlich's Syndrome* is the most common manifestation.

Various Paralyses of the muscles supplied by the cranial nerves, especially of those of the eyes, may persist. In rare instances there may be, perhaps, spasm of the extremities with increased deep reflexes.

Children usually recover from the insomnia. The chances are about even for recovery from the changes in disposition and the various hysterical reactions and tics. The disturbances of motility and the various paralyses are likely to persist. Mental deficiency and the disturbances of the functions of the pituitary gland are almost certain to be permanent.

Diagnosis.—When the onset of encephalitis is rapid, it may be confused with any of the acute forms of meningitis, except the tuberculous, or with those types of infantile paralysis in which the brain is especially involved. The signs of meningeal irritation are, however, usually less marked than in meningitis, as are also those of increased intracranial pressure. If the patient is an infant, the anterior fontanelle usually bulges in meningitis and does not in encephalitis. There is nothing

about the white count, temperature, pulse, respiration or other symptoms which is of any aid in differentiating between them. Lumbar puncture, however, clears up the diagnosis at once between meningitis and encephalitis. The spinal fluid is clear in encephalitis and cloudy or purulent in meningitis. The cell count is almost never over 150 in encephalitis and always much higher in meningitis. The cells, moreover, are almost entirely mononuclear in encephalitis and polynuclear in meningitis. There are no organisms to be seen in smears of the spinal fluid in encephalitis, while there are almost always some in meningococcus meningitis and they are very abundant in influenza and pneumococcus meningitis and in meningitis due to the pus organisms. Cultures from the spinal fluid in encephalitis are sterile, while there is an abundant growth in meningitis, except sometimes in the meningococcus type.

The diagnosis between encephalitis and the type of infantile paralysis in which the brain is chiefly affected is in the beginning often impossible. If there are other cases of either disease at the time, the chances are, of course, that the patient has this disease. There is nothing about the symptomatology and physical signs which serves to distinguish them. The characteristics of the spinal fluid are exactly the same, except that in encephalitis it may sometimes be blood-tinged. If the patient dies quickly, no diagnosis can be made. If the patient survives, however, symptoms of spinal involvement usually develop, if the disease is infantile paralysis, and some of the more characteristic manifestations appear, if it is encephalitis.

When the onset is slow, it is often hard to distinguish between encephalitis and tuberculous meningitis and, when the course is also prolonged, it is difficult to know whether the symptoms are due to a tumor at the base of the brain or encephalitis. The onset, symptomatology, temperature, pulse and respiration may for one or even two weeks be the same in both encephalitis and tuberculous meningitis. There are more likely to be signs of meningeal irritation and of increased intracranial pressure in tuberculous meningitis than in encephalitis, but they may be entirely lacking. The presence of other foci of tuberculosis in the body is in favor of tuberculous meningitis, but their absence is of no importance. The tuberculin test is of no value, because it is often negative in tuberculous meningitis and may be positive in encephalitis, if there is a tuberculous focus anywhere in the body. A polynuclear leucocytosis is in favor of encephalitis, but not positive proof of it, because there is sometimes a polynuclear leucocytosis in tuberculous meningitis and sometimes no change in the white cells in encephalitis. The findings in the spinal fluid are almost identical. The sugar is usually diminished in tuberculous meningitis and is usually normal or somewhat increased in encephalitis. There is nothing constant about the sugar content in these conditions, however, so that its estimation is of little practical value. Anything above 65 mg. of sugar per 100 c.cm. of fluid is, nevertheless, suggestive of encephalitis. The finding of tubercle bacilli in the fluid is, of course, positive proof that the disease is tuberculous meningitis. Not finding them does not prove that the disease is encephalitis, because they are often missed in tuberculous meningitis. A diagnosis is usually possible in the course of two weeks or less, as by that time, if the trouble is tuberculous meningitis, the characteristic picture of this disease has usually developed. If the illness lasts more than three weeks in a baby or five weeks in a child, it is almost certainly not tuberculous meningitis.

If the inflammatory process in encephalitis involves chiefly the nuclei of the cranial nerves, the symptoms may be very similar to those of a tumor at the base of the brain. There is, however, more likely to be fever and a polynuclear leucocytosis in encephalitis, while pressure symptoms are more common with a tumor. The presence of changes in the optic discs is much in favor of tumor. The spinal fluid is usually normal with a tumor and is also normal after the acute stage of encephalitis is over. If the spinal fluid is not normal, the chances are, therefore, strongly in favor of encephalitis, while, if it is normal, either diagnosis may be correct.

Treatment.—There is no specific treatment for encephalitis. Nothing can be done during the acute stage except to take care of the child as well as possible and to treat the symptoms as they arise. The treatment of the sequelae is equally unsatisfactory. It is usually inadvisable to give hypnotics for the insomnia. It is better to accept it as a manifestation of the disease and accommodate the child's life and routine to it as far as possible, remembering that the condition will remedy itself after a time. The changes in disposition, hysterical reactions and tics cannot be helped by drugs, but only by patience, tact and mental training. The treatment of the various disturbances of motility and paralysis is the same as when they are due to other causes.

HYDROCEPHALUS

Hydrocephalus, as the term is ordinarily used, is synonymous with internal hydrocephalus, that is, a collection of watery fluid in the ventricles of the brain. There may be a slight excess of fluid outside of the brain in certain acute inflammatory or toxic conditions and, perhaps, as the result of venous congestion in heart disease or whooping-cough. There may also be a considerable excess of fluid between the brain and the skull when there are gross cerebral defects. The term external hydrocephalus is applied to these conditions, all of which are of little importance.

It is easier to understand the etiology, classification and symptomatology of hydrocephalus, if the anatomical conditions are borne in mind. As is shown by the accompanying drawings (Figs. 155, 156), the lateral ventricles are connected with the third ventricle by the foramina of Monroe, the third ventricle with the fourth by the aqueduct of Sylvius, and the fourth ventricle with the subarachnoid space by the foramen of Magendie and the foramina of Luschka. The velum interpositum is a prolongation of the pia mater, which passes through the transverse fissure beneath the posterior rounded border of the corpus callosum. It divides, passing through the foramina of Monroe to the lateral ventricles, and also projects into the third ventricle. The choroid plexus occupies the margins of the velum interpositum, both in the lateral ventricles and in the third ventricle. The arteries of the choroid plexus enter the ventricles at the descending cornu. The veins of the choroid plexus terminate in the veins of Galen, which unite into one vein, which terminates in the straight sinus. There are no lateral anastomoses with these veins until after they have joined. The ependyma is the membrane lining the lateral ventricles.

The subarachnoid space surrounds the brain and cord. There are three enlargements of this space, known respectively as the cisterna interpeduncularis, the cisterna pontis and the cisterna cerebellomedullaris, or cisterna magna. The only connections between the ventricular

system and the subarachnoid space are through the foramen of Magendie and the foramina of Luschka from the fourth ventricle.

The cerebrospinal fluid is formed within the ventricles as the result of the activity of the choroid plexus. Normally, the cerebrospinal fluid passes from the lateral ventricles through the third and fourth ventricles

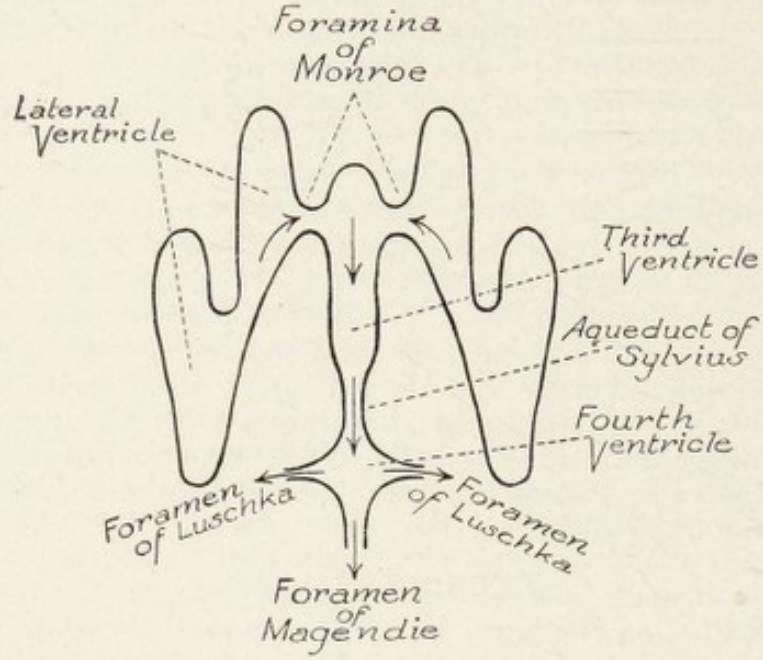

FIG. 155.—Ventricles and foramina of brain.

to the subarachnoid space. There is very little absorption of cerebrospinal fluid in the ventricles. Absorption takes place both from the cerebral and the spinal subarachnoid systems, but the absorption is much greater through the cerebral than through the spinal system, because it is so much larger. Absorption takes place directly into the

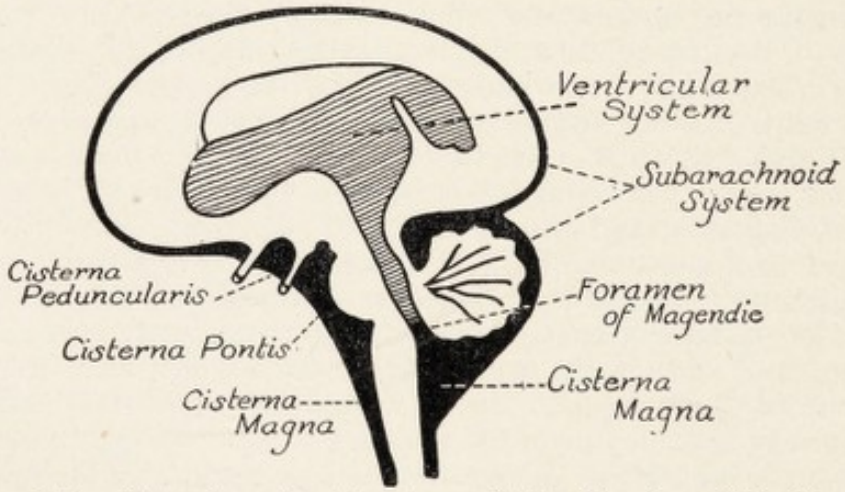

FIG. 156.—The subarachnoid system. (After Dandy and Blackfan.)

blood, not through the lymphatics. The cerebrospinal fluid is completely absorbed and renewed from four to six times in the twenty-four hours.

It is evident that hydrocephalus may develop, if there is an increase in the secretion of the cerebrospinal fluid without any increase in the powers of absorption or if there is a diminution in the powers of absorption while the secretion remains normal. It is also evident that hydro-

cephalus may result, if there is obstruction at any of the foramina connecting the ventricles or of those connecting the fourth ventricle with the subarachnoid space. If there is obstruction of one of the foramina of Monroe, dilatation of one lateral ventricle results. If there is obstruction of the aqueduct of Sylvius, dilatation of both lateral ventricles and the third ventricle results. If there is obstruction of the foramen of Magendie and of the foramina of Luschka or interference with absorption through the subarachnoid space, there is dilatation of the whole ventricular system. If there is obstruction of the veins of Galen, before or just where they unite, causing venous congestion in the choroid plexus, dilatation of the lateral ventricles and the third ventricle may sometimes develop. When the hydrocephalus is due to interference with the passage of the cerebrospinal fluid to the subarachnoid space, it is called *obstructive*. When it is due to interference with absorption and there is no interference with the passage of the fluid to the subarachnoid space, it is known as *communicating*. The two types may, of course, be combined. It is evident that when hydrocephalus is of the obstructive type but little fluid can be obtained by lumbar puncture, while, if it is of the communicating type, large amounts can be obtained. As much as 25 c.cm. may, however, be obtained, even in the completely obstructive type, while, if the obstruction is in one of the foramina of Monroe, a normal amount may be obtained. Nothing can be determined by an examination of the cerebrospinal fluid as to the cause of the hydrocephalus.

Under normal conditions, when a solution of phenolsulphonaphthalein is injected into a lateral ventricle, it is present in the spinal subarachnoid space in from one to three minutes. When it is injected into the ventricles, it appears in the urine in from ten to twelve minutes, and from 12% to 20% is excreted in two hours. When it is injected into the subarachnoid space, it appears in the urine in from six to eight minutes, and from 35 to 60% is excreted in two hours. It is evident, therefore, that by the use of phenolsulphonephthalein it is possible to determine whether hydrocephalus is of the obstructive or of the communicating types. In this test one c.cm. of a solution containing six mg. of phenolsulphonephthalein, carefully neutralized, and diluted with two or three c.cm. of cerebrospinal fluid, is used.

It is evident that, whatever the cause of the hydrocephalus, the dilatation of the ventricles must necessarily compress and thin out the brain substance surrounding them. If the hydrocephalus develops before birth or during the first nine months of life, before the cranial bones have grmly united, the fontanelles become enlarged and the bones separated as the result of the increase in pressure. After nine months, except in rare instances, there is no enlargement of the cranium, the whole of the pressure being exerted on the brain itself. The manifestations of the pressure on the various centers and nuclei of the brain are the same as when the pressure is due to other causes.

Etiology.—Hydrocephalus, developing before birth, may be due to some congenital malformation of the brain interfering with the normal passage of the cerebrospinal fluid or to meningitis occurring during fetal life. This may result in the formation of adhesions which may obstruct the foramina leading from the fourth ventricle to the subarachnoid space, compress the veins of Galen or obliterate the subarachnoid space. Hydrocephalus, developing after birth, is most often due to the inflammatory exudate occurring in meningitis, adhesions being formed as the result of the inflammation, or to the pressure of a tumor. In rare instances

hydrocephalus, developing both before and after birth, may be due to syphilis, which may cause either mechanical obstruction or disease of the meninges.

Symptomatology.—When hydrocephalus is present at birth or develops before the cranial bones have united, there is marked enlargement of the cranium. The enlargement of the cranium is symmetrical, because the pressure from within is uniform. The fontanelles bulge and the cranial bones are widely separated. The face is not small, but appears so, because the cranium is so large. The pressure from within presses down the orbital plates, so that the eyes are pushed forward and the whites of the eyes show above the irides. The head is so heavy that the baby is unable to hold it up. As the result of the pressure of the fluid on the cortical centers and, perhaps, also on the nerve tracts, various forms and combinations of paralysis and spasm develop, usually of the spastic type. Convulsions are not uncommon from the same cause. Blindness also is not uncommon as the result of the pressure. If the babies are unfortunate enough to survive, they are likely to be feebleminded.

FIG. 157.—Moderate hydrocephalus.

When hydrocephalus develops after the cranial bones have united, there may be bulging of the anterior fontanelle, if it has not already closed. There can be, however, no enlargement of the cranium. The orbital plates may be pushed down, however, and the eyes prominent. Percussion over the parietal bones gives a more tympanitic note than normal.

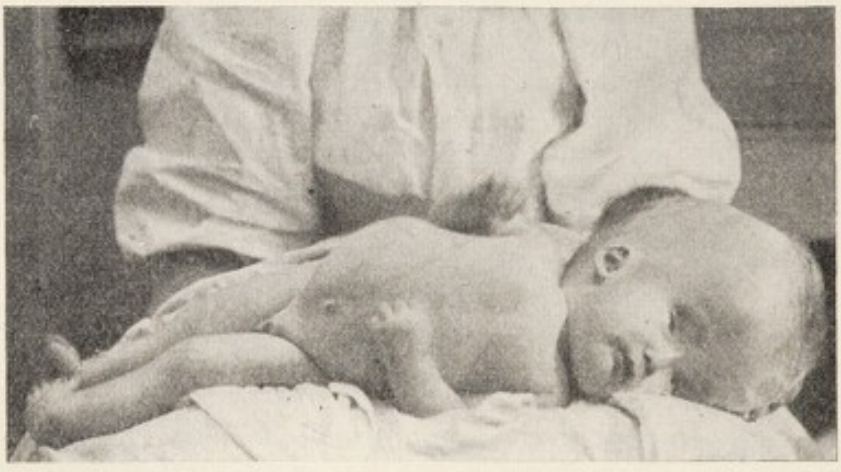

FIG. 158.—Marked hydrocephalus.

(Macewen's sign.) This is best elicited by placing the bell of the stethescope on the forehead and percussing with a hammer. The change in sound is more definite when the head is held sidewise and the percussion made on the down side. The symptoms are, therefore, almost entirely those due to the increased intracranial pressure.

Diagnosis.—The diagnosis of hydrocephalus is usually very simple in early infancy. Occasionally, however, the diagnosis of hydrocephalus is made on a perfectly normal baby that happens to have a large head. When the head is simply large, there is no bulging of the anterior fontanelle, no separation of the cranial bones, no pushing down of the orbital plates with bulging of the eyes and no evidences of increased intracranial pressure. The rhachitic head is also sometimes confused with hydrocephalus. The head in hydrocephalus is uniformly enlarged as the result of pressure from within. The rhachitic head is irregularly enlarged as the result of bony overgrowth at the centers of ossification of the frontal and parietal bones. The hydrocephalic head is rounded on top, the rhachitic head flattened. The anterior fontanelle bulges in hydrocephalus and is level or depressed in rickets. The eyes are prominent in hydrocephalus, and normal in rickets. There are no evidences of an increase in intracranial pressure in rickets and there are usually many other physical signs of rickets present.

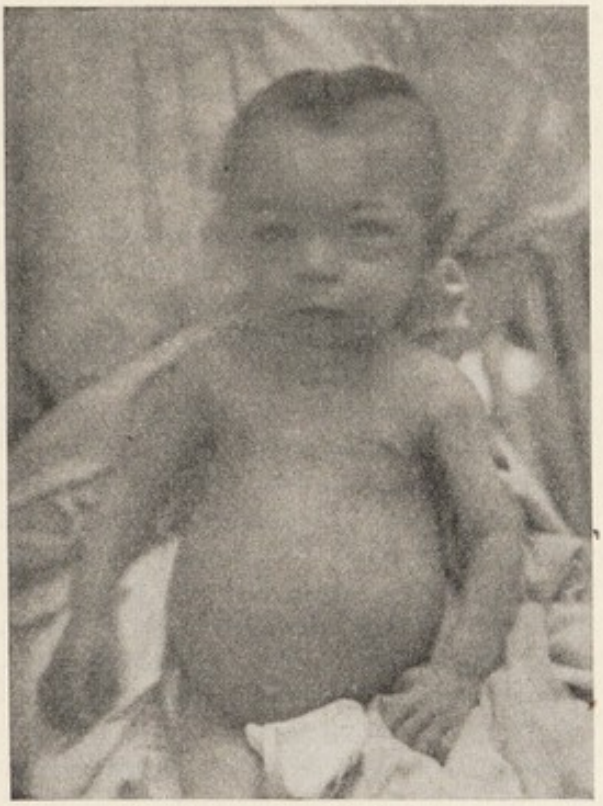

FIG. 159.—Rachitic head.

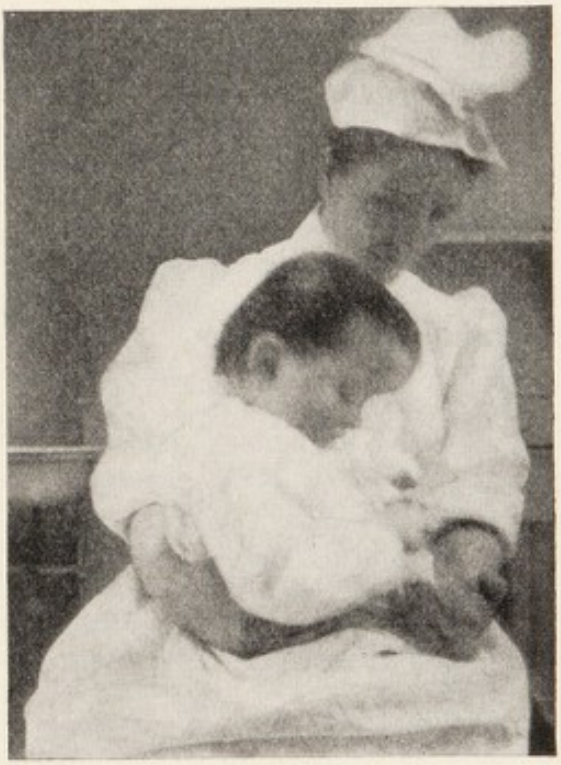

FIG. 160.—Sarcoma of skull.

When hydrocephalus develops in the course of acute meningitis, there is an increase in the intracranial pressure and, consequently, an increase in the symptoms due to this cause. It is usually impossible, however, to distinguish these symptoms from those already present as the result of the disease. A positive diagnosis can only be made when it is impossible to obtain more than a little fluid by lumbar puncture, provided the fluid is not so thick that it will not run through the needle. When the cerebrospinal fluid ceases to run out freely and is not extremely thick, it is almost certain that the foramina connecting the fourth ventricle with the subarachnoid space have become obstructed, resulting in an accumulation of fluid in the ventricles. When symptoms of increased intracranial pressure develop during convalescence from or even some

time after an attack of meningitis, it is almost certain that hydrocephalus is the cause. When hydrocephalus develops secondary to a tumor, there is nothing characteristic about the symptoms to distinguish them from those due to the pressure of the tumor.

Prognosis and Treatment.—When hydrocephalus is due to syphilis, there is some chance that the lesion causing it may be cured by active antisyphilitic treatment and further increase of the hydrocephalus prevented. There is not likely, however, to be much decrease in the size of the head and there can be no relief of the symptoms due to damage already inflicted upon the brain.

When the hydrocephalus is acute in the course of meningitis, repeated punctures of the lateral ventricles should be made, either through the anterior fontanelle or trephine openings. Sometimes, but very rarely, if the child lives, which it rarely does, the development of chronic hydrocephalus may be prevented. There are no drugs, not even mercury and the iodide of potash, so strongly recommended and universally used by our predecessors, which can in any way affect the pathologic process causing hydrocephalus. The vast majority of infants and children fortunately die within a short time as the result of the disease. In rare instances, however, the pathologic process causing the hydrocephalus ceases, or in some unknown way drainage or absorption are reëstablished. If this happens early enough, the child may be normal, except for a large head. In most instances, however, so much damage has already been done to the brain that the child grows up paralyzed, blind, epileptic or feebleminded. Death is far preferable to life under such conditions. It is evident that, if the cause of the obstruction can be removed in the obstructive type or if in this type a new communication can be made between the ventricular system and a normal subarachnoid system, there will be no further increase in the hydrocephalus. It is also evident that if, in the obstructive type, the cerebrospinal fluid can be drained into a vein or some other serous cavity, where it can be absorbed, there will be no further increase in the hydrocephalus. It is, or should be evident, however, that, even if these procedures are successful, the damage already done to the brain remains. All sorts of operations have been devised to accomplish these ends, puncture of the corpus callosum, the introduction of tubes into the foramina, drainage into the subdural space, into the veins of the neck, into the pleura and even into the peritoneal cavity. Most of these operations fortunately prove fatal at the time or soon after. If they are not soon followed by death, they almost invariably cease to accomplish their object, because no method has as yet been devised to prevent nature from healing wounds and filling up abnormal openings. Repeated lumbar punctures and repeated tappings of the ventricles can do no good, of course, as they do not remove the cause of the trouble. It is also foolish to suppose that strapping the outside of the head can do any good. It does not remove the cause or diminish the intracranial pressure. In fact, it increases the pressure on the brain. The injection of counterirritants into the ventricles is also irrational. Another operation, which has been recommended, is the tying of the carotid arteries. This operation does not seem rational. It is not reasonable to suppose that diminishing the blood supply to the brain will affect only the choroid plexus. Furthermore, if the blood supply is diminished enough to diminish the secretion of the choroid plexus, it would seem as if it would diminish the blood supply of the brain as a whole enough to do serious harm. It seems to me, therefore, that the chances of recovery are as

good, if not better, if nothing is done for the treatment of hydrocephalus as when surgical procedures are undertaken.

It is said that thyroid extract diminishes the secretion of the choroid plexus and a number of "cures" of hydrocephalus have been reported from its use. It hardly seems possible to me, however, that the secretion of the choroid plexus can be diminished enough to be of permanent value. This method of treatment is, however, less dangerous than the surgical, because all the harm that it can do it to bring on a condition of hyperthyroidism.

The removal of a portion of the choroid plexus from the lateral ventricles has, of course, the same object, that is, the diminution of the amount of secretion. This operation seems more rational than the others and to offer rather more hope of success. It is open, however, to all of the objections to surgical interference in general, except that nature cannot develop a new choroid plexus.

CEREBRAL PARALYSIS

Under the head of cerebral paralysis are included several groups of cases, dissimilar in origin, but having certain definite clinical features in common. Cerebral paralysis may be present at birth or develop at any time during childhood, most often, however, during the first three years. The paralysis is almost invariably spastic, the only exception being in those cases which are due to arrested cortical development, in which it is flaccid. The paralysis may be hemiplegic, diplegic or paraplegic. It is almost never monoplegic. Contractures of the stronger muscles, that is, of the flexors and adductors, develop in the severer cases. The deep reflexes are increased. The electrical reactions are normal, sensation is normal and there is no muscular atrophy.

Cerebral paralysis is very likely to be accompanied by feeblemindedness, convulsions and various postparalytic manifestations, such as choreiform or athetoid motions or a cataleptic condition. These complications are, of course, due to the same lesions which cause the paralysis.

It is easier to understand the cerebral palsies, if they are divided into three groups, according to the time of the origin of the lesion. This may occur before, during or after birth.

Group I. *Paralysis of Intrauterine Origin.*—In this group the paralysis is due to some congenital defect in the brain. This may be a gross arrest of development—porencephaly—or it may be due to hemorrhage or thrombosis during fetal life, which has resulted in the formation of a cyst or of scar tissue. In rare instances the brain may appear normal in gross, but the development of the cells is imperfect—agenesis corticalis. The etiology of these defects is obscure. They are certainly not due to syphilis. They are somewhat more common in families with a bad nervous history than in others. In some instances they seem to be the result of illness or of blows on the abdomen during pregnancy. The lesion in these cases is almost always a large one. The paralysis is, therefore, usually diplegic or paraplegic. When the process is due to arrested cortical development, it may sometimes be flaccid. Strange as it may seem, the paralysis is often less marked at first in these cases of intrauterine origin than in those due to injury at birth. It is often not noted until the baby is some months old. There is almost always some mental impairment, usually quite marked, in this group of cases.

Group II. *Birth Paralysis.*—This group of cases is the result of intracranial hemorrhage occuring during or soon after birth. The etio-

logy and early symptoms of this condition have already been described. Certain secondary lesions may develop as the result of the hemorrhage. Among these are sclerosis of the brain, partial atrophy and the formation of cysts. The distribution of the paralysis depends on the extent of the original hemorrhage. In these cases it is usually cortical, because, when the hemorrhage is basal and large, the babies do not survive. When cortical, it is usually diffuse and most marked at the vertex, involving the motor areas on both sides. If it does not extend far laterally, both leg areas are involved, resulting in paraplegia, while, if it extends farther down, the arms are involved as well, causing diplegia. The face is not involved in these cases, because, if the hemorrhage is large enough to involve the face centres, the baby dies. In rare instances the original hemorrhage is one-sided and the paralysis hemiplegic. The symptoms of paralysis usually develop early in these cases. Mental impairment is very common in this group, but is not as constant and usually not as marked as in the group of intrauterine origin. Convulsions and postparalytic manifestations are not quite as common as in the first group.

Group III. *Acquired Paralysis.*—The etiology of this group is very varied. It is most often a result of meningococcus meningitis, but sometimes of encephalitis or of acute poliomyeloencephalitis (infantile paralysis). It may be the result of hemorrhage or thrombosis occurring in the course of any acute disease. Occasionally, it is the result of trauma with hemorrhage. In other instances it may be due to hemorrhage resulting from cerebral congestion during paroxysms of whooping-cough or from convulsions. It is probably sometimes due to thrombosis of the sinuses in marantic conditions. It may be, but very seldom is, due to embolism from heart disease. The secondary lesions are the same as at all periods of life, atrophy or sclerosis of the brain, chronic meningitis and the formation of cysts. The original lesion is usually a small one and limited to one hemisphere. The paralysis, is, therefore, usually hemiplegic, but may sometimes be paraplegic, diplegic or monoplegic. The mental condition is usually normal, but is sometimes somewhat impaired. There is, as in all cases in which the upper motor neuron is involved high up, secondary degeneration involving the internal capsule and the lateral tracts.

Symptomatology.—The etiology and pathologic changes explain the symptomatology and physical signs. The varied distribution in the different groups has already been explained. The muscular spasm, which is present in all cases, is due to the removal of inhibition from above. It is usually most marked in the adductors and flexors, especially in the adductors of the thighs. In mild cases it may be found there when there is none elsewhere. It may also be detected there before it has developed anywhere else. The presence of adductor spasm, when there is mental impairment, is strong evidence that the mental impairment is due to a definite cerebral lesion. There is no muscular atrophy, because the nutrition of the muscles is dependent on trophic centers in the spinal cord, whose fibres pass through the spinal motor roots, none of which are involved in the pathologic process. As the result of the removal of inhibition from above, the muscles are constantly working against each other and overstimulating each other. The stronger muscles, that is, the flexors and adductors, overcome the resistance of their antagonists and permanent contractures develop. The deep reflexes are increased, because the reflex arc is intact and there is no inhibition from above. The electrical reactions are normal, because the lower motor neuron and the reflex

arc are intact. Sensation is normal, because the sensory tract is not affected. There may be interference with the growth of the arm and leg, usually more with that of the arm, in the hemiplegic cases.

The position of the extremities in the hemiplegic type is the same as that in the ordinary hemiplegia of adults. In the typical paraplegic cases, as the result of contractures of the flexors and adductors, the thighs are held closely together, the legs partly flexed at the knees and the feet extended. If the children are able to walk, they walk on their toes, cross the legs and bend the body forward. Walking is, however, impossible in the severe cases and very awkward in all. The position of the legs is the same in the diplegic cases as in the paraplegic. The arms are held close to the side, flexed at about a right angle at the elbows, partially flexed at the wrists, with the fingers partly flexed and the thumbs inside.

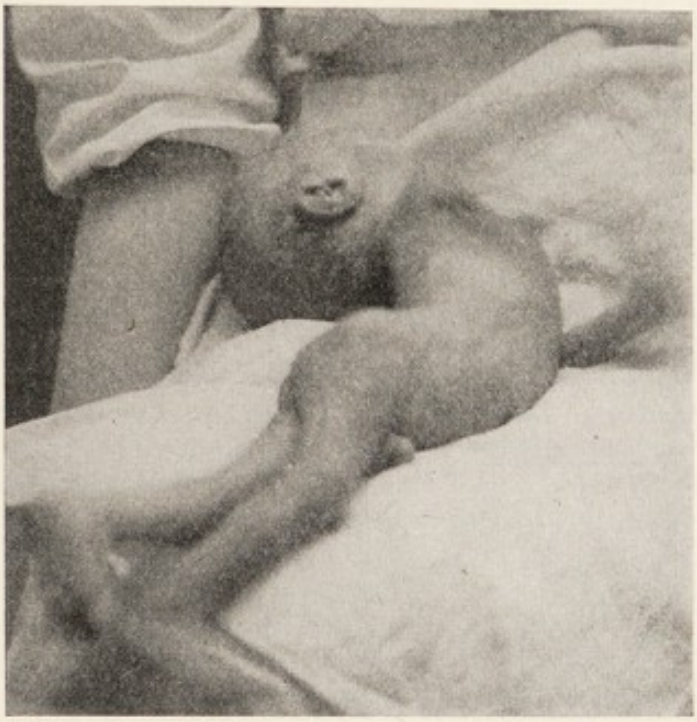

FIG. 161.—Cerebral paralysis.

Mental impairment is usually most marked in the cases in which the paralysis is most marked, but not always. The mental condition may sometimes be normal when the paralysis is very marked and much impaired when there is but little paralysis. It is impossible in early infancy to tell from the extent of the paralysis what the mental condition will eventually be. Convulsions, due, of course, to cortical irritation, occur in about 45% of all cases. They are somewhat more common in the hemiplegic type than in the others. Athetoid and choreiform movements are not very common in any type, but occur more often in the diplegic than in the hemiplegic. They may be either unilateral or bilateral. The choreiform movements are, as the name applies, much like those of chorea. The athetoid movements are slow, twisting movements of the fingers or toes over and about each other, and are always more marked in the fingers. They resemble more the movements of a bunch of angle worms than anything else of which I know.

Diagnosis.—The cerebral palsies of childhood are so characteristic that it is very hard to confuse them with anything else. They may be overlooked, however, when the paralysis is slight and shown only by slight spasm of the adductors, and feeblemindedness marked. Paraplegia, due to a lesion of the cord at birth, may also be mistaken for cerebral paraplegia. If there are no evidences of mental impairment, no convulsions or other signs of cortical irritation, the chances are that the paraplegia is spinal rather than cerebral. If there is mental impairment or there are evidences of cortical irritation, the paraplegic is certainly cerebral in origin. Cerebral paralysis ought not to be confused with the results of infantile paralysis of the spinal type, but sometimes is. In cerebral paralysis the distribution is hemiplegic, diplegic or paraplegic, in infantile paraplysis it is often monoplegic, but it may be of any type. In cerebral paralysis all the muscles of an extremity are involved; in infantile paralysis, groups of muscles. The deep reflexes are increased in cerebral paralysis, diminished or absent in infantile paralysis. Muscular rigidity is present in cerebral paralysis and absent in infantile paralysis. There is no muscular wasting in cerebral paralysis, while it is marked in infantile paralysis. The electrical reactions are normal in cerebral paralysis, while in infantile paralysis the reaction is that of degeneration or, if the muscles have disappeared, there is no reaction. The mental condition is often impaired in cerebral paralysis, while it is normal in infantile paralysis. The contractures are of the stronger of the paralyzed muscles in cerebral paralysis and of the antagonists of the paralyzed muscles in infantile paralysis.

Prognosis.—The outlook is distinctly unfavorable in the diplegic and paraplegic forms. Many die in the first few years of life of malnutrition or intercurrent disease. Many are helpless from contractures. Many are feebleminded. All are more or less incapacitated. The outlook is more favorable in the hemiplegic form, because of its later origin and the less serious damage done to the brain. Children with this type usually live, but are almost always more or less paralyzed. About one half have convulsions. The mental condition is usually normal.

Treatment.—Nothing can be done to prevent the development of the cases of intrauterine origin. Better obstetrics ought to diminish the number of cases due to intracranial hemorrhage at birth. Proper treatment at the time of the hemorrhage may also prevent the development of paralysis in some cases. Better treatment of the diseases in which the acquired group develops ought to diminish materially the number of these cases.

Much can be done by passive motions and proper exercises to prevent the development of muscular contractures. Massage and electricity are useless, because all that they do is to keep up the muscular tone. In cerebral paralysis, however, the muscles are constantly overstimulated and, consequently, do not need to have their tone improved. Much can also be done to prevent contractures by the application of suitable apparatus. Walking may also be made possible, or easier, by the use of a suitable support. The results of the muscular contractures may also be overcome to a considerable extent by proper operations on the tendons, muscles and fascia. By these operations not only is the position of the extremities improved, but reflex irritation is diminished. Operations may also be done on both the motor and sensory nerves. I do not feel competent to discuss or to criticise these operations, but am somewhat skeptical both as to their advisability and usefulness.

CEREBELLAR ATAXIAS

Acute cerebellar ataxia has been described by Griffith (American Journal of Medical Sciences, 1916, CLI, 24) and others. In all probability the majority of these cases are simply examples of acute encephalitis in which the pathologic process happened to be limited to or most marked in the cerebellum.

Cerebellar ataxia is also sometimes present at or develops very soon after birth. I have seen several such cases. The lesion is probably usually some congenital defect in the cerebellum, but may be the result, perhaps, in some instances, of an intracranial birth hemorrhage. It is possible also that the lesion may not be in the cerebellum but in the corpus striatum and the diagnosis a mistaken one. There is, of course, no curative treatment for this condition.

Hereditary, or, better, familial cerebellar ataxia is closely related to the familial spinal ataxias. The first symptoms seldom appear before the age of ten years and often not until childhood is passed. The first symptom is a disturbance in the gait, which is clumsy and rolling. The child walks with its feet wide apart, but can stand with them close together and with its eyes shut, that is, Romberg's symptom is lacking. There are more or less marked incoördinate and choreiform movements of the upper extremities and sometimes of the head and eyes. The deep reflexes are often exaggerated. The control of the sphincters is not affected. Optic neuritis, followed by optic atrophy and blindness, may develop. The mentality slowly but progressively deteriorates. Death eventually occurs after many years. There is no treatment which does any permanent good.

DISEASES DUE TO LESIONS OF THE CORPUS STRIATUM

Crothers (American Journal of Diseases of Children, 1921, XXII, 145) has recently called attention to the various syndromes caused by lesions or diseases of the corpus striatum and summarized the literature on the subject. I quote freely from his article. He describes four fairly well defined syndromes.

1. Double athetosis, as described by Anton and Vogt. In this condition there are choreiform or athetoid movements, increased on intention. There is also often difficulty in speech and swallowing. Associated movements are disturbed, but rigidity is relatively slight. The ocular movements are normal and there is no disturbance of the reflexes. The symptoms are present at birth or develop in early childhood. They increase but little in severity. This condition is consistent with a long and mentally normal life. It sometimes occurs in more than one member of a family.

2. Juvenile paralysis agitans, as described by Hunt. In this condition there is extreme rigidity with tremor. There are neither choreiform nor athetoid movements. The disease is progressive and leads to helplessness and speechlessness, without, however, as a rule, mental impairment. The reflexes, except as masked by rigidity, are normal.

3. Progressive lenticular degeneration with cirrhosis of the liver, as described by Wilson. In this condition there is rigidity, tremor and, occasionally, choreiform or tonic spasms. Disability is progressive and leads to death in a few years. In this condition there is a marked familial tendency.

4. Dystonia lenticularis. In this condition there is a great variety of athetoid movements and marked differences in the tone of various muscle groups which lead to permanent deformities.

In many instances, however, the symptoms are most atypical and do not fit exactly any one of these syndromes. They are also not infrequently associated with symptoms due to lesions of the internal capsule, which is not surprising when the close proximity of the caudate and lenticular nuclei and the internal capsule are borne in mind, as well as the fact that they are all supplied by the same blood vessels. The pathologic lesions in this group of cases may be congenital in origin or the result of birth hemorrhages or of degenerative processes.

Diagnosis.—It is important to keep the cases of this group in mind. Otherwise they may be mistaken for chorea, nervousness, backwardness or cerebral paralyses, in which the lesion is above the internal capsule. Chorea is an acute disease. These conditions are chronic. Careful study will always show in these conditions other evidences of cerebral disease, which are lacking in simple backwardness and speech defects. In these cases the deep reflexes are normal, while they are exaggerated in cerebral paralysis. The backwardness and delay in speaking in these children is, moreover, not due to enfeeblement of the mentality from cerebral changes, as in cerebral paralysis, but to inability to express themselves because of the involvement of the muscles of speech. It is very important, therefore, to distinguish between these conditions, because these children can be educated. They can also be helped to a certain extent by muscle training.

TUMORS OF THE BRAIN

Tumors of the brain are equally common at all ages. The symptoms are also essentially the same at all ages. The same rules for localizing them apply in childhood as in adult life. There are, however, some few differences as regards the frequency of the different types of tumors and the relative severity of individual symptoms in early life and later. These differences are all that it seems worth while to consider.

More than one half of the tumors of the brain in early life are tubercles. These are usually solitary, but may be multiple. They are more likely to be near the surface than are other tumors. The next most common tumor is the glioma, making up perhaps about one eighth. Sarcomata are almost as frequent, while cysts are not far behind. Both sarcomata and carcinomata are likely to be secondary to some primary focus elsewhere, often in the kidneys or suprarenal capsules. Cysts are often the result of intracranial birth hemorrhages, but sometimes there is no evident origin. I have never seen a parasitic cyst. Gummata are extremely rare, making up less than one third of 1% of the total. About 35% of intracranial tumors in early life are located in the cerebellum. Roughly, about 15% are in the pons and another 15% in the centrum ovale, while about 20% are in the neighborhood of the basal ganglia and the corpora quadrigemina. Considerably less than 10% are in the cortex.

Symptomatology.—The rapidity of the development and the severity of the symptoms of cerebral tumor depend, as in adults, very much on the rate of growth of the tumor. A small, rapidly growing tumor may cause much more marked symptoms than a slowly growing tumor of considerable size. The symptoms may also, as in adults, be divided

into two main classes, those dependent on the increased intracranial pressure and those dependent upon the location of the growth. In my experience, vomiting has usually been the earliest and the most constant symptom of increased pressure. The vomiting from tumors is more likely to occur in the early morning than at other times. It is not associated with nausea, has no relation to the diet, is often explosive and is not accompanied by other symptoms of indigestion. It is quite likely to occur in attacks, that is, the child vomits repeatedly for a number of days or weeks and then stops for a time, the vomiting recurring later. Headache has not been as frequent or as severe in my experience, as apparently it is in that of others. It is, of course, very difficult to know whether a baby has a headache or not. Babies show headache by wrinkling the forehead, scowling, rolling or twisting the head and uttering a whining cry. Children, of course, know that they have a headache, but it is rather difficult for young children to localize it. Convulsions have not been as prominent a symptom in my cases as in those of others, although many of the children have had them, usually general, sometimes localized. Vertigo has been a prominent symptom in my cases, probably because in a large proportion of them the tumors have been in the cerebellum. Insomnia from headache is less common in children than later, as is also a slow pulse. An early and very important symptom, or rather physical sign, of intracranial tumor in childhood is choked disc, due, of course, to optic neuritis or papillo-edema. The degree of optic neuritis depends considerably on the rate of the growth of the tumor and may vary materially from time to time It is almost invariably double and is seldom associated with disturbance of the vision. Optic atrophy is very rare. Disturbances of mentality, or rather changes in the disposition, are not uncommon. As I have seen them, children are more likely to be extremely irritable than to be stupid.

The focal manifestations of intracranial tumors in early life are the same as in adults. Ataxia is a very common and prominent symptom of tumors of the cerebellum. I have never been able to get much assistance from Macewen's sign.

Diagnosis.—An abscess of the brain may give exactly the same symptoms as a cerebral tumor. The diagnosis between them is, therefore, often difficult. The chances, however, are very much more in favor of cerebral tumor than of an abscess. If there is an abscess, there is almost always a history of a previous attack of otitis media or of an injury to the skull, or there is a purulent focus somewhere else in the body. An increase in temperature and a polynuclear leucocytosis count somewhat in favor of an abscess. The absence of an elevation of temperature and of leucocytosis does not, however, count against an abscess.

Chronic internal hydrocephalus may give the same general symptoms of increased intracranial pressure. Hydrocephalus, however, usually develops during the first year, while tumors may occur at any time. Hydrocephalus after infancy is most often due to a tumor interfering with the drainage of the ventricles. If not, it usually follows some form of meningitis. The fundi may show choked discs, but optic atrophy is more common. This is very uncommon with cerebral tumor.

Chronic meningococcus meningitis, located at the base of the brain, may also cause symptoms suggesting a tumor. There should be no difficulty in diagnosis, however, because the onset of the meningitis was acute, there is always some elevation of temperature and the findings in the cerebrospinal fluid are materially different.

Ataxia, which often accompanies lesions of the corpus striatum, may be confused with that of cerebellar tumors. The onset is, however, usually slower and the duration longer. It is also likely to be associated with incoördinated movements of the upper extremities and difficulty in speech. In lesions of the corpus striatum, moreover, there are no changes in the optic discs and no other evidences of increased intracranial pressure. It is often very difficult for a time to determine whether convulsions, especially if they are localized, are due to a cerebral tumor or not. On the chances, they are not. If they are due to a cerebral tumor, the optic discs are almost always choked and there are other evidences of an increase in intracranial pressure, which are absent when the convulsions are reflex or epileptic in origin.

Lumbar puncture usually shows an increase in intracranial pressure, when there is a cerebral tumor. It is, however, a very dangerous procedure when there is a cerebral tumor, especially if it is in the cerebellum or at the base of the brain, as there may be a prolapse into the canal with instant death. When there is a possibility of a cerebral tumor, therefore, the fluid should never be allowed to run off rapidly, but only drop by drop. The cerebrospinal fluid in cases of cerebral tumor is normal. Examination of the fluid is, therefore, only of negative value. If lumbar puncture is done, the Wassermann test should always be done on the cerebrospinal fluid, on the bare chance that the tumor may be a gumma.

Prognosis and Treatment.—There is no medicinal treatment for cerebral tumors in early life, except in the occasional instances when the tumor is a gumma. They are universally fatal, unless they can be removed. The chances of a successful removal are, however, extremely slight. As surgery offers a slight chance and as there is no chance without it, an operation should, therefore, be done in every case in which it is possible to locate the position of the tumor. Once in a great while the tumor is where it appears to be and is of such a nature that it can be entirely removed. In most instances, however, the tumor is not found, is of such a nature that it cannot be removed, or the patient dies at the time of, or soon after, the operation. When the child is having severe headaches, repeated convulsions or losing its sight, a decompression should be done for the relief of these symptoms. The duration of life without an operation varies from a few to many months.

ABSCESS OF THE BRAIN

Abscess of the brain is rare in childhood and very rare in infancy. When it occurs, it is almost always secondary to otitis, which is usually chronic. Abscess of the brain is almost never a complication of acute otitis media in early life, because children, instead of developing an abscess, have a purulent meningitis or thrombosis of the lateral sinus and die before there is time for extension to the brain. Abscess of the brain may occasionally be due to trauma and in some instances is a complication of a general septic infection.

Symptomatology and Diagnosis.—There is nothing characteristic about the symptomatology of abscess of the brain in early life. The symptoms may be divided into two main classes, those due to the pressure of the abscess, which acts like any other tumor, and those due to a purulent focus. When there is no absorption from the abscess, that is, when it is "latent," as sometimes happens, it is impossible to differentiate between an abscess and other cerebral tumors, although, if there is a history of otitis media and, perhaps, a slight elevation of temperature

and a slight polynuclear leucocytosis, abscess may be a little the more probable. When there are evidences of septic absorption in connection with the symptoms of a cerebral tumor, especially if there is a discharging ear, a history of trauma or a purulent focus elsewhere, and the cerebrospinal fluid is negative, a probable diagnosis of cerebral abscess is justifiable. If an abscess has broken through and caused a purulent meningitis, as sometimes happens, the symptoms of the abscess are overshadowed by those of the meningitis and the diagnosis is impossible.

Prognosis and Treatment.—The prognosis of cerebral abscess without operation is hopeless. There is a chance of recovery, if it is opened and drained. An operation should always be performed, therefore, in every case in which there is a reasonable probability that there is a cerebral abscess. Cerebral abscesses are usually situated in the temperosphenoidal lobes, next most often in the cerebellum or in the frontal lobes, all of which places are fairly easy of approach.

FEEBLEMINDEDNESS

Under this head are included all degrees of mental deficiency, from the almost complete failure of development of the intellectual faculties in the lowest grade of idiocy to the highest grade of moronity, in which it is often hard to draw the line from normalcy. Feeblemindedness signifies mental deficiency in a brain which is not fully developed, while the term dementia, in contradistinction, is applied to the loss of the mental faculties after they have been normally developed. The classification of feeblemindedness is for me, at least, very difficult, as the subject may be looked at from several points of view. If the present and possible future mental development of the individual alone is taken into consideration, the classification is necessarily very different from one based on the etiology, the appearance of the individual or a combination of the two. I have worked out a double classification for myself, which is, I think, different from that of anyone else and with which, I am sure, no one else will agree. It does not satisfy even me. I have attempted to classify, in the first place, feeblemindedness largely on the basis of the physical appearances but partly on the etiology. Most of the children in the types which can be set apart in this way belong, when the classification is based on the possible degree of mental development among the idiots and imbeciles. Those which cannot be grouped in any special type according to their physical appearances can only be classified according to the degree of their mental deficiency.

Classification on the Basis of Physical Appearances.—The feebleminded, or idiots, may be divided on this basis into two main classes:

A. Those with evident cerebral defects or changes.
   1. Those associated with spastic paralysis.
   2. Hydrocephalic.
   3. Microcephalic.

B. Those without evident cerebral defects or changes—"genetous."
   1. Mongolian.
   2. Amaurotic.
   3. Unclassified—"plain idiots."

Etiology.—The etiology of feeblemindedness associated with spastic paralysis is that of cerebral paralysis and that of feeblemindedness associated with hydrocephalus, that of hydrocephalus. Nothing definite is known as to the etiology of feeblemindedness associated with micro-

cephalus. The primary lesion, however, is unquestionably a defective development of the brain, not of the skull.

There is no doubt that feeblemindedness without evident cerebral defects or changes is more apt to occur in families in which there is a marked neurotic taint, as evidenced by insanity, hysteria, epilepsy or feeblemindedness, than in families which are normal. There is considerable evidence to show that chronic alcoholism in the parents has some influence in the etiology. Drunkenness of the parents, especially of the father, at the time of procreation is especially important. Syphilis in either parent is unquestionably one of the causes. Intermarriage is probably unimportant, except when it exaggerates a neurotic family tendency. Traumatism during pregnancy may possibly play a part. Amaurotic idiocy almost never occurs except in members of the Jewish race. It is evident, therefore, that the race must have something to do with the etiology. What this is, however, is absolutely unknown and difficult to explain, because even among the Jews amaurotic idiocy is a very uncommon condition. It is possible that disturbances of the secretion of the endocrine glands may have something to do with the etiology of feeblemindedness when there are no evident cerebral defects or changes. At present, however, there is no proof that they do and all the evidence which is brought forward is either supposition or theory.

Pathology.—The pathologic changes found in the cases of feeblemindedness associated with evident cerebral defects or changes are those dependent upon the etiology. In those without evident cerebral defects or changes there may be more or less defective development of the convolutions and various microscopic changes which differ somewhat in the different types. These are taken up in the discussion of the various types.

These types of feebleminded children are characteristic enough to be considered separately.

Type Associated with Spastic Paralysis

The physical characteristics of this class are those of the various types of cerebral paralysis. In a general way the degree of feeblemindedness varies directly with the amount of paralysis. There are, however, many exceptions. There may be no mental deficiency when there is marked paralysis and there may be, on the other hand, marked mental deficiency, when there is but little paralysis. Whenever there is mental deficiency, evidences of cerebral paralysis, such as abnormal rigidity of the extremities or adductor spasm in the thighs, should be looked for, because, when such signs are present, they indicate the cause of the feeblemindedness. There is nothing especially characteristic about the feeblemindedness in this class of case.

Hydrocephalic Idiocy

When the head is enlarged, as it is when the hydrocephalus develops before the union of the cranial bones, the appearance is dominated by the hydrocephalus. When the hydrocephalus develops after the union of the cranial bones, there is nothing characteristic about the appearance and this type of feeblemindedness cannot be distinguished from others, unless the hydrocephalus is discovered by lumbar puncture and the appropriate tests. If the children do not die as the result of the hydrocephalus and the process is arrested, the prognosis as to life is good. The mental development is usually slight. Hydrocephalic idiots are, as a rule, pleasant and easy to get on with. They are usually quiet and indifferent,

but are sometimes vain. Their movements are usually slow. The treatment of hydrocephalus is discussed under that disease.

Microcephalic Idiocy

In microcephalic idiocy the head is smaller than normal. It may be of good shape and small in all its dimensions or the deficiency may be most marked in the frontal or occipital regions. Sometimes it is in the parietal regions. The appearance of some of these idiots is most peculiar. The "Aztecs" and the "bird men," which are often exhibited in circuses and dime museums, belong in this class. This type of idiocy is compatible with long life. In general, idiots of this class are fond of moving about and restless. The impressions derived from the senses are usually lively. They are usually good tempered, but have little power of continuous attention. They are late in learning to walk, but have the free use of their limbs. Their movements are usually quick.

Fig. 162.—Microcephalic idiocy.

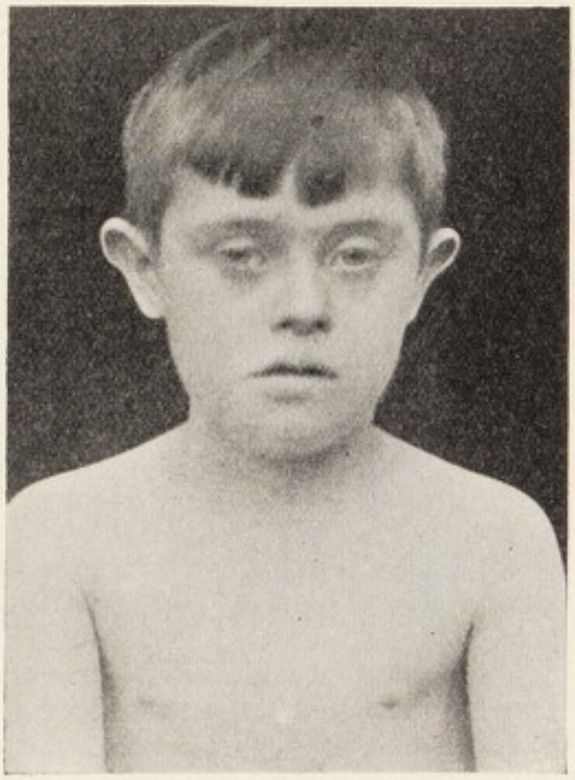

Fig. 163.—Mongolian idiocy.

The small size of the head is due to a gross defect in the development of the brain. The sutures and fontanelles close early because of the lack of pressure from the inside to keep them open. It is evident, therefore, that the removal of portions of the skull, as was done some years ago, cannot possibly do any good, as it does not remedy the cause of the malformation.

Mongolian Idiocy.

The appearance of Mongolian idiots is most characteristic. The head is smaller than normal, but not markedly so. It is obtusely rounded, that is, the anteroposterior and lateral diameters are nearly equal. The features are broad. The bridge of the nose is flat and the space between the eyes wide. The nose is short. The eyes slant downward and inward,

and in most instances there is an epicanthic fold which hides the caruncle, that is to say, the physical appearance is like that of the Mongolian races. Strange to say, however, this form of idiocy is unknown among these races. The tongue is of normal size and shape in infancy but, if the patient lives, usually becomes broadened, thickened and fissured. The mouth is often kept open. The figure is squat. The hands and feet are short and broad. The thumb is short. The little finger is short and markedly curved. There is a single line across the palm instead of two, as normally. There is some delay in the ossification of the bones. The testicles are small. Other congenital malformations and stigmata of degeneration are quite common. There is nothing characteristic about their behaviour.

The vitality of Mongolian idiots is usually subnormal and their resistance to disease much diminished. Other congenital malformations are often present, especially of the heart. Most of them, therefore, die in infancy and early childhood. They almost never reach adult life.

There has been much discussion as to the etiology of Mongolian idiocy. It is certain that it is not due to syphilis. It is also certain that it occurs most often in the first children of very young parents, in the last children of old parents, especially if the mother is near the end of the child bearing period, or when the mother has had a considerable number of children in rapid succession. It is conceivable in this connection that Mongolism may be due either to a defect in the germ plasm or to imperfect nutrition during pregnancy. A considerable number of cases of Mongolism have been reported in one of twins. In all but one of these cases, in which the sex of the twins is mentioned, the sex of the twins was different, showing that they were the result of a two-egg pregnancy. In the few cases in which both twins were Mongols the sex of the twins was the same. In these cases the pregnancy might have been, of course, one-egg or two-egg, but, in view of the fact that in the cases in which only one of the twins was a Mongol, the pregnancies were practically invariably two-egg, it seems likely that they were one-egg. The importance of these observations is that they point strongly to Mongolism being germinal rather than acquired in origin. There is nothing constant in the pathologic findings in the brain in Mongolian idiocy. In many instances the findings are normal. In others the development of a lobe or of certain convolutions has been imperfect or the development of the gray or white matter has been delayed. No constant variation from the normal has been found in the ductless glands, which, in the vast majority of cases, are normal. There is no pathologic basis, therefore, for the belief of certain authorities that Mongolian idiocy is due to an abnormality in the secretion of one or more of the endocrine glands.

Diagnosis.—The only condition with which Mongolian idiocy can reasonably be confused is cretinism. The appearance of the face in the two conditions is superficially somewhat similar, but the eyes do not slant downward and inward in cretinism, there is never an epicanthic fold, the tongue is large and thick from the beginning, the head is large and the extremities markedly shortened. There are, furthermore, the characteristic manifestions of cretinism, especially the scanty hair, the myxoedematous skin and the marked delay in ossification. Mongolian idiocy is sometimes mistaken for adenoids, because of the open mouth and stupid expression. The other evidences of Mongolian idiocy are, of course, lacking when the difficulty in breathing is due simply to adenoids. Mongolian idiots, however, almost always have adenoids.

Treatment.—There is no specific treatment for Mongolism. It seems to me not only foolish, but wicked, to promise parents that Mongolian idiots can be cured by the administration of glandular extracts, as is sometimes done. In a certain number of instances the condition is undoubtedly somewhat improved by the administration of thyroid extract. When this takes place, it is due in part to an associated defect in the function of the thyroid and in part to the stimulating action of the thyroid extract on the metabolism, which occurs in everyone, whether normal or feebleminded. The administration of the extracts of other organs must necessarily be entirely at random, with about as much chance of doing good as a hunter has of hitting a bird, if he goes out in the woods, shuts his eyes, turns around three times and then fires. The treatment must necessarily consist, therefore, of training and education, as in all cases of feeblemindedness.

Amaurotic Family Idiocy

This disease is very uncommon and almost never occurs except in the Jewish race. It often occurs in several children in the same family

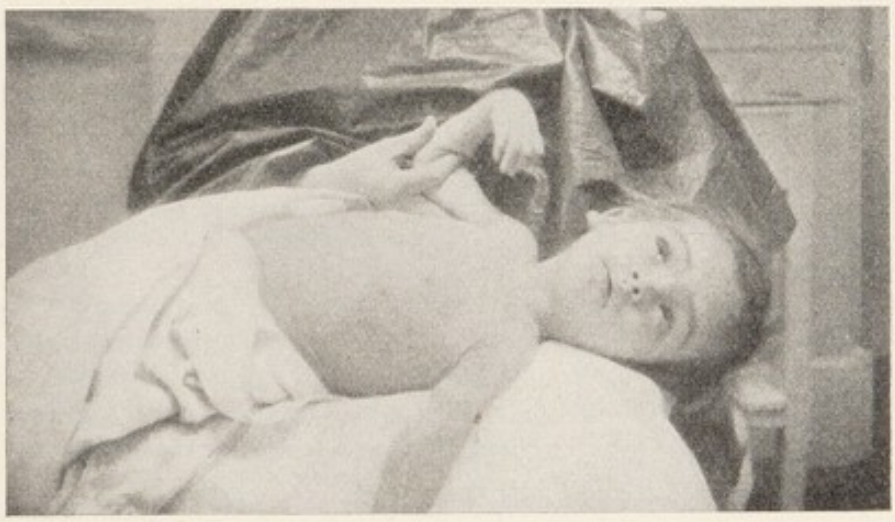

Fig. 164.—Amaurotic family idiocy.

and is equally common in boys and girls. It is universally fatal. Nothing definite is known as to its etiology. Judging from the symptomatology, the central nervous system is normal at birth and develops normally for a number of months, after which degeneration begins in the ganglion cells throughout the entire central nervous system. This degeneration is progressive. It is associated with secondary degeneration of the white fibres of the hemispheres and of the anterior and lateral pyramidal tracts. Degeneration of the ganglion cells of the retina goes on simultaneously with that of those of the central nervous system.

Symptomatology.—These babies are apparently normal in every way at birth and develop normally for from three to six months. At some time between three and six months, however, they cease to develop muscularly and mentally, and soon begin to deteriorate. If the baby held up its head, it ceases to do so. If it had begun to coo and take notice, it gradually stops doing it. It usually, but not always, becomes evident to the parents that the baby is losing its sight. Deterioration progresses quite rapidly, so that in a few months the baby does not see at all and lies perfectly limp. Finally, it becomes so stupid that it does

not know enough to suck and swallow. Up to this time the nutrition is usually very good. If it is not fed with a tube now, it quickly dies of starvation. Death usually occurs between the ages of twelve and sixteen months, but sometimes not until two years. I kept one baby alive by feeding it with a tube until it was three years old. In most instances these babies are limp and lie perfectly motionless for hours at a time. In such cases the reflexes are diminished or absent. In other cases, however, the reflexes may be normal or increased and the babies generally rigid. Convulsions are not uncommon in the cases with rigidity and sometimes occur in the flaccid cases. The expression and position of the flaccid cases is so characteristic that a tentative diagnosis can be made at once from the appearance. A peculiar characteristic is hypersensitiveness to sound.

Diagnosis.—The history and appearance of this condition are so characteristic that a mistake in diagnosis is almost impossible, provided the physician knows about the disease. A positive diagnosis, however, is always possible on the findings in the fundi, which are pathognomonic. There is an oval, gray patch in the region of the macula lutea, where the ganglion cells are thickest and, therefore, the evidences of degeneration are most marked. At the fovea centralis, where the ganglion cells are wanting, the choroid shows through by contrast as a cherry-red spot. To my eyes, however, this spot has more the appearance of iron rust than of a red cherry. There is also atrophy of the optic nerve.

Treatment.—There is no treatment for this disease. It is questionable whether it is advisable to keep these babies alive for a time by feeding them through a tube. It is probably wiser and kinder to let nature take its course.

Juvenile Type of Amaurotic Family Idiocy.—A very similar condition, both clinically and pathologically, sometimes occurs in older children. It is not limited to the Jewish race and runs a much longer and slower course. The symptoms of degeneration in the eyes usually precede those of muscular and mental deterioration. I have never seen a case of this sort.

Unclassified Feeblemindedness

The diagnosis of feeblemindedness, even when it is considerable, if there are no characteristic physical manifestations, is often a difficult one in infancy. It must be made on the failure of the infant to do the things which infants ordinarily do at the proper time. In this connection it is necessary, of course, in order to recognize tardy development, to know how the normal infant develops, that is, when it begins to notice, smile, sit up, hold things in its hands, say simple words and so on. The age at which the mental deficiency can be recognized depends partly on the degree of the deficiency and partly on the carefulness of the observer. Little help can be expected from parents at this time, or even later, as the inability or unwillingness of most parents to perceive or acknowledge that there is anything wrong with their child's mentality is beyond the comprehension of the outsider. In fact, many of them apparently deliberately try to deceive the physician. Some of the early signs of idiocy in infancy are an aimless stare, peculiar inarticulate noises, like those of a young animal, sharp screams, aimless movements of the hands and the failure of the baby to recognize its bottle or its nurse. The presence of stigmata of degeneration is also of some assistance.

Feebleminded children that are able to run about are likely to be very restless. They are constantly running about and feeling of everything that they can lay their hands on. They are also very likely to be awkward in their movements. Many of them try to take hold of things with the hand turned backward. They are also likely to be irritable, and are often cruel to animals and younger children. Most of them are slow about learning to feed themselves and in learning to control the sphincters. Drooling is quite characteristic of the more marked cases. The expression of the face is not normal. It is hard to describe just how it differs from the normal, but with a little experience the difference is not difficult to recognize.

Diagnosis.—Great care must be taken not to mistake blindness, deafmutism and simple backwardness, especially in talking, in young children for feeblemindedness. I have several times known children with congenital cataracts to be called feebleminded, when a single glance at the eyes was sufficient to reveal the cause of the trouble. In my experience it is sometimes very difficult to determine whether a young child is deaf and dumb or feebleminded. Theoretically, it is easy to tell by making sudden noises and the like whether it can hear or not; practically, it is often very hard to decide. Many young children are backward in both their physical and mental development as the result of impaired nutrition or long and severe illnesses. Others are simply slow in their mental development, as are quite likely to be other members of the family. It is advisable, therefore, to be careful about making a diagnosis of feeblemindedness in a young child when the nutrition is poor, in one that has had a serious illness or in one belonging to a family slow of development, unless the evidences of feeblemindedness are unmistakable.

Classification of Feeblemindedness According to the Degree of Possible Mental Development.—In this classification it is assumed, probably correctly, that the mind of each individual is capable of reaching a certain level of development and that it is impossible for it to go above this level, no matter how much time and effort is expended upon it. These levels are fixed by the development of the minds of normal children of given ages. This classification of the feebleminded is as follows:

TABLE XXXIIII

			MENTALITY
Feeblemindedness	A. Idiot	1. Low	Less than one year
		2. Middle	More than one year
		3. High	Two years
	B. Imbecile	4. Low	Three and four years
		5. Middle	Five years
		6. High.	Six and seven years
	C. Moron	7. Low	Eight and nine years
		8. Middle	Ten years
		9. High	Eleven and twelve years
		10. Normal	Above twelve years.

Just as normal individuals vary in their ability to use their minds and in their intellectual attainments according to the opportunities and educational advantages which they have had, so does the knowledge which the members of one of these groups possess depend on the amount of instruction which they have received. A low grade moron, for example, cannot be taught anything more than a nine year old child is able to learn, but it may be either quite ignorant or know far more than the

average child of nine years, according to how much of the knowledge which a child of nine years is able to grasp it has been taught.

It is often very difficult to determine from simple observation whether a child is normal mentally or not, especially if it belongs in one of the higher groups of morons. It is also often hard to determine whether adults are normal mentally or not. As the old Quakeress is reported to have said to her sister, "All the world is queer but thee and me and even thee art a little queer at times." Moreover, children belonging in these classes sometimes show unusual ability in certain directions, most often in music or mathematics, so that their abnormality is overlooked. The best and only safe way to determine the mental status of children is by the use of the Binet-Simon Intelligence Scale or of one of its modifications. This scale is intended, not to determine how much the child knows, but how far its mental development has proceded. The use of these scales is very well explained in Terman's "The Measurement of Intelligence," published by the Houghton Mifflin Company.

It is extremely important to recognize the morons, because they are a menace to the community. Idiots and imbeciles are easily recognizable and, in consequence, are kept from doing harm to themselves and others. Furthermore, their mentality is so low that they are not likely to commit serious crimes. The morons, however, especially if of the higher grades, are not easily recognizable and are, in consequence, treated as normal members of society. Many of them are normal physically and have all the normal physical and sexual desires. They do not, however, have intelligence enough to control their emotions and passions and, in consequence, yield to the impulse of the moment. Even though they may know that what they do is wrong, they do not hesitate to lie, steal, set fires and even commit murder. One of the greatest dangers, however, from the morons is on the sexual side. This danger is greater, moreover, from the female than from the male morons. It is the exception for the female moron to get much beyond puberty without becoming pregnant. She continues to become pregnant as often as the opportunity offers. The morons are to a considerable extent responsible for the continuance and increase of feeblemindedness in the community.

Prognosis.—It is evident, of course, that it is impossible to develop a feebleminded child above a certain level, this level being dependent on the cerebral defect, for which nothing can be done. The best that can be hoped for is to educate it as widely as possible on this level. It is not only idle, but wicked, to encourage the parents of feebleminded children to believe that their children can be cured.

Treatment.—There is no doubt that it is better, not only for the patients but for their families, to have idiots and imbeciles and all but the highest grade morons taken care of in institutions. These institutions may be either public or private, according to the financial status of the parents. It is extremely bad for the other children in a family to have a feebleminded child or adult among them. It sets them a very bad example and interferes materially with their own training and education. Furthermore, the feebleminded children can be far better trained and what mental capacity they have more fully developed in institutions by teachers trained for the purpose than at home. The only exception is in the case of the very rich, who are able to provide special nurses and teachers. In addition, when these children are in institutions, they are under constant supervision and cannot commit crimes against the community. It is more difficult to know what to do with the highest

grade morons. There is no doubt, however, that it would be better both for them and for the community, if they were confined or, at any rate, restrained in some way. There is also no doubt that it would be to the advantage of the community, if all feebleminded persons were, by the authority of the State, rendered sexually impotent.

Diseases of the Meninges

It is conceivable that toxic substances, bacterial in origin or otherwise, in the circulation may cause sufficient irritation of the cerebral meninges to produce symptoms. It is also conceivable that the irritation may be so great as to cause an oversecretion of normal cerebrospinal fluid. If bacteria are also present, inflammation results and the cerebrospinal fluid shows the evidences of inflammation and usually contains the causative microörganisms. It is evident that the border lines between these various pathologic conditions must necessarily be very indefinite and that clinically it may be very difficult to distinguish between them. Nevertheless, they are distinct enough to justify description as separate entities. The term "meningismus," or cerebral intoxication, is applied to the condition in which there are symptoms of meningeal irritation without an increase of fluid and that of "serous meningitis" to the condition in which there is simply an increase in the quantity of the cerebrospinal fluid without any evidences of inflammation in it. Inflammation of the meninges, meningitis, may be caused by any of the pyogenic cocci, the pneumococcus, the influenza bacillus, and occasionally by other organisms, such as the typhoid bacillus, as well as by the meningococcus and the tubercle bacillus, which are, of course, the organisms usually found.

MENINGISMUS AND SEROUS MENINGITIS

In the first place it is to be remembered that any or all of the symptoms of true meningitis may develop in these conditions. The physical signs may also be the same for a time, although they are usually less marked than the symptoms. In infancy meningismus and serous meningitis occur most often in connection with diseases of the digestive tract. In older children pneumonia is the most common cause. They often develop in connection with diseases of the middle ear, especially in infancy and, in fact, may appear at the onset or come on during the course of any acute disease associated with a high temperature and the production of toxic substances.

Diagnosis.—These conditions are always associated with some other disease; meningitis is a disease sui generis. If they come on at the onset of some other disease, the symptoms of that disease may be temporarily lacking or easily overlooked, but soon become evident. If they come on during the course of some other disease, the symptoms of that disease are, of course, already present. The presence of the symptoms of some other disease, therefore, points strongly to these conditions and against meningitis. In an infant the absence of bulging of the fontanelle is strongly in favor of meningismus, although occasionally it may not bulge in meningitis. Bulging of the fontanelle points towards true meningitis, but also occurs in serous meningitis. The symptoms of cerebral irritation in meningitis and serous meningitis are, as a rule, general, and focal manifestations are unusual. In meningitis, however, focal manifestations are very common and are often present when the symptoms of general cerebral irritation are not at all marked. The presence of focal manifes-

tations suggests very strongly, therefore, that the condition is a more serious one than meningismus or serous meningitis.

If there is any doubt, however, as to the diagnosis between one of these conditions and meningitis, lumbar puncture should be done. In meningismus the cerebrospinal fluid is normal in every way and runs out slowly. In serous meningitis the fluid runs out faster than normal, showing that the intracranial pressure is increased, but is itself normal. It may, however, show a slight increase in the number of cells, the cells all being mononuclear, without differing in any other way from the normal. This is contrary to the usual teaching, but I have seen a slight excess of mononuclear cells so often in cases of this sort, which never showed any other signs of meningitis, encephalitis or infantile paralysis and which recovered completely, that I am confident that a small increase of mononuclear cells is not at all uncommon in serous meningitis. Such an increase is, moreover, not uncommon in connection with the serous meningitis which often accompanies otitis media.

Prognosis and Treatment.—The prognosis of these conditions is good, except in so far as they alter that of the etiologic disease by increasing the strain. Lumbar puncture and the withdrawal of fluid often diminishes or stops the symptoms in serous meningitis. Otherwise the treatment is purely systemic; ice-cap to the head, baths, sedatives.

MENINGITIS DUE TO PYOGENIC ORGANISMS

Meningitis may be caused by any of the pyogenic organisms. It is much more often due to streptococci than to other members of the group. When it is not due to infection from trauma, it is most often secondary to otitis media and its complications. It may develop, however, in the course of any septic infection. It is probable that lumbar puncture in these infections predisposes to the development of meningitis. There is nothing characteristic about the symptoms and physical signs of meningitis due to the pyogenic organisms, except that they develop in the course of diseases caused by these organisms. The spinal fluid is under pressure, turbid or purulent, shows positive tests for globulin and either forms a fibrin clot or deposits a sediment of pus. The number of cells is much increased, the cells being all or nearly all polynuclear neutrophiles. Numerous organisms are present.

The prognosis is almost invariably bad, although occasionally a mild case, secondary to otitis media, recovers. The treatment consists of the relief or the removal of the causative condition and of meeting the symptoms as they arise. Vaccines and antisera are of no value. Repeated lumbar punctures are of little help. It is possible that cistern drainage in the hands of an expert may do some good.

PNEUMOCOCCUS MENINGITIS

Pneumococcus meningitis almost always develops towards the end of or during convalescence from lobar pneumonia. It may, however, develop in the course of any disease due to the pneumococcus in which there is infection of the blood stream. In rare instances it may be the first and only manifestation of pneumococcus infection. All types of pneumococcus may cause it. Pathologically it is characterized by a greater exudation of fibrin and pus than is usual in the other forms. The exudate is greatest over the convexity of the brain and anteriorly. The meninges of the cord are usually not involved.

Symptomatology and Diagnosis.—The symptoms are sometimes latent or disguised by those of the primary infection. In general, however, the onset is acute and stormy, like that in severe meningococcus meningitis. In fact, the two diseases are indistinguishable on their symptomatology except that, when the symptoms develop in the course of pneumonia or of some other pneumococcic infection, the chances are that it is pneumococcus meningitis. When symptoms of meningeal irritation develop at the onset or during the first two or three days of pneumonia, the chances are that they are signs of meningismus or serous meningitis rather than of pneumococcus meningitis.

The only way in which the diagnosis can be made is by lumbar puncture. The cerebrospinal fluid is usually under increased pressure. It is purulent, contains a large number of polynuclear neutrophiles and many pneumococci. It, of course, gives a marked test for globulin and either forms a fibrin clot or deposits a sediment of pus.

Prognosis and Treatment.—Pneumococcus meningitis is almost always fatal within three or four days. It is said that occasionally the course may be prolonged and that recovery may occur. I have never seen a child live more than a few days and have never known one to recover. The prognosis is, therefore, practically hopeless and it is not justifiable to hold out any hope of recovery.

The treatment is entirely symptomatic. Antipneumococcic serum, even when the organism is of Type I, whether given intravenously or intraspinally, does no good. Vaccines are useless.

INFLUENZA MENINGITIS

Influenza meningitis is more common than pneumococcus meningitis and is more common in infants than in children. It may apparently sometimes be primary, but is almost always secondary to an influenzal infection elsewhere, most often in the nasopharynx and its adnexa. The pathologic process is essentially the same as in pneumococcus meningitis.

Symptomatology.—The onset is acute and resembles very closely that of meningococcus meningitis. There is nothing about the symptomatology to distinguish it from other forms of acute meningitis. The cerebrospinal fluid is purulent and contains a large number of polynuclear neutrophiles and influenza bacilli. It, of course, gives a marked test for globulin and either forms a fibrin clot or deposits a sediment of pus.

The course is usually short and violent, seldom more than a few days, but in some instances may be one or two weeks. It is said that recovery sometimes takes place, but I have never known a case to recover.

Treatment.—Wollstein in 1911 produced an antiserum in goats which protected monkeys against infection. It was used in but a few cases in humans with not very satisfactory results. It has not been procurable for many years. The treatment is, therefore, purely symptomatic.

MENINGOCOCCUS MENINGITIS

Meningococcus meningitis, commonly known as cerebrospinal or epidemic meningitis, is caused by the diplococcus intracellularis of Weichselbaum, which is usually spoken of as the meningococcus. There are many different strains of this organism. An antiserum to one strain has no effect on others. It is almost always found in pairs or tetrads and may be either inside or outside the leucocytes. It is stained by all the

ordinary stains and is decolorized by Gram's method. It can be cultivated, but nothing is known as to its habitat outside the human body.

The meningococcus is present in the nasopharynx of persons ill with the disease, of many of those who are associated with them and of a certain number of healthy persons who have had no known exposure to the disease. Infection presumably occurs through the nasopharynx and nose. It was formerly supposed that infection of the meninges occurred directly through the lymphatics. Observations made during the World War apparently show, however, that in many, if not in all, cases, at any rate during epidemics, infection occurs through the blood. It is presumable that the meningococcus leaves the body through the nasopharynx.

Sporadic cases of meningococcus meningitis are liable to occur at any time in any community and epidemics occur from time to time, usually in the winter and spring, dying out in the summer. The crowding together of many young people and poor ventilation predispose to the development of epidemics. Except during epidemics, the disease must, of course, be spread by carriers. It is probable that it is spread chiefly by carriers, even during epidemics, because the disease almost never spreads in hospitals, even when no special precautions are taken to prevent it. I have seen it treated for many years in the open wards without any special precautions and have never known a case to develop in the hospital. Nothing is positively known as to the period of incubation. It apparently may be as short as one or two days or, as is the case in diphtheria and infantile paralysis in which the organisms may live in the nasopharynx without causing symptoms, indefinitely long, infection not taking place until the resistance of the individual is weakened or the virulence of the microörganism increased in some way. About one half of the cases of meningococcus meningitis occurs in children under five years of age. It may occur in very young infants.

Pathology.—The earliest change is an intense hyperemia of the meninges, which is soon followed by an exudation of leucocytes. As the disease progresses, there is a fibrino-purulent exudation over the surface of the brain. It is usually widely distributed, but is ordinarily most abundant over the anterior portion of the brain and at the base. In some instances it is limited to the base. The brain substance may appear normal or be injected. Microscopically, minute capillary hemorrhages are common. Degenerative changes are usually present in the nerve cells and sometimes there are small abscesses. The spinal meninges show similar changes. The exudation is chiefly on the posterior surface of the cord and may be either general or limited. The inflammation not infrequently extends along the cranial nerves, especially the optic and auditory.

The cerebrospinal fluid is increased in amount, often causing dilatation of the ventricles. It is turbid or purulent. It sometimes forms a fibrin clot, but more often deposits a sediment of pus. It gives all the tests for globulin. The test for glucose is usually diminished or absent. The number of cells is very much increased, the cells being almost entirely polynuclear neutrophiles. Meningococci are present both within and without the leucocytes.

When the course of the disease is at all prolonged, blocking of the foramina of Luschka and of the foramen of Magendie is likely to occur as the result of the accumulation of fibrin and cause an acute internal hydrocephalus. In the chronic cases and in many of those which recover

cicatricial changes are likely to take place at the base of the brain and cause either an obstructive or communicating hydrocephalus or both. In other cases which recover cicatricial changes are likely to develop which cause various forms of spastic paralysis and feeblemindedness. Blindness and deafness also not infrequently occur as the result of the changes in the optic and auditory nerves.

Acute degeneration of the parenchymatous organs occurs as in other acute infectious diseases. They are usually most marked in the liver and kidneys. In rare instances there may be an associated infection of the lungs, pericardium, endocardium or joints by the meningococcus. In general, however, complications in other organs are due to associated organisms.

Symptomatology.—A number of epidemics of cerebrospinal meningitis occurred in the various army camps during the World War, where the patients could be kept under very close observation. The conclusions were reached by the officers in charge that there is a stage of systemic infection lasting from a few hours to several days, with an average of about forty-eight hours, before the meninges are attacked and that this stage of meningococcus sepsis can be recognized in about 50% of the cases. They also concluded that meningococcus sepsis, in which the meninges were never attacked, was not uncommon. In their experience, (Herrick, Journal American Medical Association, 1918, LXXI, 612) meningococcus sepsis is usually characterized by a prodromal period of malaise and languor, often associated with evidences of infection of the upper respiratory tract. The patient either rapidly or gradually becomes ill, weakness increases and is accompanied by apathy. Frontal headache is usually present, but may be absent. The tongue is coated and the oral secretions are sticky and viscid. The most characteristic symptom is a dull apathy. The patient is conscious when roused, but promptly lapses into silence and immobility. All voluntary physical and mental activity is lacking. The patient lies on his side with the head bent forward and the knees drawn up. A petechial rash is present in about 50% of the cases. The deep reflexes are very irregular. Specific signs of meningeal irritation are often lacking. The spinal fluid may or may not be under increased pressure and may or may not show a trace of globulin. It does not contain an excess of cells.

This stage of systemic infection has certainly not been generally recognized in the past in either sporadic or epidemic cases in early life. It may be because it is not as definite or because it has not been looked for or noted. I have seen only a few sporadic cases in childhood since the war and have not been able to form an opinion. It is very important, however, for everyone to bear this possibility of a systemic stage of infection with fairly definite symptoms in mind, because in very many cases infection of the meninges can be prevented by the use of the antiserum intravenously in the systemic stage. It will be a great step in advance, if it can be determined whether this stage occurs in sporadic cases and its symptomatology established.

The symptoms of cerebrospinal meningitis, after invasion of the nervous system has occurred, are, in the first place, those common to all acute febrile diseases, to which are added those due to the involvement of the meninges and deeper structures. The onset is almost always sudden, sometimes extremely so. The temperature is always elevated, usually running between 103° F. and 104° F. In many instances it is irregular. When recovery occurs, it falls by lysis. Headache is probably

a very constant symptom, especially at the onset, although infants and young children do not, of course, complain of it. Pain in the neck is also common and there is also often pain in the extremities. Vomiting and convulsions are common at the onset, but unusual after the first forty-eight hours. The mental condition is almost always affected. Apathy, going on to coma, is more common than excitability or delirium.

Rigidity of the neck is almost always present. Retraction of the head is common and opisthotonos the rule in the severe and chronic cases. There is almost invariably more or less bulging of the anterior fontanelle, when it is open. Strabismus is uncommon. The pupils may be either contracted or dilated and are often unequal. Other evidences of involvement of the cranial nerves are uncommon, except that deafness and blindness may develop and finally disturbances of the rate of the pulse and respiration. Tonic spasm of the extremities is very common; paralysis is rare and, if present, usually only temporary. Tremor is not uncommon. There is nothing constant about the behavior of the abdominal and cremasteric reflexes. The knee-jerks are ordinarily increased, but

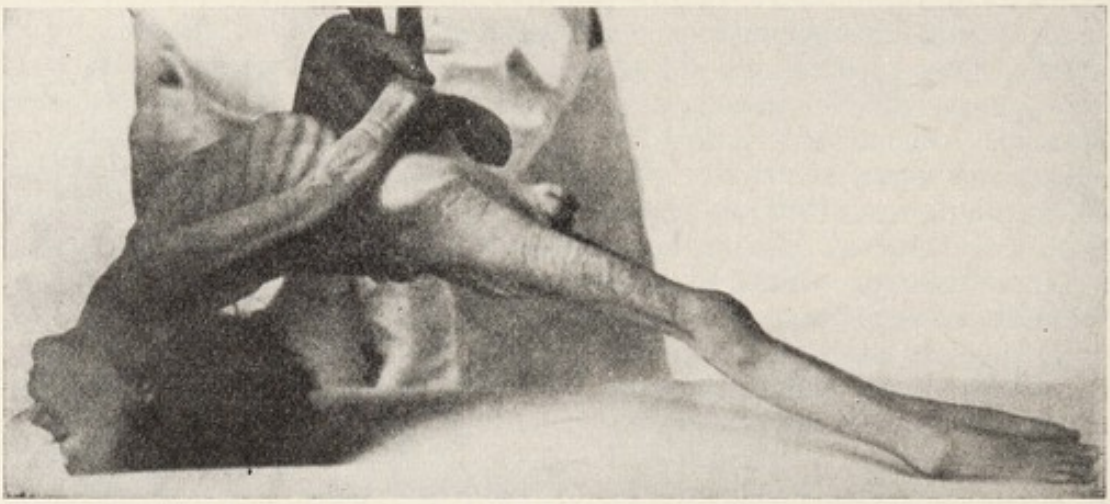

FIG. 165.—Opisthotonos in cerebrospinal meningitis.

may be normal, diminished or absent. Kernig's sign is usually present, but may be absent. Brudzinski's neck sign, the contralateral reflex and Babinski's and Oppenheim's phenomena are inconstant. Hyperesthesia is common. The tâches cérébrales are also common, but are of no importance in diagnosis. Eruptions, whether petechial or macular, are in my experience usually conspicuous by their absence. Herpes labialis is not uncommon.

There is always a polynuclear leucocytosis, which is usually marked, counts of 30,000, 40,000 or even higher not being uncommon. The urine shows the usual characteristics of acute degenerative nephritis.

The cerebrospinal fluid has already been described. When recovery takes place without the use of serum, the extracellular cocci first disappear and then the intracellular. The number of cells diminishes and the formula gradually changes from polynuclear to mononuclear. The fluid becomes less and less turbid and finally clears, while the globulin diminishes in amount and eventually disappears.

Before the days of the serum treatment, when the disease took its own course, five different types were easily distinguishable.

1. The malignant or hyperacute.
2. The ordinary.

3. The mild or abortive.
4. The intermittent.
5. The chronic.

In the *malignant type* the onset is extremely acute, usually with severe headache, high fever and convulsions. Coma quickly develops and death occurs in most instances before the signs of meningeal irritation, such as rigidity and retraction of the neck, Kernig's sign and spasm of the extremities, have become marked. Eruptions are more common in this type than in the others. The duration is from twenty-four hours to forty-eight hours.

In the *ordinary type*, which may, of course, vary in severity, the onset is less acute and the development of the symptoms less rapid. Rigidity, retraction and spasm are more marked. The course varies from one to three weeks and may end in either death or recovery. The children who recover are likely to be left blind, deaf, paralyzed or feebleminded.

In the *abortive form* the onset and symptoms are all mild and of short duration. The onset is sometimes quite stormy, however, but improvement begins almost at once. The course of the disease is seldom more than a week.

In the *intermittent type* the course is that of the ordinary type, but after a marked remission of the symptoms they all recur again, to remit and recur again, perhaps, several times. In such cases death almost invariably finally occurs.

In the *chronic type* the disease may run on, with or without remissions, for many weeks or even months. I have seen a baby survive for eight months, being kept alive by gavage. Marked rigidity and opisthotonos are the rule in these cases. The patients are usually stupid or comatose, and emaciation becomes extreme. It is this type which has been described in the past as *chronic basilar meningitis*. Death almost invariably finally occurs. It is unfortunate if it does not, because, if recovery occurs, so much damage has been done that the child is hopelessly incapacitated both physically and mentally.

Complications and Sequelæ.—These are, in most instances, due to the direct involvement of the nerves or brain during the course of the disease or to internal hydrocephalus, which may be the result of blocking of the foramina leading from the fourth ventricle by the exudation or of the obliteration of these foramina or of the cerebral subarachnoid space by adhesions or the contraction of new-formed connective tissue. Most of them have already been mentioned. In rare instances there may be a localization of meningococci in other organs, especially the lungs and joints. In general, however, complications in the lungs and other organs are caused by secondary invaders.

Prognosis.—The mortality from cerebrospinal meningitis in infancy before the serum treatment was discovered was between 85 and 100%, while the mortality in childhood was about 70%. The general mortality with the serum treatment is between 25 and 30%. The earlier the serum is used the better is the prognosis. It is of little value in prolonged and chronic cases. The serum not only improves the chances of recovery but also shortens the course of the disease and prevents the development of complications. Before the introduction of the serum treatment a large proportion of the children who recovered from the disease were left blind, deaf, paralyzed or feebleminded. With the serum treatment they either die or, with rare exceptions, recover entirely.

Diagnosis.—The diagnosis of cerebrospinal meningitis in the stage of systemic infection, except during epidemics and in camps or institutions, is extremely difficult. It must be made on the symptomatology described in the discussion of this stage. The onset of the stage of invasion of the central nervous system is often not very different from that of many of the other acute infections, such as scarlet fever, pneumonia and tonsilitis, especially if it is associated with meningismus or serous meningitis. In other diseases other evidences of these diseases soon appear, however, and in cerebrospinal meningitis other evidences of this disease. For a time, however, the diagnosis can often be made only by lumbar puncture. This should be done when there is a reasonable probability that the trouble is meningitis. It must not be forgotten, however, that lumbar puncture favors the invasion of the nervous system. It is impossible to distinguish between meningococcus meningitis and pneumococcus meningitis, influenza meningitis or meningitis due to the pyogenic cocci either on the symptomatology or physical signs. Meningococcus meningitis is, however, more often a primary disease, while the others usually develop in the course of other diseases. The diagnosis must be made by lumbar puncture. The characteristics of the spinal fluid are the same in all, except as regards the bacteria which they contain. If the fluid is purulent and no organisms are found, the disease is almost always meningococcus meningitis. It is also sometimes very difficult to distinguish between meningococcus meningitis and tuberculous meningitis without a lumbar puncture. The onset is, of course, usually more acute in meningococcus than in tuberculous meningitis, but may be quite acute in tuberculous meningitis, especially in infancy, and sometimes quite slow in meningococcus meningitis. The fontanelle almost invariably bulges in meningococcus meningitis, but is not infrequently level, and sometimes depressed, in tuberculous meningitis. Strabismus and other evidences of involvement of the cranial nerves are more common in tuberculous than in meningococcus meningitis. There is always a polynuclear leucocytosis in meningococcus meningitis; in tuberculous meningitis there usually is not, but may be. The tuberculin test is of little value. It is likely to be absent in tuberculous meningitis, because tuberculous meningitis is a form of acute miliary tuberculosis, and it is often present in meningococcus meningitis, if there is a tuberculous focus elsewhere. The presence of a tuberculous focus, especially if it has been recently operated upon, is strong evidence in favor of tuberculous meningitis. Lumbar puncture, however, is often the only way to settle the diagnosis and should always be done. The cerebrospinal fluid is usually under increased pressure in both conditions. It is clear or slightly opalescent in tuberculous meningitis, turbid or purulent in meningococcus meningitis. A fibrin clot is formed in tuberculous meningitis, a fibrin clot or a deposit of pus in meningococcus meningitis. The number of cells is usually under two hundred in tuberculous meningitis and these cells are usually almost entirely small mononuclear, while in meningococcus meningitis the number of cells is usually much greater and almost all of them are polynuclear. Meningococci are present in meningococcus meningitis and tubercle bacilli in tuberculous meningitis, although it is often difficult to find them.

Treatment.—There is no treatment which has much influence on the course of meningococcus meningitis except that with the antiserum, although the repeated withdrawal of fluid by lumbar puncture, even without the injection of serum, sometimes seems to do good. Hexamethylenamin is useless. It has no bactericidal action unless it is

broken up. It is not broken up except in an acid medium. The cerebrospinal fluid is alkaline. The serum is bactericidal in action. It limits the multiplication and causes the rapid disintegration of the organisms. Phagocytosis is also stimulated and the organisms are rapidly taken up and digested. It is for this reason that there are no toxic symptoms from the rapid destruction of the organisms. Since the action of the serum is directly on the organisms themselves, it is very important to have it reach them in as concentrated a form as possible. It is best, therefore, to introduce it directly into the spinal canal by lumbar puncture. It is so much diluted when given subcutaneously that it is of no practical value. There is no doubt that when given intravenously it passes through the choroid plexus and into the ventricles. Even so, however, it is much more diluted than when given intraspinally. It does, however, reach the organisms which are high up, which may not be reached from below. It is possible, therefore, that when given intravenously it may tend to prevent the development of internal hydrocephalus. It may be wise, therefore, in severe cases to give the serum both intraspinally and intravenously, remembering, however, that the intravenous injections are simply adjuncts to the intraspinal. They should not be given at the same time, but some hours apart.

There are many different strains of meningococci. The antiserum is bactericidal only to those strains from which it was prepared. If the meningitis in a given case is caused by some strain which was not used in the preparation of the antiserum, the antiserum cannot do any good. It is for this reason that no benefit is derived from its use in certain cases.

As much cerebrospinal fluid as possible should be allowed to run out, provided in the meantime no symptoms develop from the change in intracranial pressure, in order that the serum may reach the organisms in a concentrated form. Almost, but not quite, as much serum should be introduced as cerebrospinal fluid has run off. In babies the introduction should be stopped, however, before the fontanelle bulges. The serum must be warm and should be introduced slowly. It is hardly necessary, as has been advised, to control the introduction by simultaneous observation of the blood pressure. It may be introduced either by gravity or with a syringe. The gravity method is perhaps a little safer, but there is no danger in using a syringe, if the serum is introduced slowly and the condition of the patient is carefully watched. If disturbances of the pulse and respiration or twitchings develop, they are easily relieved by letting a little of the serum run out. In the beginning the serum should be given once in twelve hours. In some of the severest cases it may be advisable to give it once in eight hours. After the first two or three times, however, the injections should not be given oftener than once in twenty-four hours. It is often very difficult to decide just when to stop giving the serum. Improvement is shown, of course, by improvement in the general condition and a fall in the temperature. It is shown best, however, by the changes in the cerebrospinal fluid, which follow the same course, but much more quickly, that they did in the past in the untreated cases which recovered. It is certain that the injections should be continued as long as organisms are visible in the spinal fluid. It is probably advisable to stop the injections, if the organisms are not found on two consecutive examinations.

It must be remembered that the antiserum itself acts as an irritant to the meninges, so that the fluid will continue to be turbid and to

contain a large number of polynuclear cells even after the meningococci have been destroyed. The presence of a turbid fluid with polynuclear cells is, therefore, not an indication for the continued administration of the serum. The persistence of rigidity of the neck and Kernig's sign is also not an indication for the continued use of the serum, as they may persist long into convalescence.

The dose of serum intravenously is from 15 to 60 c.cm., according to the age of the child. This may also be repeated at intervals of twelve hours in the beginning and of twenty-four hours later. It is advisable to give a desensitizing dose of one c.cm. of the serum subcutaneously before using it intravenously. It is also safer to do this, even when it is used intraspinally, although accidents from the intraspinal use are most uncommon.

If, after the spinal fluid has been running out freely, only a few c.cm. can be obtained by lumbar puncture, there is obstruction above. It is useless under these conditions to continue the intraspinal injections, because the serum cannot be forced above the obstruction and, therefore, can do no good. In such instances the intravenous use of the serum should be pushed. In babies the serum can easily be introduced into the ventricles through the anterior fontanelle and should be given in this way. In older children it may be introduced through trephine openings. The results of the introduction of the serum in this way, while occasionally apparently quite brilliant, are, however, usually most disappointing.

If the temperature continues elevated or if there is a recurrence of the fever and of the symptoms after the organisms have disappeared and the injections have been stopped, another lumbar puncture should be done. If the cerebrospinal fluid again contains meningococci, the injections of serum should be resumed, the same amounts and intervals being used as before. If the fluid still contains many polynuclear cells, but no organisms, it is doubtful whether further injections will do any good. It is advisable, however, to repeat them.

If the meningococci do not diminish in numbers and disappear in the course of the injections, it means that the serum is inactive against the special strain causing that case. If possible, another serum should be procured and used.

The vaccine treatment has been recommended by certain authorities in the treatment of the chronic resistant cases, an autogenous vaccine being, of course, preferable. I never saw vaccines do any good at the time that they were used in the treatment of this disease before the serum was available. I have had no experience with their use since the introduction of the serum, but am very skeptical as to whether they do any good. The treatment of meningococcus meningitis, outside of the use of the serum, is entirely symptomatic. An ice-bag to the head makes older children more comfortable, but usually only bothers young children. The bromides and opium must be used, when necessary, to control restlessness and pain. Water must be forced and food given through a tube, if it is not taken otherwise. Stimulants should be used as necessary, but strychnia is contraindicated, because of its stimulant action on the nervous system.

Although the contagiousness of meningococcus meningitis under civil conditions is very slight, it is probably advisable to isolate children ill with this disease. As the organism leaves the body in the secretions of the nasopharynx, everything which can be contaminated by these secre-

tions should be thoroughly disinfected or destroyed. It would theoretically be advisable to continue the isolation until meningococci were no longer present in the secretions of the nasopharynx. Practically, however, this is hardly necessary and the isolation may be stopped when the patient is well. As a matter of fact, healthy carriers for some unknown reason seem to be more dangerous than children who have had the disease. After the disease is over, the room should be thoroughly cleaned and aired. Sunlight destroys meningococci very quickly. When the disease is epidemic in camps or in institutions, the carriers should be searched for by routine cultures from the nasopharynx and quarantined until they are clear. Such a procedure, however, seems neither necessary nor advisable under civil conditions and when the cases are sporadic.

TUBERCULOUS MENINGITIS

Tuberculous meningitis occurs throughout infancy and childhood, but is much more common in infancy. It is most common in the late winter and early spring, presumably because respiratory infections at this time light up latent tuberculous foci in the tracheobronchial lymph nodes or lungs. It often follows measles or whooping-cough for the same reason. It is always secondary to tuberculosis elsewhere in the body. In infants the tuberculous focus is usually a chronic diffuse tuberculosis or in the lungs; in children, it is usually in the bones, joints or lymph nodes. Tuberculous meningitis is very likely to follow operations on tuberculous lesions. The infection is in the vast majority of cases caused by the human bacillus, the proportion of bovine infection increasing somewhat with age. The much greater frequency of human infection in infancy is presumably due to the greater exposure which infants have to tuberculous adults than do children. In a very large proportion of cases, at least 70 %, a history of exposure to adults with open pulmonary tuberculosis can be obtained, if sufficient care is taken in getting the story. Tuberculous meningitis is equally common in boys and girls. It is responsible for a very considerable proportion of the deaths from tuberculosis in the first few years of life.

Pathology.—The characteristic lesion is the miliary tubercle. These develop in the pia mater. They are most numerous at the base, but may be scattered all over the convexity of the brain, especially along the fissure of Sylvius. They may also develop in the pia mater of the upper portion of the spinal cord and sometimes in the ocular choroid. Associated with the miliary tubercles is an exudation of fibrin and pus. This is most marked at the base. It is not as profuse as in the other types of meningitis. There is an excess of fluid in the ventricles. This fluid is usually clear, but may be turbid or flocculent. Adhesions may form at the base, but they are almost never sufficient to cause an obstructive hydrocephalus. The cortex may be injected and slightly softened.

Symptomatology.—The symptomatology of tuberculous meningitis is so varied that it is impossible to describe it accurately. This is especially true of the early symptoms. The only physician who has not misinterpreted the early symptoms of tuberculous meningitis is the one who has never had any cases of it.

The onset is usually slow, but may be very sudden. In most of the cases in which it is apparently abrupt careful questioning discloses, however, that the child has not appeared quite like itself for several days, or even a couple of weeks, before it was appreciated that it was

really sick. Among the earliest signs are a change in disposition and vomiting. The children become irritable, fussy, sleep poorly and act entirely unlike themselves. Sometimes they are drowsy and uninterested. The vomiting is independent of the ingestion of food and is not associated with other evidences of indigestion, except sometimes constipation. Older children often complain of headache. Young children rarely complain of anything, however, as children of this age rarely do complain unless they are quite uncomfortable. This is one of the reasons, probably, why the onset often seems sudden.

In some instances the first marked symptom is a convulsion. More often the children show other signs of meningeal irritation, such as twitching, rigidity, photophobia and screaming. The cry is quite characteristic, being sudden, shrill and intermittent. These symptoms are usually associated with increasing drowsiness, but in some instances the children are very restless and delirious. In other instances the symptoms of meningeal irritation are very slight and the first marked symptom is drowsiness, rapidly changing to coma. Drowsiness and coma may alternate with periods of excitability. As a rule, however, the patient becomes progressively more stupid and finally lies perfectly quiet with its eyes widely open, staring, but seeing nothing and knowing nothing. It goes without saying that at this time the child is unable to take food and is incontinent of urine and feces. In some instances, however, there is retention of urine and usually marked constipation. At this time there may or may not be rigidity. Convulsions may occur at any time, but are more common early and late in the course of the disease. Marked remissions in the symptoms may occur. I have, for example, known a child who had been comatose for a week to sit up in bed one morning, know everyone and ask for and eat its breakfast. It died the same afternoon. Such remissions are, however, more common in the text-books than in real life and seldom occur. The progress of the symptoms is almost invariably steadily from bad to worse.

There is nothing characteristic about the temperature. It is usually moderately elevated and irregular, but at times may be high and at other times temporarily normal. There is almost invariably a terminal rise in the temperature during the last two or three days. It not infrequently reaches 107° or 108° just before the end.

The pulse usually varies with the temperature. It is much more often increased than diminished in frequency. As a matter of fact an infrequent pulse instead of being the rule in tuberculous meningitis, as is often supposed, is really quite uncommon. The pulse is not infrequently irregular in rhythm. There is almost invariably a terminal rise in the pulse during the last few days.

The respiration, like the pulse, usually varies with the temperature. Like the pulse it is also more often increased than diminished in rate. It is not infrequently irregular in rhythm. Typical Cheyne-Stokes respiration is very unusual, the respiration usually being of the Biot type, that is, it is irregular in rhythm and in depth, but not characteristically Cheyne-Stokes. The pulse often varies directly with the respiration, when it is irregular. There is almost always a terminal rise in the rate of the respiration during the last two or three days, as there is in the rate of the pulse and the height of the temperature.

The leucocyte count is very variable. Early in the disease it is likely to be normal, but after a few days is often increased. The usual range is between 10,000 and 15,000. It may run as high as 50,000. If there

is an increase in the count, it is due to polynuclear neutrophiles. There is often a decrease in the eosinophiles.

The urine is normal or shows the changes of acute degenerative nephritis.

The tuberculin test may or may not be positive. In my experience it is more often negative than positive. The failure to react is, of course, because the system is overwhelmed by the infection, tuberculous meningitis being simply one of the forms of acute miliary tuberculosis.

The cerebrospinal fluid is usually said to be either clear or opalescent. In my experience it is almost never really clear. It may seem to be, but, if it is compared with water, it is evident that it is really a little opalescent and not perfectly clear. The various tests for globulin are positive. The glucose is normal or diminished. The results of this test are so variable, however, in all types of meningitis and cerebral disease that it is of no practical value. The number of cells is increased, usually being between one hundred and two hundred per cubic mm. In some instances, however, there may be as many as a thousand. The cells are chiefly mononuclear, there being from 90% to 100% of mononuclears in 70% of the cases and over 80% in 90% of the cases. In rare instances the polynuclear cells may predominate. The fluid always forms a fibrin clot, which becomes larger as the disease progresses. It is presumable that the fluid always contains tubercle bacilli. The frequency with which they are found, however, depends very largely on the skillfulness and care of the observer. Some people can find them in nearly 100% of the cases, while others are almost never able to discover them. As the organisms are more numerous in the latter part of the fluid, it is wise to get the fluid in two portions and examine the latter for organisms. The fluid may be centrifugalized and the sediment examined. It is more satisfactory, however, to allow the fibrin clot to form and then to examine the clot. Various methods of digesting the fluid have been described, but they are not necessary, if care is taken in the examination of the sediment or of the clot.

The course of tuberculous meningitis is much shorter than is generally supposed. The average duration in infancy is about two and one half weeks. It is very seldom over three weeks. I have seen a number of cases in which the duration was not more than a week and one in which it was only thirty-six hours. This patient was under treatment in the hospital for another disease and did not show any signs of meningitis before the acute onset. The diagnosis was confirmed by autopsy. The duration of the disease is a little longer in childhood, the average being a little over three weeks. It is almost never over five weeks. The reason of the old belief that tuberculous meningitis often runs a very long course is presumably because, before the days of lumbar puncture and examination of the cerebrospinal fluid, the chronic type of meningococcus meningitis was mistaken for tuberculous meningitis.

Physical Examination.—The anterior fontanelle, when it is open, is usually bulging, but may be level or depressed. There is usually some rigidity of the neck and also some retraction. The rigidity and retraction usually increase and are most marked towards the end. Opisthotonos is unusual. Rigidity of the neck and spine and retraction of the head may be entirely absent in the flaccid type. Brudzinski's neck sign is often present, but is not at all constant. Dilatation of the pupils, when the head is bent forward, is far more common than is Brudzinski's sign and is, perhaps, of more importance. Strabismus is common. Absence of the

reaction of the pupils to light is also common. The pupils are often irregular and are more often dilated than contracted. Tubercles are said to be common in the choroid. I have never seen one, however, and have met very few people who ever have, although all the text-books speak of them as one of the points in diagnosis. Spasm or paralysis of other muscles supplied by the cranial nerves is unusual, except late in the disease. There are usually evidences of involvement of the pneumogastric. The disturbances of the pulse and respiration from this cause have already been mentioned. The abdomen is usually sunken. The sinking in of the abdomen is not, however, in most instances due to muscular retraction, as is the common belief, but simply the result of the absence of anything in the stomach and intestines to keep the abdominal wall out. The liver and spleen are not enlarged. The abdominal reflexes are very variable, as are also the cremasteric. There is more or less spasm of the extremities in most cases. In the flaccid type, however, there is not only no spasm but the extremities are absolutely limp. There is nothing

FIG. 166.—Tuberculous meningitis.

constant about the deep reflexes. The knee-jerks may be increased, normal or diminished and are often different on the two sides. Babinski's and Oppenheim's phenomena and the contralateral reflex may or may not be present. There is nothing characteristic about the sensation, which may or may not be disturbed. There is, however, usually no hyperesthesia. The tâches cérébrales are usually present, but are of no special value in diagnosis. Other vasomotor disturbances, especially intermittent flushing of the cheeks, occur earlier and are far more important in diagnosis.

The common conception that all infants and children ill with tuberculous meningitis have a bulging fontanelle and strabismus, are in the position of opisthotonos, constantly twitching and having repeated convulsions is erroneous. In my experience marked symptoms of this sort are comparatively unusual. Only a few of them are present, as a rule, in any individual case. In most instances there is some paralysis or spasm of the eye muscles and some tenderness and retraction of the neck, but little or no opisthotonos. There is also likely to be some spasm of the extremities with some twitching and often, towards the end, convulsions. It is not uncommon, moreover, for the disease to run its whole course without any convulsions, twitching or rigidity, the condition being one of flaccidity with diminished or absent reflexes. In such cases there is usually no bulging of the fontanelle and no crying out.

Diagnosis.—It is impossible to make a positive diagnosis of tuberculous meningitis in the early stages, unless the onset is very acute. It must be thought of as a possibility, however, whenever a child becomes irritable or uninterested or vomits without apparent cause. It is not wise, however, to mention this possibility to the parents unless the symptoms persist. The physician must always have tuberculous meningitis in mind when the symptoms are indefinite and hard to explain in early life. Even when the physician is aware of the indefiniteness of the symptoms of tuberculous meningitis, he is almost certain not to suspect it in the first few days, however, and to attribute the early symptoms to something else. As already stated, the only physicians who have not failed to recognize the early symptoms of tuberculous meningitis are those who have not had the opportunity to miss them. When these early symptoms come on in a baby or a child known to have had a definite exposure to tuberculosis, in one known to have a tuberculous focus somewhere in the body or after an operation on some tuberculous process, it is less difficult to recognize them and to appreciate what they mean.

When symptoms pointing directly to involvement of the central nervous system have developed, it is necessary to distinguish tuberculous meningitis from other forms of meningitis, acute encephalitis and infantile paralysis. The differential diagnosis between tuberculous and cerebrospinal meningitis is taken up under cerebrospinal meningitis. The findings in the cerebrospinal fluid make the diagnosis between tuberculous meningitis and other forms of meningitis easy. In tuberculous meningitis the cerebrospinal fluid is opalescent, while in other forms of meningitis it is turbid or purulent. The increase in the number of cells is usually much greater in other forms of meningitis than in tuberculous meningitis. The predominating cell in tuberculous meningitis is the mononuclear and in other forms the polynuclear. The causative microorganisms are usually abundant in the other forms of meningitis. In tuberculous meningitis they are present, but may be difficult to find. The differential diagnosis between tuberculous meningitis and meningococcus meningitis is discussed under meningococcus meningitis and that between tuberculous meningitis and encephalitis and infantile paralysis in the diagnoses of these diseases.

The question occasionally arises, when it is evident both from the symptomatology and the findings in the cerebrospinal fluid, although no organisms are found in it, that the child has a disease of the central nervous system, whether it is justifiable to make a positive diagnosis of tuberculous meningitis, when the weight of the evidence points strongly towards it. This question fortunately seldom arises or can be settled when careful examinations of the fluid for tubercle bacilli are made, because they can usually be found if carefully sought for. When they are carefully sought for and not found, it is usually advisable not to make a positive diagnosis of tuberculous meningitis, but only a strongly probable one. If careful search for some reason cannot be made, it is usually safe to make a diagnosis of tuberculous meningitis with the reservation that it possibly may be a mistaken one.

Tuberculous meningitis may sometimes be confused with other acute diseases, such as influenza and typhoid fever. The diagnosis is usually easy, however, if sufficient care is taken in physical examination, and can always be made positive by lumbar puncture and examination of the cerebrospinal fluid.

Prognosis.—I have never seen a case of tuberculous meningitis, in which the diagnosis was proved by the finding of tubercle bacilli in the

cerebrospinal fluid, recover. There have been, however, a number of cases of recovery from tuberculous meningitis in which tubercle bacilli have been said by competent observers to have been present in the cerebrospinal fluid and one or two cases in which children have died later after having had what was supposed to have been tuberculous meningitis with recovery, and healed tuberculous lesions found in the meninges. Granting, however, that some twenty cases of tuberculous meningitis have recovered, this number is so small in comparison with the number of cases of tuberculous meningitis which must have occurred throughout the world during this same period that it does not justify anything but an absolutely hopeless prognosis in this disease. It seems to me that the chances of recovery are so infinitesimally small, when the diagnosis is proved by finding tubercle bacilli in the cerebrospinal fluid, that it is unfair to hold out the slightest hope of recovery. It is safe to say, however, when the diagnosis of tuberculous meningitis is made without the finding of tubercle bacilli in the cerebrospinal fluid, that there is a chance of a mistake in the diagnosis. Even then it should be made plain that, if the diagnosis is correct, there is no chance for recovery.

Treatment.—The treatment of tuberculous meningitis can, of course, be only symptomatic. It should be carried out, however, as if the child was certain to get well, because there is always the possibility of a mistake in the diagnosis. Even laboratory men have been known to make mistakes and call things tubercle bacilli which were not. They have even been known to mix up the cerebrospinal fluids from two cases. I have known of one instance in which the staining fluid used in the laboratory contained tubercle bacilli. I have known of other cases in which the apparatus was not properly cleaned. It is always necessary, therefore, to act as if the patient may get well. The child should be fed with a tube, if necessary. It should not be allowed to die of starvation. Bromide or opium in some form should be used to control discomfort and marked nervous symptoms. Repeated lumbar punctures to relieve the increased cerebral pressure have, however, been more useful in my experience than either bromide or opium. I have frequently seen a child screaming with pain or having repeated convulsions become perfectly quiet and go to sleep after a lumbar puncture. This procedure has, of course, no curative affect, but does materially diminish the suffering, both of the child and of the family.

Diseases of the Spinal Cord

Except for infantile paralysis, diseases of the spinal cord are much less common in early life than later, although children may have all the diseases of the spinal cord which occur in adult life. Those diseases of the spinal cord which are due to syphilis are much less common in children than in adults, because for some unknown reason syphilis is less likely to attack the nervous system in early life than it is later. Degenerative lésions of the cord are also less common in early life. On the other hand, children have certain rather characteristic familial diseases of the cord and may have certain congenital malformations of it. Infants also have lesions of the cord as the result of injuries at birth.

Myelitis in early life is usually due to an injury or to pressure, either from the bones or from an abscess, in the course of Pott's disease. In rare instances it may be, as in later life, inflammatory in origin. The symptomatology and course are the same as in adults.

Tumors of the spinal cord may occur at any age and are probably as common in childhood as in adult life. The symptomatology, course, prognosis and treatment are the same as in adults. It is worth remembering that, when the cerebrospinal fluid is clear, yellowish and coagulates spontaneously, it indicates that there is an obstruction somewhere in the spinal subarachnoid space. In early life the obstruction is in most instances due to a tumor.

PARALYSES DUE TO INJURY TO THE SPINAL CORD DURING BIRTH

Injury to the spinal cord occurs not very infrequently during birth, especially in breech extractions. The cord may be ruptured locally or a large portion of it may be injured as the result of hemorrhages in or about it. The symptoms must necessarily vary markedly according to the location of the lesion. Lesions high in the cervical region are almost invariably fatal. Cross lesions below this level, if accompanied by hemorrhage enough to destroy the cells in the segments below, cause anesthesia and permanent flaccidity below the lesion. Cross lesions of small extent, which do not involve the lumbar enlargement, show at first anesthesia with flaccid paralysis. After a few weeks, however, while the anesthesia remains, the zone of flaccidity is limited to the muscles supplied by the anterior horn cells in the segments destroyed. Below this level there is marked reflex activity. The bladder and rectum become "automatic," that is, they are emptied reflexly from time to time, when full. (Crothers, American Journal of Medical Sciences, 1923, CLXV, 94.)

The general tendency in these cases is to slow improvement. The upper limit of the area of anesthesia gradually moves downward and the upper limit of the paralysis also moves downward. Complete recovery is, of course, entirely out of the question. There being no involvement of the brain, pons or medulla, there is no disturbance of the mentality and no involvement of any of the muscles supplied by the cranial nerves.

Diagnosis.—These cases, especially when there is exaggeration of the deep reflexes and spasm of the lower extremities, are often mistaken for cerebral paralysis. Careful examination, however, shows marked differences. In this condition the mentality is normal, while it is likely to be affected in cerebral paralysis. Sensory disturbances, at any rate to the same extent, are not present in cerebral paralysis. If there are sensory disturbances, there is never such a complete absence and such a definite upper level as in these cord paralyses. There is less reason for confusion, of course, when the deep reflexes are absent and the paralysis is flaccid.

Treatment.—In the treatment of these cases the first thing to remember is that the children are normal mentally and that their education must, therefore, be looked after. The next thing to remember is that improvement can be expected in all these cases. It is worth while, therefore, to do everything for them that can be done. Treatment consists in massage, muscle training and in suitable cases the application of proper orthopedic apparatus.

The most important part of the treatment of these cases, however, is the preventive. Physicians in general apparently do not realize how much danger there is of injury to the spinal cord in breech extractions, and also in head extractions, when the body is twisted. More care at the time of delivery to prevent twisting or undue bending of the spine will unquestionably diminish the number of these sad cases very materially.

SYRINGOMYELIA

In this disease cavities form in the spinal cord, either as the result of a congenital malformation or of a gliomatous process starting in the vicinity of the central canal and causing destruction of tissue. These cavities may occur anywhere in the spinal cord, but are most common and largest in the cervical segments. It is a very rare condition and the symptoms seldom appear before adolescence. It is evident that they must vary according to the location of the lesions in each case. The most characteristic symptoms are vasomotor manifestations, trophic disturbances, various forms of cutaneous anesthesia and muscular atrophy. All of these symptoms are likely to be limited to one extremity or to one side of the body. The more severe manifestations, which develop late in the disease, are never seen in childhood. The diagnosis of this disease in childhood is almost always impossible, but peculiar manifestations, such as localized sweating, disturbances of sensation, or local muscular atrophies, should always suggest it as a possible cause.

The progress of the disease is slow and often interrupted, but the end is always in death after many years. There is no treatment.

FRIEDREICH'S DISEASE

This disease, also known as Hereditary Spinal Ataxia and Friedreich's Ataxia, is the least uncommon of the familial group of ataxias, which includes, in addition, hereditary cerebellar ataxia and hereditary ataxic paraplegia. It is seldom directly inherited, but there are often other cases in the same family or in near relatives. The cause of the degenerative lesions in the cord is unknown, although syphilis and intemperance in the parents and an inherited lessened resistance of the nervous system apparently play some part in the etiology. It is about equally common in the two sexes.

Pathology.—The spinal cord is smaller than normal and shows degenerative changes—sclerosis—of various tracts which differ at different levels and in different cases. In general, the posterior and lateral columns are most involved, but often to a varying degree. The column of Goll is more seriously affected than that of Burdach. The cells of the anterior horns are almost never involved. There is sclerosis of the posterior and, to a less degree, of the anterior nerve roots. The peripheral nerves, however, show very few changes.

Symptomatology.—The symptoms usually first appear between the fourth and seventh years, but may be present in infancy. They are ordinarily fully developed by twelve years. The first symptom is unsteadiness in walking and standing. The children stand and walk with their legs wide apart and have a peculiar rolling gait. Romberg's symptom is usually, but not always, marked. The ataxic motions of the legs are present, even when the child is lying down. The knee-jerks are absent, but the superficial reflexes are retained. After a time the arms and head are involved as well as the legs. There is a coarse tremor in many cases and in others involuntary movements, much like those in chorea. There is at first no impairment of muscular power, but weakness and atrophy of the muscles gradually develop and the child becomes bedridden. A peculiar deformity of the foot often develops. The toes, especially the great toe, are hyperextended and the foot is in the position of equinovarus. Deformities of the spine are also not uncommon late in the disease. Sensation is usually normal and control of the sphincters of

the bladder and rectum is retained. Lancinating pains are unusual. The speech becomes slow, scanning and difficult, finally being impossible. Nystagmus develops after a few years, but the Argyll-Robertson pupil is never present. The intelligence remains normal for a long time, but, towards the end, deteriorates. The course of the disease is steadily from bad to worse and death finally ensues, usually as the result of some intercurrent disease, after twenty or thirty years of suffering and incapacity. There is no treatment which has any effect on the course of the disease. All that can be done is to make the patient as comfortable as possible.

Hereditary Ataxic Paraplegia usually does not develop before adolescence, but some slowness in locomotion and awkwardness in the use of the legs may be noticed earlier. The legs are stiff, weak and ataxic. The progress of the disease is very slow and it is not inconsistent with long life. The deep reflexes are exaggerated. There may be some paresthesia. The control of the sphincters is normal and there is no disturbance of the intelligence. The symptoms point to lesions in the lateral columns associated with others, either in the cerebellum or the posterior columns. *Hereditary Cerebellar Ataxia* has already been spoken of. All sorts of combinations and variations of these types of familial ataxias may occur, in accordance with the distribution of the degenerative lesions in the cord.

PROGRESSIVE MUSCULAR ATROPHY

It is evident that, theoretically, progressive muscular atrophy may be due primarily to a lesion either of the lower motor neuron or of the muscle itself. It is also conceivable that the lesion of the neuron may be primarily in either the ganglion cells of the cord or in the peripheral nerves. It is doubtful, however, whether the lesions are really so sharply defined, especially as regards those of the neurons. The ordinary adult type (Aran-Duchenne) of progressive muscular atrophy and amyotrophic lateral sclerosis occur so seldom in childhood that it is not necessary to consider them. The Werdnig-Hoffmann type of spinal muscular atrophy, however, is characteristic of childhood and the peroneal type of neural muscular atrophy (Charcot-Marie-Tooth) usually begins in middle or late childhood.

Werdnig-Hoffmann Type of Progressive Muscular Atrophy

Pathology.—In this condition there is degeneration of the ganglion cells of the anterior horns throughout the whole extent of the spinal cord, with secondary degeneration of the anterior roots and motor nerves. There is marked atrophy of the muscles. The sensory nerves and the sensory tracts of the cord, as well as the pyramidal tracts, are normal.

Symptomatology and Course.—This rare condition is definitely a familial disease, occurring in several children of the same family and in several generations. The first symptoms usually appear towards the end of the first or during the second year in a child that has up to that time developed normally. They are weakness of the muscles of the thighs and back. The muscles of the shoulder and neck are next involved and finally those of the arms and legs. The hands and feet are almost never affected. Atrophy of the muscles goes hand in hand with increasing weakness, and is most marked about the shoulders and pelvis. The muscles sometimes show fibrillary twitchings. The reflexes, both deep and superficial, gradually diminish and finally disappear. There is also a progressive diminution in the response to both the faradic and galvanic currents, with either a complete or partial reaction of degenera-

tion. All sorts of deformities may develop and the child becomes more and more helpless. Death usually occurs in two or three years as the result of some respiratory infection.

The muscles supplied by the cranial nerves are not involved. The mentality is normal and there is no disturbance of the control of the sphincters. Sensation is normal.

There is no treatment which delays in any way the progress of the disease.

Diagnosis.—The early appearance of the symptoms, the rapidity of the course and the location of the paresis are sufficient to distinguish this type of muscular atrophy from the neural type and from the muscular dystrophies. The similarity of the pathologic lesions in this disease and in amyotonia congenita suggests that they may be different forms of the same disease. Clinically, however, they are quite different. The symptoms in amyotonia congenita are almost always present at birth; those in this disease develop at about one year. Amyotonia congenita sometimes occurs in more than one member of a family, but the familial tendency is not as marked as in this form of progressive muscular atrophy. There is general hypotonia in amyotonia congenita and localized atrophies in this disease. The muscular atrophy is masked in the former, apparent in the latter. There is no reaction of degeneration in one, while it is present in the other. There is a definite tendency to improvement in amyotonia congenita and a progressive increase in all the symptoms in Werdnig-Hoffmann's disease.

Peroneal Type of Neural Muscular Atrophy

This type of progressive muscular atrophy, also known as the Charcot-Marie-Tooth type, is markedly familial in origin, often occurring in several successive generations. It is a little more common in boys than in girls. The onset is usually after six years. The degenerative changes are more marked in the nerves in this type than in the others, but the ganglion cells of the anterior horns also show some changes. There are also degenerative changes in the posterior columns of the cord and degenerative and atrophic changes in the muscles. This type of progressive muscular atrophy ought not, therefore, to be looked upon as a special form of disease, but merely as one of a large class, very similar to each other, which has a rather characteristic symptom-complex.

Symptomatology.—The extensor muscles of the toes and those of the peroneal group are almost always the ones first involved. The small muscles of the feet are involved at the same time or soon after. As the result of the paresis of the extensors and of the peroneal group, equinevarus develops. Incidentally the development of equinovarus at any time after infancy ought always to suggest this disease as a possible cause. As the result of the weakness of the extensors, the foot hangs down, and in order to walk the child has to bend the knees. The peculiar gait which results is known as the "stepping" or "steppage" gait. After a time, perhaps of years, the small muscles of the hands are affected, although in rare instances they may be attacked at the same time or even before those of the feet. The process gradually extends to the other muscles of the legs and to the forearms, but those of the thighs and upper arms are seldom attacked. There is never any hypertrophy or pseudohypertrophy of the muscles, simply wasting. The reflexes in the lower extremities are diminished or absent, as they often are later in the upper extremities. The electrical reactions are diminished and often

changed in character—reaction of degeneration. The control of the sphincters and the intelligence are normal. Sensory disturbances, both subjective and objective, are not uncommon.

The progress of this disease is slow and not inconsistent with long life. There are not infrequently intermissions, and complete arrest sometimes occurs.

Treatment.—There is no treatment which has any effect on the course of the disease. Proper massage and the use of electricity serve, however, to keep up the tone of what muscle there is left. Muscle training is also of some value. Much can be done to prevent contractures and to improve the walking by the application of splints and by suitable operations on the tendons.

PROGRESSIVE MUSCULAR DYSTROPHIES

The muscular dystrophies are familial and hereditary. Transmission usually occurs through the mother. Boys are more often affected than girls. Further than this nothing whatever is known as to the etiology of these diseases. Several types of muscular dystrophy are recognized, according to the location of the muscles involved and whether they are at first diminished or increased in size. While these types are quite characteristic, many intermediate forms occur which it is impossible to classify. The three main types are:

1. The Pseudohypertrophic Type of Duchenne
2. The "Juvenile," or Scapulohumeral, Type of Erb
3. The "Infantile," or Facioscapulohumeral, Type of Landouzy and Déjerine.

Pathology.—The pathologic lesions in the muscular dystrophies are confined to the muscles and are essentially the same in all types. The essential process is an atrophy of the muscle fibres. Some of the fibres may hypertrophy for a time, but they also finally atrophy. Microscopically the muscle fibres show loss of striation, fatty degeneration and vacuolation. The muscle fibres are replaced by connective tissue and fat. If there is sufficient fat deposited, the muscles appear of normal size, or may be even larger than normal. In time, however, the fat is absorbed and the muscles become small. Permanent deformities may result from the contraction of the connective tissue which has replaced the muscle fibres.

Pseudohypertrophic Paralysis

This type, which is also known as *Muscular Pseudohypertrophy*, usually begins between the second and seventh years. The first symptoms are weakness in the muscles of the legs and an increase in the size of the calves. In some cases the muscles of the thighs are also enlarged and occasionally those of the buttocks. All the muscles of the thighs and calves become involved, as well as the dorsal flexors of the feet. The deep muscles of the back and also those of the abdomen are often involved quite early. As the result of the weakness of the muscles of the lower extremities and back, the child walks awkwardly, usually with a waddling gait, and has a marked degree of lumbar lordosis. It also has great difficulty in rising from a recumbent position. It first turns on its face, draws its legs under it and then lifts itself from the floor on its extended arms and legs, standing like a four-legged animal. It then "climbs up itself" by putting one hand on the corresponding knee, then the other on the other knee and then working them up the thighs.

The muscles of the shoulders and scapulae are involved not long after those of the lower extremities. There is usually no hypertrophy, but in some instances the deltoids, spinati and triceps may be enlarged for a time. In some instances the child works the hand up along the head in order to stretch its arm upward. The muscles of the face, forearms and hands are not involved, except in the last stages.

The enlarged muscles finally waste with the others, and the child becomes weaker and weaker and finally helpless. Before this happens, various deformities from contractures, especially pes equinus and at the knee and elbow joints, often develop. The progress of the disease is slow, death occurring, usually from some intercurrent disease, in from five to ten years. The mentality usually remains normal to the end. Control of the sphincters is retained. There are no fibrillary twitchings. Sensation is not impaired. The reflexes are retained as long as there is muscle enough to give them. There is no reaction of degeneration, but the response to electrical stimulation steadily diminishes with the wasting of the muscles.

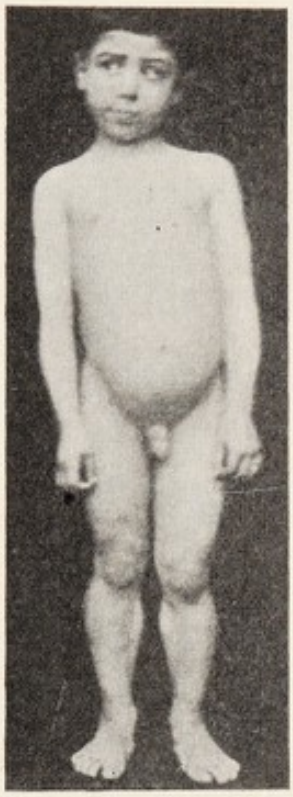

FIG. 167.—Pseudohypertrophic paralysis.

Treatment.—There is no treatment which has any effect on the course of the disease. Something can be done, however, to prevent the development of contractures by passive motions and manipulations and by the application of suitable apparatus. Proper apparatus may also make locomotion possible for a longer time and put off the time when the patient has to go to bed to stay.

JUVENILE, OR SCAPULOHUMERAL, TYPE

The onset of this type is usually between the tenth and sixteenth years. The muscles of the shoulder girdle are first affected. Some are atrophied from the beginning, while others are for a time enlarged. The pectorals, trapezii, latissimi dorsi, serrati and rhomboids waste from the beginning. The deltoids and spinati are likely to be at first hypertrophied. The muscles of the upper arms also waste early. Soon after the involvement of the muscles of the shoulder girdle those of the pelvic girdle are attacked. These may waste from the first, but at times there is hypertrophy of some of them. The deep muscles of the back are also involved, but those of the face, forearms, hands and legs escape until late in the course of the disease. As in the other types, there are no fibrillary twitchings and no sensory disturbances. There is never any reaction of degeneration. The response to electrical stimulation and the strength of the reflexes depends on the amount of muscular tissue which remains. Intelligence is not impaired and the control of the sphincters is normal. The prognosis is as bad as in the other types and the treatment just as unsatisfactory.

INFANTILE, OR FACIOSCAPULOHUMERAL, TYPE OF LANDOUZY AND DÉJERINE

This type begins within the first few months of life. The muscles first affected are those of the face, especially the sphincters of the eyes and mouth. Atrophy is marked from the beginning, except of the lips, which are thickened and protruding, giving rise to the term of "tapir mouth." The muscles of deglutition and mastication, as well as the

ocular muscles, are not affected; neither are the respiratory nor laryngeal muscles. The absence of involvement of these muscles makes it possible to distinguish this condition from bulbar paralysis. Later on the atrophy spreads to the shoulder and arm muscles. The spinati are, however, not involved. After a time the atrophy may extend to the lower extremities. There are no fibrillary contractions and no reaction of degeneration. Sensation is normal. The course is very slow. There is no treatment.

AMYOTONIA CONGENITA

This rather uncommon condition was first described by Oppenheim in 1900. It is characterized by muscular weakness, which is probably always congenital. At any rate, it is usually noticed at or soon after birth and in the majority of cases there is a history of an absence or feebleness of the fetal movements during pregnancy. It is probable that when the symptoms apparently develop later, they were overlooked or misinterpreted during the early months.

Symptomatology.—The chief characteristic of this disease is flaccid paralysis, which may, however, differ in degree. The lower extremities are most often and most severely involved. The muscles of the arms, abdomen, spine and neck may also be involved, and usually in the order named. In the most severe cases the babies are absolutely helpless and perfectly limp. There is abnormal motility of the joints as the result of the hypotonicity of the muscles. When the intercostal muscles are involved, as they often are, marked pigeon breast results, with interference with respiration and a tendency to pulmonary infections. The diaphragm is almost never affected. The muscles of the face are occasionally involved to a slight extent and sometimes those of deglutition. In some of the less severe cases contractures of the stronger muscles—adductors and flexors—may develop. In spite of the marked muscular weakness there is no wasting and the baby is plump and well nourished. The tendon reflexes are diminished or absent, according to the severity in the individual case. The reaction to the faradic current is much diminished, but normal in character. Sensation is normal. If the baby lives to be old enough to control its sphincters, it is able to do so. The mentality is normal. Milder forms of the disease undoubtedly exist in which there is no real paralysis, but merely unusual weakness of the muscles.

Prognosis.—The tendency in this disease is undoubtedly towards improvement. In most of the cases, however, unless they are of a mild type, death occurs from respiratory infections, resulting from the deficient expansion and deformity of the chest, before improvement has become very noticeable. Improvement is only relative, moreover, and never goes on to complete recovery, even in the least serious cases.

Etiology.—Little definite is known as to the etiology of this disease. It is certainly not due to syphilis nor any abnormality in the secretions of the endocrine glands. There is apparently a certain, but not marked, tendency to familial occurrence. The disease also apparently occurs somewhat more often in the children of aged parents and in the later children of large families, which suggests that it may be due to reproductive exhaustion. It is certainly a congenital failure of development, not a manifestation of degeneration of the nervous system.

Pathology.—All observers agree that the muscles are abnormal, but there is considerable difference of opinion as to the changes in the nervous system. The muscles are macroscopically pale, soft and flabby and

usually much diminished in size. The muscle fibres are mostly very small, but some of them are normal or hypertrophied, the relative proportions varying in different muscles and in different portions of the same muscle. The nuclei are of normal size and the cross striations are well preserved. There is a considerable increase in the amount of fat and connective tissue in the muscles, but usually no evidences of degeneration.

The brain, cerebellum and medulla are almost always normal. The ganglion cells of the anterior horns of the spinal cord are considerably diminished in number and those that are left are usually small. The cord tracts show a deficiency in myelinization, but no evidences of degeneration. The posterior roots are normal, but the anterior roots are small and many of the fibres are deficient in myelin. The peripheral nerves show a defective myelinization of the nerve fibres, with an increase of connective tissue, but seldom any evidences of degeneration. The data as to the muscular nerve-endings are conflicting, but apparently they may at times be lacking in the affected muscles.

At present it is very difficult to state positively whether the pathologic process in amyotonia congenita is primarily a congenital developmental defect of the lower motor neuron, of the muscles, or of both. It seems probable, however, that it is a congenital defect in the development of both the lower motor neuron and the muscles.

Diagnosis.—The symptomatology of well-marked cases of this disease in early infancy is so characteristic that it is almost impossible to mistake it for anything else. When it is overlooked, it is usually because the physician does not know about it. It is sometimes mistaken for simple muscular weakness or for backwardness in development. Muscular weakness, at any rate as marked as it usually is in amyotonia congenita, does not occur without some evident cause, such as some serious illness or marked disturbance of nutrition. Simple backwardness is not accompanied by so much muscular weakness, the reflexes are not diminished and there is always some evident cause for it. Feebleminded infants are backward and often very quiet. A little observation shows at once, however, that they are deficient mentally, while infants with amyotonia congenita are normal mentally. Feebleminded infants do not move because they do not know how, infants with amyotonia congenita because they cannot. There may be a superficial resemblance between amyotonia congenita and some of the transverse lesions of the cord, due to injury at birth. Sensation is lost in the paralyzed areas in birth injuries, however, and retained in amyotonia congenita. The knee-jerks are often exaggerated in birth injuries, absent or much diminished in amyotonia congenita. The paralysis is sharply limited in birth injuries, less so in amyotonia congenita. All the muscles may be involved in amyotonia congenita, but never are in birth injuries, because death would have occurred, if the injury was high enough to involve all the muscles. There is a certain resemblance between amyotonia congenita and some of the muscular dystrophies. The symptoms are present at birth in amyotonia congenita, however, and do not develop until later in muscular dystrophy. Definite muscles are involved in muscular dystrophy, while all the muscles of an extremity are involved in amyotonia congenita. There is evident wasting or hypertrophy of individual muscles in dystrophy; the muscles appear normal in amyotonia congenita. There is a tendency to improvement in amyotonia congenita; the condition grows steadily worse in muscular dystrophy.

Treatment.—There is no specific treatment for amytonia congenita. It is useless to give any of the glandular extracts. Something can be done to improve the condition of the muscles by regular massage and, perhaps, by the use of faradic electricity. Passive motions to prevent contractures are also indicated. Muscle training helps when children are able to use their muscles at all. It is also extremely important, especially in the cases in which the intercostals are involved, to protect them against respiratory infections. Great care must be taken also to prevent the development of disturbances of nutrition, especially of rickets, which is a very serious complication in this condition. Fresh air, sunlight and cod liver oil are, therefore, important adjuncts in the treatment.

ACUTE POLIOMYELITIS

This disease, commonly known as infantile paralysis, is better described by the term poliomyeloencephalitis, because of the wide distribution of the lesions. It is not a new disease, but is probably as old as man. It occurs sporadically every year and at all times of the year, but is more common in the late summer and early autumn. Recently, there have been quite extensive epidemics in various parts of the world. There is likely to be a flaring up every six or seven years in a given community. It may occur at any period of life, but is most common in the second year, relatively uncommon after eight years and rare after twenty years.

Etiology.—It is almost certainly due to the microörganism discovered by Flexner and Noguchi. This organism belongs in the class of the filterable viruses. It can be cultivated only under anaerobic conditions. It is a small globular or globoid organism, averaging from 0.15 to 0.3 micron in size. It appears in a variety of arrangements, single, double, in short chains and in masses. It is stained reddish-violet by Giemsa's solution, and by Gram's solution. Rosenow has also described an organism which be believes to be the cause of the disease. It is possible that it may be the same organism as that described by Flexner and Noguchi. Rosenow's arguments in favor of his organism are not as convincing as those of Flexner and Noguchi.

This virus is very potent. It resists glycerinization, drying and freezing, but is readily injured by heating. It is destroyed by a 1% soiution of peroxide of hydrogen. The ordinary domestic animals are not susceptible to it, and the epidemic paralysis of animals is not caused by it. It can survive in the body of the biting fly, and in the bedbug for seven days. It does not survive in the mosquito. It may be carried on the feet and body of the ordinary fly. It may be present in the dust of rooms occupied by patients ill with the disease. It is not contained in water, milk or food, unless they are contaminated by flies.

It is present in the nasopharyngeal secretions of those ill with the disease and often for many months after recovery. It may also be present in the nasopharynx of persons who are associated with those ill with the disease, but who are healthy themselves, and may be present in them also for many months. It may also be present in the intestinal discharges of those ill with the disease.

Infection in man almost certainly takes place through the nasopharynx. It is just as certain that it does not take place through the digestive tract. It is barely possible, but very improbable, that it

may be transmitted by the bite of an insect. The infection is at first systemic, but finally the barrier of the choroid plexus is passed and infection of the nervous system results. The virus leaves the body through the nasopharynx and in the discharges from the bowels. It may continue to be present in the secretions of the nasopharynx for many months.

An attack of the disease confers a lasting immunity. A second attack is almost unknown. The vast majority of people are apparently immune. This apparent immunity may be due to the fact that infection with the virus is almost universal, but that in only a small proportion of cases it passes into the nervous system and the nature of the infection is, therefore, not recognized. It may be also due to the presence of protective substances in the nasopharyngeal secretions of most people.

Infantile paralysis is unquestionably a contagious disease. Its contagiousness is, however, very slight, as is shown by the following facts: More than one case seldom occurs in the same family. If there are more, they almost always occur at about the same time, not one after another at intervals. Infantile paralysis does not spread in hospitals, even when treated in the open wards without special precautions. In the past I have seen it treated for many years in the open wards in several hospitals without any special precautions and have never known a case to develop in a hospital. Nothing is known accurately as to the period of incubation, although it is probably under two weeks. On the other hand, it may be very long, as the virus may probably be present in the nasopharynx for a long time without causing infection, just as in meningococcus meningitis and in diphtheria. It is impossible, moreover, to make any statements as to the duration of the period of contagiousness, because there is no practicable method of determining whether the virus is still present in the secretions of the nasopharynx or not.

Pathology.—Nothing is known as to the pathologic changes in man before the nervous system is involved. Experimentally the virus localizes itself first in the spleen and bone marrow. In fatal cases in which the nervous system has been attacked there is, in addition to the changes in it, hyperplasia of the lymphatic glands, spleen and intestinal lymph nodes. There is also interstitial small cell infiltration in the portal spaces of the liver and general cloudy swelling of the parenchymatous organs. The tonsils and lymphatic structures of the pharynx are also hyperplastic. The important lesions are, of course, in the nervous system. The earliest change is a perivascular infiltration of small round cells along the blood vessels of the leptomeninges, showing that the virus, after passing through the choroid plexus, is suspended in the spinal fluid and passes in it into the perivascular lymph spaces. The blocking of the spaces with cells causes pressure upon the blood vessels. The virus enters the interior of the cord by the anterior fissure along the branches of the anterior spinal artery, the pathologic changes being the same. On account of the engorgement of the vessels minute capillary hemorrhages often occur. In addition to the cellular infiltration and hemorrhage there is also marked edema, which exerts pressure upon the cells of the anterior horn. Degenerative changes, varying from slight swelling to complete destruction, develop in the ganglion cells. It is probable that these changes are due in part to the interference with the blood supply and in part to a direct toxic action of the virus. The lesions, however, are not confined to the blood vessels and anterior horns of the spinal cord, but similar interstitial changes may be found throughout the rest of the cord. Similar changes,

both in the gray and white matter may occur throughout the whole central nervous system. It is evident, therefore, that the old conception of anterior poliomyelitis with lesions limited to the anterior horns was erroneous and that our conception of the disease must be enlarged to include the whole central nervous system. It is also evident that all sorts of manifestations may develop, according to the location of the pathologic process in the individual case.

In order to understand the later progress and the results of this disease it must be remembered that, while certain ganglion cells are much affected, others are merely compressed or but slightly affected. With the subsidence of the inflammation the compressed cells recover their function and those that were but slightly affected recover theirs in part. The final paralysis is, therefore, much less than the initial. Another reason that the final paralysis is less than the initial is that individual muscles receive their impulses from cells situated at different levels of the cord. The groups of motor cells run lengthwise in the cord and have their association in this direction, while the blood supply is horizontal and the virus enters by the blood vessels.

The pathologic process is most marked, as a rule, in the cervical and lumbar enlargements. Hence, the extremities are most often involved. It is also usually more marked on one side of the cord than on the other. Hence, there is monoplegia and hemiplegia or paralysis of single muscles. The peculiar distribution of the paralysis in an extremity is due to the association of cells vertically, while the blood supply is horizontal.

The reflex arc is destroyed in the spinal cases. Hence, the deep reflexes are diminished or absent. It is not affected in the cerebral cases but, as the inhibition from above is removed, the deep reflexes are exaggerated. The lower motor neuron is involved in the spinal cases and, hence, there is atrophy of the muscles, reaction of degeneration and flaccidity. The paralyzed muscles offer no resistance. Consequently, permanent contractures of the non-paralyzed muscles result. The sensory neurons are but little involved. Hence, there are no disturbances of sensation, except that there may be much pain.

Symptomatology.—There is nothing very characteristic about the onset or the symptomatology of the first stage of infantile paralysis, that is, the stage of systemic infection. In general, however, the early symptoms are like those of an acute nasopharyngitis or tonsilitis, those of an acute disturbance of the digestion, usually associated with diarrhea, or those of an acute intoxication without evident cause. There is a rise of temperature, which may reach 103° F. or 104° F., but which is usually not as high. There is nothing about these symptoms, as a rule, to suggest in any way that they are due to infantile paralysis. In certain cases, however, there are other symptoms which are highly suggestive. Many writers speak of a stupid expression and puffiness about the eyes. I have seldom seen this. Quite characteristic, however, is a condition of excitability and irritability out of proportion to and greater than would be expected from the other symptoms. Still more suggestive is a general hyperesthesia. Excessive sweating is said to be rather characteristic, but, in my experience, is uncommon. Another sign, which is very suggestive, is pain on motion of the spine with a certain amount of stiffness to prevent motion. This is known as the "spine sign". The stiffness and pain on motion is not localized in the neck, as in meningitis, but is general throughout the spine.

There is a polynuclear leucocytosis in all cases. It may be as high as from 15,000 to 25,000. The cerebrospinal fluid at this stage is normal, except that the pressure may be somewhat increased.

The symptoms of the early stage may entirely cease and the child seem well, and then, after one or several days, those of the second or paralytic stage, that is, the stage of invasion of the nervous system, appear. In other instances there is no invasion of the nervous system and, therefore, no reappearance of symptoms. It is these cases which can be truly described as abortive. It is impossible to recognize them positively, even during an epidemic. They are not suspected at other times. Nevertheless, the blood serum of cases of this sort protects against infection.

In another class of cases there is no intermission between the symptoms of the stage of systemic infection and those of the invasion of the nervous system. In some of these cases, however, death may occur as the result of the infection before the evidences of invasion of the nervous system are sufficient to justify a positive diagnosis. In still other cases the symptoms of the stage of systemic infection are so slight that they are entirely overlooked and paralysis is the first symptom of the disease recognized.

The following table is a schematic representation of the possible combinations:

TABLE XXXIV

Systemic infection	Changes in spinal fluid	Symptoms of invasion of the nervous system
I. *General symptoms with improvement or cessation.*	Occur at this time	None
A. Intestinal		Present
B. Respiratory		
C. Toxic		
II. *General symptoms without improvement.*	Occur at this time	None*
A. Intestinal		Present
B. Respiratory		
C. Toxic		
III. *No symptoms.*	Occur at this time	Present

* Death during stage of systemic infection.

Certain changes occur in the spinal fluid just before the symptoms of the invasion of the nervous system appear and others after they are evident. The fluid is usually under somewhat increased pressure. It is clear or slightly opalescent and shows a slight trace of globulin, which later may become more marked. There may be a fibrin clot. The cell count is increased, usually being under two hundred, but it may be as high as twenty-five hundred. If the fluid happens to be taken at the very time of the invasion of the meninges, the cells are largely polynuclear. In a few hours, however, they become almost entirely mononuclear. The cell count may be increased as long as three weeks. The cells gradually diminish in number, however, and before they disappear the polynuclears predominate. The fluid becomes clear, the test for globulin negative and the fibrin clot ceases to form long before the disappearance of the cells. The cerebrospinal fluid always contains sugar.

The white blood count continues to show a polynuclear leucocytosis, usually not very marked, throughout the stage of the invasion of the nervous system.

With the invasion of the nervous system there is a rise in temperature, which may be either moderate or high and may last from a few days to one or even two weeks. Accompanying it are the general symptoms of an acute febrile disease. Sometimes there are convulsions. Hyperesthesia is common and may last for a week or more. There is almost always pain in the extremities and in the back, which in certain types is very severe. The spine sign, already mentioned, is likely to be more marked for a few days. In most instances the children are rather dull and stupid and wish to be left alone. In others they are excitable and irritable. Control of the sphincters is retained. The other manifestations of the disease vary according to the part of the nervous system upon which the stress of the disease falls. No two cases are exactly alike. They may, however, be divided into certain main types. Wickman's classification, made some years ago, has not been improved upon. This is as follows:

1. Ordinary spinal paralysis; anterior poliomyelitis.
2. Progressive paralysis, usually ascending, less often descending; Landry's paralysis.
3. Bulbar paralysis; polioencephalitis of pons.
4. Acute encephalitis, giving spastic, mono or hemiplegia.
5. Ataxic Form.
6. Polyneuritic; multiple neuritic type.
7. Meningitic Form.

It is impossible to describe the symptomatology of all of these types in detail and, in consequence, I shall give illustrative cases of some of them and simply take up some of the important points regarding others.

The Ordinary Type.—In the ordinary type of spinal paralysis it must be remembered that the paralysis may be either very slight or very marked and that in many cases only one or a few muscles may be involved. It must also be remembered that the knee-jerks are not affected if the muscles which give them are not affected. The involvement of the muscles is sometimes very peculiar. In some instances only the muscles of the neck or face are involved, in others only those of the spine, in still others only those of the abdomen, perhaps of only one side. I have known diagnoses of abdominal tumor, hernia and peritonitis made in cases of infantile paralysis involving the abdominal muscles. In one instance, discovered with the Roentgen ray, one half of the diaphragm was all that was involved.

The Progressive Type.—The following case is an example of this type, in which the respiratory muscles were involved:

A boy, ten years old, began to complain of pain in the legs August 18th and by night was unable to walk without help. Pain and loss of power were rather more marked on the 19th. A physician, who was called, found a temperature of 102° F. and made a diagnosis of rheumatism. The pain and fever continued. Pain and loss of power began in the arms August 21st. The morning of the 22nd he began to have difficulty in breathing and to have what were supposed to be convulsions. He rapidly became unconscious. He was seen at 2 P.M.

There was marked cyanosis. The respiration was short and gasping, but not very rapid. Frothy mucus ran from the mouth occasionally or was spit up with considerable difficulty. He was entirely unconscious. There was no rigidity of the neck. The pupils were equal and reacted to light. The chest was fully expanded and immobile. It was tympanitic on percussion. The abdomen was markedly distended and tympanitic. The diaphragm moved, but the excursion was limited. There was a complete flaccid paralysis of all the extremities. The knee-jerks were absent, as were also Kernig's and Babinski's signs. He died a few hours later.

In this instance it is possible that the convulsions and unconsciousness were due to an extension of the process to the brain. My impression is,

however, that they were rather manifestations of infection and carbonic acid poisoning from respiratory paralysis.

Bulbar Paralysis.—Bulbar symptoms may precede or follow paralysis of the extremities. When they are the only ones, they have been described in the past as polioencephalitis superior or inferior, according to the nuclei involved. The type of polioencephalitis superior is an ophthalmoplegia. The following case is illustrative:

A boy, five years old, seemed a little out of sorts August 16th. The next day, while walking along the street, he felt weak, leaned against a fence and vomited. He then walked a short distance to the house where he was going, sat down on the piazza and slept for an hour. On waking he was unable to walk because of staggering and had to be taken home in a carriage. His eyes were turned upward and remained in this position until the next day. He talked and seemed rational. There was no paralysis of the face or extremities. He twitched a great deal for several days and had cold sweats. The staggering gait lasted about a week. He continued to be careful about walking, however, acting as if objects seemed nearer to him than they really were. He complained at times of double vision. Strabismus was noticed about September 1st. There was no nystagmus. He was seen September 19th, a month after the onset.

The physical examination was entirely negative, except for complete paralysis of the left external rectus and partial paralysis of the right. The pupils were equal and reacted to light. The optic discs were normal. There was no paralysis of the soft palate and no disturbance of taste, smell or hearing. His voice was strong and clear.

The following case is an example of involvement of some of the lower nuclei—polioencephalitis inferior:

A boy of five was taken suddenly sick with fever and vomiting, July 26th. The vomiting and fever continued. The morning of the 28th he began to have difficulty in swallowing and his voice was a little hoarse. When seen at 4 P.M., he talked in a whisper and apparently had some trouble in swallowing. Examination of the mouth and throat, however, showed nothing abnormal. The neck was held rather stiffly. Nothing else was detected by a very careful general physical examination. The next day he was unable to swallow anything, even his saliva. When seen at 9 P.M., the whole of the right side of the face, including the forehead, was paralyzed. The tongue was protruded in the median line. The uvula could not be elevated. The pupils were equal and reacted to light. There was no strabismus and no stiffness of the neck. There was a complete paralysis of the extensor muscles of the right arm. The left arm and legs were normal. The knee-jerks were present. Paralysis of the muscles of the right side of the neck was noted the following day. He recovered completely.

The Cortical Encephalitic Type.—It is very difficult, if not impossible in many instances, to distinguish between this type of infantile paralysis and acute encephalitis, except when there is an epidemic of one of these diseases. The following case was supposed to be one of infantile paralysis:

A boy, five years old, was taken suddenly ill February 18th with a severe frontal headache and vomiting. He was very feverish that night, but not much afterward. The next day he became rather stupid and was partially unconscious for twenty-four hours. He did not vomit again and his bowels moved regularly. He was seen February 20th. He was moderately stupid, but noticed and heard. He would not answer questions. The physical examination was entirely negative, except that the knee-jerk on the right was rather more active than that on the left. Two days later it was noticed that he opened the right eye rather less widely than the left and that the mouth was drawn a little to the right. He was mentally very dull. He would sometimes answer a question after several trials but the answer was always irrational. The knee-jerk was more lively on the right than on the left. There was a definite Kernig sign on the right and a suggestion of it on the left. That night the face was definitely drawn to the right and there was a slight loss of power in the left arm. Lumbar puncture, February 22nd, showed a clear fluid under normal pressure. There were thirty cells to the cubic millimeter, all of which were lymphocytes. No fibrin clot formed, but the test for globulin was slightly positive. He was slightly brighter

February 23rd and would answer questions, although his speech was very thick. There was ataxia and intention tremor in both arms, especially on the left. All the extremities were a little rigid. Kernig's sign was present on both sides. That day he had a slight convulsion involving the right arm. The next day he had several severe, general convulsions. He was moderately rigid all day, the neck and back being involved as well as the extremities. From this time on he improved gradually, but steadily, and was discharged March 19th perfectly well in every way, except that the knee-jerk was rather more marked on the right than on the left. Another lumbar puncture was negative and repeated examinations of the fundi showed nothing abnormal.

The Ataxic Type.—The most striking characteristic of this type, as its name signifies, is ataxia. This may be general or confined to the lower extremities. The ataxia may be overlooked, when limited to the legs, unless the child attempts to stand. The symptoms are very similar to those in some types of encephalitis.

The Polyneuritic Type.—The chief characteristic of this type is pain, which at times is very severe. It is usually associated with tenderness. When the children are old enough, they localize the pain along the nerve trunks and in the muscles, not in the joints. The tenderness is also over the muscles and along the nerve trunks and not in the joints. Because of the pain the extremities are often held rigidly, if there is not sufficient involvement of the muscles to prevent it. In certain cases it is very difficult to decide whether there really is any paralysis or not. I have seen several cases in which a whiff of ether, just sufficient to overcome the pain, was necessary, before the paralysis could be recognized.

The Meningitic Type.—The symptoms of the meningitic type are often very similar during the first few days to those of acute meningitis. The diagnosis is often impossible without a lumbar puncture. The following case is an example:

A girl, three and one half years old, was taken suddenly sick September 5th with a high fever. She was very irritable and complained of pains all over, especially in the head and back. She cried out when touched and lay with her back arched and her head thrown back. She had not vomited, the bowels were constipated, she passed urine normally. She was seen September 9th.

She was irrational and very irritable, crying out sharply when disturbed. The ear drums were normal. The pupils were equal and reacted to light, but the eyes were kept closed and there was a moderate amount of photophobia. The neck was rigid and apparently tender. The spasm, however, seemed to be partly voluntary. The back was arched and rigid, but the spasm here also seemed to be partly voluntary. She did not use her right arm. The hand was clenched with the thumb inside. She did not move her left leg at all, but moved the right slightly at the hip. The extremities were somewhat rigid and passive motions caused much pain. The knee-jerks were absent, as were Kernig's and Babinski's signs. There was no disturbance of sensation.

Two days later she was still very irritable, but perfectly rational. She opened her eyes and showed some interest in her surroundings. The rigidity of the neck and back was somewhat less. She could make all motions with her right arm, but rather feebly. She could not move her right leg, but moved the left leg a little at the hip and toes. All movements, whether active or passive, caused pain. The knee-jerks were absent and the plantar reflexes were weak. Sensation was not disturbed

Diagnosis.—The diagnosis of infantile paralysis is impossible in the early stage of systemic infection, but it may be suspected if there is an epidemic. Drowsiness out of proportion to the rest of the symptoms, unusual excitability or irritability, hyperesthesia, stiffness of the back and pain in the back on motion are, however, very suggestive symptoms. At this stage even lumbar puncture does not help in the diagnosis. Lumbar puncture, if the symptoms persist or after they have ceased, may show the changes in the cerebrospinal fluid already described. The

cerebrospinal fluid is always abnormal, when the symptoms of invasion of the nervous system have appeared. Lumbar puncture is, however, hardly necessary for a diagnosis in the ordinary spinal case. At this time the only condition for which it is likely to be mistaken is rheumatism. In infantile paralysis the pain is more general and the tenderness is along the nerve trunks, while in rheumatism the pain and tenderness are localized in the joints. There is never any redness or swelling of the joints in infantile paralysis, while there may be in rheumatism. The chief point in the diagnosis is that there is no loss of power in rheumatism while there is in infantile paralysis. If the trouble is in the legs, the knee-jerks are diminished or absent in infantile paralysis, normal in rheumatism. There is nothing about the white count or the temperature which helps in the diagnosis. The diagnosis may be more difficult in the neuritic type, because of the rigidity which is present in some cases of this type. As already mentioned, it may be necessary to give a whiff of ether before a diagnosis is possible. Lumbar puncture will, of course, settle it, because there are no changes in the cerebrospinal fluid in rheumatism, while there are in infantile paralysis. In the types in which the cerebrum, pons, medulla or meninges are involved, the diagnosis from encephalitis and meningitis may be for a time very difficult. The diagnosis between infantile paralysis and encephalitis is discussed under encephalitis. The onset and early symptoms of tuberculous meningitis are sometimes very similar to those in the toxic type of the stage of systemic invasion of infantile paralysis and much like those of the early part of the stage of invasion of the nervous system. The findings in the cerebrospinal fluid in the two conditions are the same. The white count is always increased in infantile paralysis, while it is usually low in tuberculous meningitis. It is sometimes high in that condition, however, so that while a low white count is in favor of tuberculous meningitis, a high white count is of no importance in the diagnosis. The tuberculin test is useless, because it is often absent in tuberculous meningitis and, when present, may as well be due to some hidden tuberculous focus as to the acute disease. The finding of evidences of tuberculosis elsewhere is in favor of tuberculous meningitis, but by no means positive proof of it. The development of flaccid paralysis is much in favor of infantile paralysis, but there is a type of tuberculous meningitis in which flaccidity is a marked symptom. In tuberculous meningitis the progress of the disease is, with occasional remissions, steadily downward, while in infantile paralysis there is, after a short time, a tendency to improvement. In other types of meningitis an examination of the spinal fluid usually settles the diagnosis at once, because in them the fluid is turbid or purulent, contains a large number of polynuclear cells and almost always some, and usually many, microörganisms.

After the very acute stage is passed, if pain and tenderness persist, infantile paralysis may be confused in babies with syphilitic epiphysitis or scurvy. In syphilitic epiphysitis, however, the onset of the symptoms is usually slower, the pain and tenderness are localized at a joint and the failure to use the extremity is due, not to paralysis, but to the pain which motion causes. The onset of the symptoms is also slower in scurvy, the tenderness is over the long bones, there is usually swelling about the bones and there are usually other evidences of scurvy. Here again, the reason that the baby does not move is because it hurts, not because it cannot. The knee-jerks are present or exaggerated in scurvy, absent or diminished in infantile paralysis. In both babies and children infan-

tile paralysis at this time may be mistaken for rheumatism. The diagnosis depends, of course, on the same principles as in the very acute stage.

Still later, infantile paralysis may be confused with multiple neuritis or the pseudoparalysis of rickets. The onset of multiple neuritis is slower than that of infantile paralysis and in early life it almost always follows diphtheria. Pain and tenderness are usually much less marked in childhood in multiple neuritis than they are in infantile paralysis. The characteristics of the paralysis are, of course, the same in both cases. It is possible, but very unusual, moreover, to have in infantile paralysis a combination of paralysis of the soft palate and eye muscles with a general peripheral paralysis, which is the rule in diphtheritic paralysis. Hysterical paralysis may possibly be mistaken for infantile paralysis, but ought not to be, because in hysterical paralysis there is no paralysis. The child simply will not move its extremities, while in infantile paralysis it cannot.

Long after the acute stage, the diagnosis between obstetrical paralysis, involving the arm, and infantile paralysis may be very difficult. Obstetrical paralysis, however, dates from birth, while infantile paralysis develops later. Furthermore, while possible, it is extremely unlikely that infantile paralysis will have exactly the same distribution as obstetrical paralysis. Infantile paralysis and cerebral paralysis ought not to be confused, but sometimes are. The differential diagnosis is taken up under cerebral paralysis.

Lumbar puncture and the characteristics of the spinal fluid have been referred to repeatedly in discussing the symptomatology and differential diagnosis of infantile paralysis. It is questionable, however, if it is justifiable to do lumbar punctures simply for diagnosis when there is a question of infantile paralysis, except when the symptoms suggest that there may be some form of meningitis, other than tuberculous, or when convalescent serum is to be used in the treatment, if the condition is infantile paralysis. The reason why it is doubtful if it is justifiable to do lumbar puncture, except under these conditions, is that there is a certain danger of diminishing the resistance of the choroid plexus and favoring the passage of the virus by removing the cerebrospinal fluid, and thus, perhaps, causing an invasion of the nervous system, when otherwise it would not have taken place. Unless convalsecent serum is to be used in the treatment, it can do no possible good to determine a few hours or a day or two sooner whether a child has infantile paralysis or not. Lumbar puncture is, of course, a relatively simple and harmless procedure. Nevertheless, infection may sometimes occur and it should not be done so lightly as it often is. It seems to me absolutely wrong to do lumbar punctures on every sick child in times of epidemics, as has been so often done.

Prognosis.—It is impossible to give any general prognosis as regards life in this disease, as this depends entirely on what portion or portions of the nervous system are involved in the given case. Death almost never occurs in the meningeal and acute encephalitic types. Death, however, is the rule in the bulbar type, except when the higher centers alone are involved. Recovery may occur, however, in the most desperate cases, even when it seems impossible. Death is also likely to occur in the progressive type, whether ascending or descending, although the process may stop anywhere at any time. It is almost certain when the respiratory muscles are involved. In the other types the prognosis as to life is good. In general, it is safe to say that, if there has been no extension of the paralysis for forty-eight hours, there will not be.

In the ordinary spinal case it is certain that the final paralysis will be less than the initial paralysis. In a general way it is true that the more severe the onset and the greater the number of muscles involved, the more extensive will be the final paralysis. This is true, however, only in a general way, because certain cases in which the paralysis is general recover entirely and others in which only a few muscles are involved improve but very little. Favorable signs are early improvement and rapidity in improvement. At least 15% of the paralyzed cases recover entirely. It is impossible in the beginning to tell positively whether an individual belongs in this lucky 15% or not.

After the complete development of the paralysis, which usually occurs within a few days, there is a stationary stage, in which there is no change in the paralysis, lasting from one to six weeks. Spontaneous improvement then begins and continues for at least three months. Little spontaneous improvement can be expected after eight months. Improvement after this time is due usually to muscle training and education.

Treatment.—The virus of infantile paralysis being present in the secretions of the nasopharynx and the stools, it is evident that the patient should be quarantined and all the discharges from the nasopharynx and bowels destroyed or disinfected. As the virus may be carried by flies, the room in which the patient is quarantined should be screened. As the virus has been found in the dust of rooms in which patients with infantile paralysis have been confined, the room should be cleaned when the period of quarantine has expired. The usual period of quarantine is three weeks. This is merely an arbitrary period and at present there is no way of determining whether it is longer or shorter than is necessary. The contagiousness of infantile paralysis is so slight that there is no reason why it should not be treated in hospitals; in fact, with reasonable precautions it is safe to treat it in the open wards. The attendants and family of the patient may possibly be carriers. At present, however, it does not seem either feasible or advisable to quarantine them.

There is no justification for the hysteria which prevails in times of epidemics which, moreover, compared with the number of children in the community, are very small. It is foolish to quarantine towns and cities, close the schools, boil the milk and cut out raw fruit and vegetables from the diet. Promiscuous kissing and spitting should, however, be stopped and care should be taken that the hands are washed before eating. These are reasonable precautions, because they prevent contagion from the secretions of the nasopharynx and from the stools. There is, of course, less chance of contracting the disease in a community in which there are no cases than in one in which it is prevalent. Well children ought not, therefore, to be deliberately brought into such a community. People cannot be blamed for taking their children away from such a community, if they so desire. It is, however, usually unnecessary and the children are about as likely to be exposed during the journey away from home and in the place to which they are taken as at home.

While it is true that the virus of infantile paralysis is easily destroyed by weak solutions of peroxide of hydrogen and other weak disinfectants, it is also true that it is inadvisable to use these things as sprays and gargles with the idea of preventing infection, because they are likely to irritate the mucous membrane, weaken its resistance and destroy the protective bodies which are normally present in the nasopharynx of many people. Hexamethylenamin has no protective action It has no bactericidal

action unless it is broken up, and it is broken up only in an acid medium. It can, therefore, do no good in the mouth, which is alkaline.

It has been proved experimentally that the blood serum of monkeys and human beings that have had infantile paralysis contains immune bodies; that when such serum is mixed with the virus it protects animals from infection; and that, when given intraspinally after the virus has been introduced by other routes, it may prevent the development of the disease. The immune bodies are present in the serum from a few weeks to many years after an attack. They are probably most active in the serum between three months and five years after the attack. Normal animal and human sera contain no immune bodies and have no protective power. The injection of serum intraspinally may favor the development of the disease by breaking down the barriers of the choroid plexus. It is impossible to produce an immune serum, except in monkeys. On the basis of these experiments it seems rational to try the serum from people who have had the disease in the treatment of it. It is evident, however, that to do good during the stage of systemic infection, it must be given intravenously or subcutaneously, preferably subcutaneously. If given intraspinally at this time, it favors the invasion of the nervous system, although it may possibly neutralize the virus, if invasion of the nervous system does occur. It is evident that to do good after the invasion of the nervous system, it must be given intraspinally. It is also advisable to give it intravenously at this time, because, the barriers having been broken down, immune bodies can also pass through the choroid plexus. It is evident also that after the cord has been damaged the serum can do no good, except, perhaps, to prevent the further progress of the disease. If the serum is to do good, it must be given, therefore, as soon as the spinal fluid begins to show changes or at least as soon as there are any evidences of invasion of the nervous system. It is practically useless to give it after the development of the paralysis. Opinions differ greatly as to whether the serum does any good or not. Some observers are enthusiastic about it, others are very guarded in their expressions and others think that it does no good. In my own experience, which has been somewhat limited, I have never seen it do any good. I am rather inclined to the opinion that the dangers of weakening the resistance of the chorid plexus by repeated lumbar punctures for diagnosis and by the too early injection of serum may more than counterbalance any advantages which may possibly come from its use. If it is given intraspinally, from 15 c.cm. to 20 c.cm. of spinal fluid should be allowed to run off and not more than 15 c.cm. of serum introduced. Not more than 10 c.cm. should be given to babies. The serum must, moreover, be warmed and introduced very slowly. The dose intravenously is 20 or more c.cm. The injections may be repeated at intervals of twelve hours.

The intraspinal injection of animal sera is not only useless but dangerous, in that the foreign serum tends to break down the barriers of the choroid plexus and thus favors the invasion of the nervous system.

Solutions of adrenalin, which have been recommended on the basis that by contracting the blood vessels the extension of the process may be limited, have been proved to be useless. They may, moreover, do harm, in that they also by their action on the choroid plexus favor the invasion of the nervous system.

Hexamethylenamin cannot possibly do any good, either before or after the invasion of the nervous system. It is inert of itself and has a bactericidal action only after being broken up. This drug is broken

up only in an acid medium. The reaction of the blood and of the cerebrospinal fluid is alkaline.

It hardly seems necessary to call attention to the fact that the application of blisters and of the actual cautery to the outside of the back cannot have any effect in limiting the activity of a microöroganism within the spinal canal. Nevertheless, these things are still sometimes done. They, of course, serve only to increase the discomfort of the patient.

The treatment in the acute stage consists mainly in keeping the patient as quiet as possible both mentally and physically. It is impossible to emphasize too much the importance of quiet and rest. If the child is restless, sedatives should be given to keep it quiet. If it has pain, some form of opium in doses large enough to keep it comfortable should be given. Heat externally often helps to relieve the pain. It goes without saying, of course, that the child should be carefully fed, the bowels kept open and any symptoms which develop treated as they arise. If it cannot swallow, it should be fed through a tube.

It is extremely important to prevent the development of contractures, especially in the lower extremities. The weight of the bed clothes should be kept off of the feet by a cradle. As always, when a cradle is used, the feet and legs should be wrapped up to keep them from being cold. Toedrop can often be prevented by the use of sand bags. If there is much tendency to toedrop or drawing up of the knees, however, light posterior wire splints (Cabot splint) should be applied.

It is very important, as the child begins to improve, not to allow it to begin to do things, to sit up, to get up or to begin to walk about during the acute stage. Roughly, the acute stage lasts about six weeks. Furthermore, the acute stage cannot be considered as passed as long as there is any pain. If pain persists, it is not safe to consider the acute stage over until three weeks after the cessation of pain. During this acute stage electricity and massage, like exercise, can do nothing but harm. The object of treatment during this period is to give time for the inflammation of the nervous system to quiet down and disappear. Exercise, electricity and massage all tend to keep up the inflammation by stimulating the nervous system. Strychnia also does harm during this stage, because its action is also to stimulate the lower motor neuron, which ought not to be stimulated.

Much can be done after the acute stage is over to improve the condition of the muscles by massage, the use of electricity, muscle training and the application of suitable apparatus. The same rules apply to the use of electricity in this condition as in multiple neuritis, where they have been described. Strychnia apparently sometimes helps at this time.

It is very easy to do a great deal of harm by overtreatment during this stage. Too much massage or exercise often not only does no good, but may undo much that has been accomplished. The treatment during this stage should, therefore, be in the hands of an expert. There is seldom much spontaneous recovery after three months, although it may continue for six or eight months. Even with treatment, little muscular recovery can be expected after this time. Further improvement is usually not due to an increase of muscular power, but to better use of the muscles that are left as the result of muscle training.

Much can be done for late and neglected cases by proper muscle training, the application of apparatus and by suitable operations on the tendons, muscles and fascia.

DISEASES OF THE PERIPHERAL NERVES

MULTIPLE NEURITIS

The causes of multiple neuritis are the same in early as in adult life. Neuritis from lead pipes is less common than in the past, but occasionally neuritis develops in early childhood as the result of sucking or eating the paint off of toys, cribs or window seats. Neuritis from arsenic in wall papers and hangings occurs very seldom now, because of better health regulations, but it may still occur from arsenic used in the preservation of stuffed birds and animals or from the use of arsenic in the treatment of chorea. Alcohol is always a possible cause of neuritis in early life and may be due either to a single large dose or to the repeated ingestion of small doses. Neuritis is more often due, however, to toxic absorption in the course of various acute diseases. Practically, multiple neuritis in early life is almost always a sequela of diphtheria.

Pathology.—The lesions in multiple neuritis, depending on the cause, may be inflammatory at first, becoming degenerative later, or degenerative from the first. The lesions involve both the motor and sensory fibres. The ganglion cells of the cord are little, if at all, involved. The lesions become progressively more marked, the further away they are from the spinal centers.

Symptomatology.—The symptomatology of multiple neuritis in early life is the same as in later life. There are certain things, however, to be remembered as regards multiple neuritis. The sensory fibres of the nerves being involved, there is likely to be pain and also tenderness on pressure over the nerve trunks, as well as various subjective disturbances of sensation. Objectively, there may be anesthesia and various disturbances of sensation, which differ in different cases. The motor fibres being involved, which practically necessitates involvement of the trophic fibres, degeneration of the muscles results. There is, therefore, loss of power varying from slight paresis to complete paralysis. The paralysis is, of course, flaccid. As the result of the degeneration of the muscles, there is a more or less marked reaction of degeneration and in the severe cases the reaction to electricity is entirely lost. Partly as the result of the involvement of the nerves and partly as the result of the degeneration of the muscles, the reflexes are diminished or absent. The sphincters are never involved. The intelligence is normal. If the cause is discovered and removed, complete recovery always occurs after a longer or shorter time.

DIPHTHERITIC PARALYSIS

This is the most common form of multiple neuritis in early life and differs to a certain extent in its manifestations from the other forms. It is rather more common now than it was before the days of antitoxin, occurring roughly in about 10% of the cases of diphtheria. The reason that it is more common now than in the past is probably because many children survive now, as the result of the use of antitoxin, to have paralysis, who formerly died. That the increased frequency of paralysis is not due to the antitoxin is shown by the fact that the earlier it is given, the less frequently paralysis develops. Diphtheritic paralysis is more likely to follow severe than mild cases of diphtheria, but it may follow cases of any type. It may be, indeed, the first thing to call attention to the fact that the child has had diphtheria.

Symptomatology and Prognosis.—The onset of the paralysis may be as early as the end of the first week or in the second week. The muscles of the soft palate are those most frequently involved when the paralysis appears early. As a rule, however, the symptoms do not develop until from three to five weeks after the throat has cleared up. Involvement of the pneumogastric nerve may occur either early or late. The phrenic is seldom involved early. The distribution of the paralysis is very variable. It may be general or only a few muscles involved. These may, moreover, be widely separated. The facial muscles are seldom affected. Paralysis of the throat and diaphragm is characteristic of diphtheritic paralysis and does not occur in other forms of multiple neuritis.

In most instances it is the soft palate which is first involved. This is shown by a nasal voice and by the regurgitation of liquids through the nose. The paralysis may extend to the muscles of the pharynx and of the larynx, making swallowing difficult or impossible and causing weakness of the voice or aphonia. In some cases the paralysis may be limited to the muscles of the throat. As a rule, however, other muscles are also involved. In other instances the eyes are involved, sometimes first. The ciliary muscles are the ones usually involved, with resulting paralysis of accommodation. The external muscles of the eye are less frequently attacked. There is very likely to be more or less marked paralysis of the extremities. The legs are involved more often and are usually more seriously affected than the arms. The paralysis is flaccid and there is atrophy of the muscles. Toedrop is usually marked. The reaction of degeneration is present. The knee-jerks are always diminished and often absent when the legs are involved. They are also almost always diminished or absent even when there are no other evidences of involvement of the legs. Subjective sensory disturbances are less common and less marked than in other forms of multiple neuritis. Pain is uncommon, but there is occasionally paresthesia. Various disturbances of sensation may be detected objectively.

There is always danger of the development of inhalation bronchopneumonia, when there is paralysis of the muscles of deglutition. If this does not occur, recovery always takes place. Recovery always takes place when the eye muscles are involved, but it may be many weeks and occasionally many months before vision is again normal.

Paralysis of the muscles of respiration may occur and is always very serious. Either the intercostal muscles, the diaphragm or both may be involved. Usually it is the diaphragm. This is ordinarily involved, however, only in cases in which the paralysis is widely distributed. It is shown by attacks of dyspnea and cough. In these attacks the respiration is thoracic in type, rapid, irregular and shallow. Cyanosis is marked and there is much distress. These attacks are repeated every few hours and are likely to increase progressively in severity. Death occurs in an attack in about 25%.

When the pneumogastric nerve is involved, the pulse becomes irregular or intermittent. It sometimes is slow, but more often fast. The first heart sound is usually somewhat feeble. Other symptoms of involvement of the pneumogastric nerve are pallor, cold extremities, restlessness, anxiety, precordial distress, dyspnea and vomiting. Vomiting may either accompany or precede the symptoms pointing directly to the heart. Death may occur inside of the first twenty-four hours after the onset of severe symptoms or not for several days. It is quite likely to occur in these cases as the result of sudden exertion, like sitting up in

bed, or getting out of bed. In other cases sudden death may occur after a slight strain without there having been any previous symptoms, except, perhaps, slight irregularity or change in the rate of the pulse. In other instances, death occurs more slowly as the result of progressive cardiac failure. Recovery occasionally occurs. I am inclined to think, however, that in many instances the symptoms which are attributed to pneumogastric involvement are really due to myocarditis or, perhaps, to a combination of the two. I have, nevertheless, never been able to distinguish between the symptoms due to pneumogastric involvement and myocarditis.

Diagnosis.—Paralysis of the extremities developing gradually and reaching its maximum in a few weeks in a child is almost certainly diphtheritic. If there is also paralysis of the soft palate or of the eye muscles, it is unquestionably diphtheritic. The history of a sore throat or of a "cold" several weeks previously corroborates the diagnosis. It is true that infantile paralysis may have the same distribution, even including paralysis of the palate or of the eye muscles, and in both the paralysis is flaccid. The onset of infantile paralysis is, however, much more rapid and, while the palate and the eye muscles may be involved as well as the extremities, they very seldom are, because other cranial nuclei would have been involved as well and have caused death. When the paralysis is limited to the muscles of the palate or of the eyes, it is conceivable that it may be due to a tumor in the pons or to infantile paralysis. It is very improbable, however, that these muscles would be involved alone under these circumstances. Localized paralysis of the palate or of the eye muscles in a child, therefore, is almost pathognomonic of diphtheritic paralysis.

Treatment.—The treatment of diphtheritic paralysis is preventive. When the Schick test is universally employed and children rendered immune to diphtheria by injections of toxin-antitoxin, there will be no diphtheritic paralysis. If diphtheria is recognized early and sufficient doses of antitoxin given, diphtheritic paralysis will never develop.

The most important element in the treatment of paralysis of the extremities is rest in bed until the paralysis has practically disappeared. Light splints should be applied in the severe cases to prevent contractures. It is especially important to prevent toedrop and contractures at the knees, as it often takes longer to correct these deformities, if they have developed, than it does to get over the paralysis. It is also important to keep the affected extremities warm. Regular massage is of advantage. It must be remembered, however, that all that massage does is to keep up the nutrition and tone of the muscles. It has no effect whatever on the neuritis. Electricity may be used for the same purpose, that is, to keep up the nutrition of the muscles. In using electricity it must be remembered, however, that the muscles and nerves are both degenerated. If there is much degeneration, faradic electricity does as much good when applied to the bed as it does when applied to the affected extremities. Galvanism should, therefore, be used, unless there is a definite response of the muscles to faradism. When the muscle is sufficiently regenerated, faradism should be used. It is customary in these cases to give strychnia either by mouth or subcutaneously. It seems to me doubtful, however, whether it does any good, because it is hard for me to see how strychnia can stimulate a degenerated muscle through a nerve which is unable to carry normal stimuli.

There is nothing to be done for the eyes except to rest them. Much harm can be done, however, by attempting to use them too early. When there is much trouble with swallowing, the food should be given with a tube, introduced preferably through the mouth rather than through the nose.

There is little, if anything, which does any good when there is respiratory paralysis. Artificial respiration may help tide over an emergency. Strychnia, subcutaneously, is strongly recommended for this condition. The same criticism, however, holds as in the case of paralysis of the extremities. If it is given, the dose for a child of two years is $\frac{1}{240}$ of a grain and for a child of four years $\frac{1}{120}$ of a grain. Faradization of the diaphragm is useless, because the degenerated muscle cannot respond to faradism.

When there is pneumogastric paralysis, the all important thing in treatment is rest. The child must be kept absolutely flat in bed and not allowed to move hand or foot. Everything must be done for it. If it does not keep still or is restless and uncomfortable, it must be quieted by the use of bromides or some form of opium. Sufficient doses must be given to keep it still, however much is required. Cardiac failure may be treated as in other conditions. Little, however, can be expected from such treatment.

FACIAL PARALYSIS

Facial paralysis may occur occasionally in early as in adult life as a part of a hemiplegia. In such cases the upper branch of the nerve is not involved and the child can close its eye and wrinkle its forehead. There is, moreover, no reaction of degeneration. The reason that the upper branch of the nerve is not involved is because the nerve receives fibres from both hemispheres. Paralysis of the face may sometimes be associated with paralysis of the arm and leg on the other side. In such cases there is a lesion in the pons, usually a tumor. In such cases all the branches of the nerve are usually involved, because the lesion is almost always below the decussation of the facial nerves. Other cranial nerves are also almost always involved. The facial nerve may also be involved in meningitis, perhaps more often in tuberculous than in the other forms. When the trunk of the nerve is involved in its passage through the temporal bone or after its passage through the stylomastoid foramen, all the branches are involved. The cause of the involvement of the nerve in its passage through the temporal bone is otitis media or one of its complications. The usual cause of involvement after it leaves the bone is exposure to cold. It sometimes is involved, however, in mumps and may be injured by traumatism. It is possible, of course, to determine just what part of the trunk of the nerve is involved by study of the taste, the salivary secretion, the hearing and the soft palate. It is so difficult to determine these points, however, in children that it is hardly worth while. Furthermore, it makes no difference in the treatment.

When all the branches of the nerve are involved, the child cannot close the eye or wrinkle the forehead on the affected side. It cannot pull up its nose and, when it attempts to whistle or to pucker up its lips, there is diminished action on the affected side. When it attempts to show the teeth, laughs or crys, the mouth is pulled towards the sound side. There is a reaction of degeneration. There are no disturbances of sensation, as the facial nerve is purely motor.

Prognosis.—The prognosis of facial paralysis depends very largely on the cause. When it is due to a cerebral lesion high up, the chances are that it will finally disappear. When it is due to a lesion in the pons, it is a minor matter, because the causative lesion is almost certain to cause death. When it is due to meningococcus meningitis, it is likely to be permanent. It is also quite likely to persist, if it is due to an injury to the nerve in its passage through the temporal bone. When it is due to disease of the nerve after it leaves the bone, complete recovery almost always takes place.

Treatment.—There is little to be done in the way of treatment. It is important to keep that side of the face warm and to avoid exposure to cold and wind. Massage and electricity may be used in the same way as in the treatment of diphtheritic paralysis. It is very doubtful if any of the preparations of salicylic acid do any good, although they may be used if the physician believes in them. External applications are valueless.

SECTION XVII

DISEASES OF THE GLANDS OF INTERNAL SECRETION

There is not only much difference of opinion as to what organs have an internal secretion but also as to what an internal secretion really is. Of those organs which without much question have an internal secretion, the only ones which are of much importance to the pediatrist are the thymus, thyroid, pancreas and pituitary. Lesions of the adrenals, parathyroids and the pineal are so uncommon that they are of little importance. The knowledge of the functions of these glands is very limited, except, perhaps, in the case of the thyroid and pancreas. Nevertheless, on the basis of this very limited knowledge all sorts of suppositions have been formed and theories advanced, which have been accepted by many as proven facts. Practically nothing is known as to the relations of these glands to each other and as to the interaction of their secretions. Nevertheless, here again, all sorts of theories have been advanced and accepted as facts. On this flimsy and entirely insufficient basis of suppositions and theories a method of treatment has been built up, which can, of course, be no better than the foundation upon which it is erected. There are a few instances in which our knowledge is sufficient to justify the removal of a part of one of these glands or the administration of the extract of that gland. In most of the instances in which one of these extracts is given, however, the evidence is not sufficient to show that it is needed and the knowledge of what it does is insufficient to justify its use. In the present state of our knowledge, or lack of knowledge, it is absolutely irrational to give a mixture of glandular extracts, pluriglandular therapy, because no one knows just what each gland does or what each does to the other. I should expect to know as much as to what would happen if I threw a monkey-wrench among the wheels of the universe as I would if I gave a child a combination of glandular extracts. Physicians must be careful that in their enthusiasm in the use of glandular extracts they do not do more harm than good. They must also be careful that they do not use glandular therapy, as Mr. Dooley said of Christian Science, as "another way of getting money."

Diseases of the Thymus

The thymus gland is situated behind the manubrium and in front of the trachea, great vessels and other structures which fill the superior entrance of the thorax. The anteroposterior diameter of this space is two cm. Extension of the head shortens this diameter.

The thymus gland at birth is between four and five cm. long, 1.5 and 2.5 cm. wide and 0.8 and 1.4 cm. thick. The average weight at birth and during the first two years is six grammes. Anything over ten grammes is pathologic. There is not much change in the size of the gland during the first two years. After this time it gradually diminishes in size and, as a rule, only vestiges remain after puberty.

The thymus gland is made up of two lobes, which are attached above and separate below. Its shape is roughly that of a half opened pea pod.

Physical Examination.—It is said that the thymus gland gives a triangular area of dullness under the manubrium, the base being at the top of the manubrium and the apex at the lower end of the manubrium a little to the left of the mediam line. This area is said also to extend a little into the first left space. I personally am not able to make out, even with light percussion, any dullness in this area when the gland is normal. Dullness in this situation in my hands, therefore, means enlargement of the thymus. Dullness in the second space on either side of the manubrium is also quite characteristic of enlargement of the thymus. If it is much enlarged, it can be felt in the suprasternal notch.

With the Roentgen ray the shadow of the normal thymus is continuous with that of the heart, being directly above it. When the thymus gland is enlarged, the shape of the shadow may remain the same, but be considerably enlarged, or there may be a columnar widening of the shadow about the great vessels or a bulbous or globular widening of the shadow.

Function of the Thymus.—Very little is really known as to the function of the thymus. It is possible that it produces some sort of an internal secretion which supplies the deficiency of those of the reproductive organs up to the time when they are fully developed. If so, this internal secretion has nothing to do with the development of the secondary sex characteristics. Whether it bears any relation to the other glands of internal secretion or not is unknown.

ENLARGEMENT OF THE THYMUS

Enlargement of the thymus may occur as the result of new growths in it, syphilis, tuberculosis and the formation of cysts or abscesses. It may also enlarge in the course of acute diseases. Enlargement of the thymus from these causes is relatively unimportant. The most common and important cause of enlargement of the thymus is simple hyperplasia. Nothing is known as the cause of this hyperplasia. It may be accompanied by congestion. The congestion may be either acute or chronic.

Enlargement of the thymus, on account of its anatomic relations, causes pressure on the other important organs which are located in the superior entrance of the thorax. The arteries are so stiff that they resist the pressure. The nerves are usually displaced and, therefore, avoid pressure. The veins are sometimes compressed, but they, like the nerves, often slip to one side. It is the trachea which bears the brunt of the pressure. The symptoms of pressure on the trachea when there is chronic enlargment of the thymus are noisy inspiration and expiration, dyspnea, retraction of the intercostal spaces and cyanosis. The cyanosis may be due either to pressure on the veins, imperfect oxygenation of the blood, as the result of the interference with respiration, or a combination of the two. Physical examination shows an increased area of dullness under the manubrium, a palpable tumor in the suprasternal notch and an increased shadow with the Roentgen ray. The larynx is not depressed during inspiration, because it is kept up by the enlarged thymus. Extension of the head increases the symptoms. Other important negative signs are that the voice is clear and that intubation does not relieve the symptoms.

Diagnosis.—The symptoms due to chronic enlargement of the thymus may be confused with those due to congenital stridor, tracheobronchial adenitis, retropharyngeal abscess and catarrhal or diphtheritic laryngitis. In congenital stridor the noise is made entirely during inspiration. There are no symptoms of interference with breathing, the larynx moves

during respiration, and there are no physical signs of enlargement of the thymus, while in enlargement of the thymus the noise is made during both inspiration and expiration, there are symptoms of interference with breathing, the larynx does not move and the physical signs of enlargement of the thymus are present. In bronchial adenitis the noise may be present in both inspiration and expiration, but is more marked in expiration, because the diameter of the thorax is diminished during expiration. The evidences of enlargement of the tracheobronchial glands are present and those of enlargement of the thymus absent. A retropharyngeal abscess may cause both noisy inspiration and expiration and also evidences of interference with respiration. Examination, however, shows the abscess in the throat while, when the symptoms are caused by enlargement of the thymus, the larynx does not move and the physical signs of enlargement of the thymus are present. In both catarrhal and diphtheritic laryngitis the onset is relatively acute, the difficulty in breathing is entirely in inspiration, the voice is hoarse or lost, the larynx moves more than usual and there are no evidences of enlargement of the thymus. The diagnosis from enlargement of the thymus is, therefore, easy.

Sudden exacerbations of the symptoms occur not infrequently in chronic cases as the result of extension of the head, crying and excitement, all of which cause congestion in the thymus. Exacerbations may also occur without any evident cause, presumably also as the result of congestion from some unknown cause. When these sudden exacerbations occur, especially if there have been few or no symptoms from the enlargement previously, the symptoms may be confused with those of laryngismus stridulus and asthma. In laryngismus stridulus the attack begins with a cry, several short breaths are taken and then respiration stops in full inspiration. In acute congestion of the thymus the interference is both with inspiration and expiration. Breathing may stop in either phase and there is no initial cry. The spasm lets up in laryngismus stridulus as the result of carbonic dioxide poisoning and the baby quickly recovers, while in congestion of the thymus it rarely ceases soon enough to prevent death. Acute congestion of the thymus ought not to be confused with asthma, because the onset of dyspnea is slower in asthma and the difficulty is entirely in expiration.

Prognosis.—Chronic hyperplasia of the thymus is a very serious and frequently fatal condition. Death may occur slowly as the result of exhaustion from the interference with the breathing, but more often occurs in an acute exacerbation from congestion. When death occurs, respiration stops before the heart. An acute attack of congestion may cause death without there having been any previous symptoms of the chronic enlargement. Fortunately, enlargement of the thymus, if recognized, can be cured by proper treatment with the Roentgen ray.

Treatment.—Shrinking of the thymus is caused by proper application of the Roentgen ray. All cases in which there are any symptoms from enlargement of the thymus should, therefore, be treated in this way. It is questionable if it is necessary to treat enlargements of the thymus, discovered accidentally with the Roentgen ray, which cause no symptoms and are not easily demonstrable by physical examination.

STATUS LYMPHATICUS

Infants, children and adults sometimes die suddenly without any apparent cause or from some cause entirely insufficient to account for

death, as at the beginning of anesthesia, from some sudden shock, the introduction of a needle or in the course of some mild disease. In such cases enlargement of the thymus, spleen, lymph nodes, tonsils and Peyer's patches, hyperplasia of the bone marrow and hypoplasia of the heart and aorta are usually found. Persons who die in this way are often of the pale, pretty and flabby type. The pathologic combination described above is spoken of as the status lymphaticus. When this condition is found, when death has occurred suddenly without apparently sufficient cause, the death is said to be due to the status lymphaticus.

There are various theories as to the etiology of this condition and as to the cause of death. It has been supposed to be connected primarily in some way with the thymus. It is not known, however, whether an excessive secretion by the thymus, as the result of hypertrophy, is the cause of the changes in the other organs and of the fatal termination in this condition or whether the enlargement of the thymus and the changes in the other organs are due to some common, unknown cause. If they are, the secretion of the thymus, hypothetical at best, presumably has nothing to do either with the symptoms or with the course of the disease.

Symptomatology.—Unfortunately, there is no positive way of recognizing the existence of this condition in advance. Even if there was nothing could be done about it. It is presumable that many persons live out their allotted span of life in spite of this condition. When death occurs as the result of this condition, it does not always occur suddenly. The symptoms may sometimes persist for days or even weeks, but the termination in all is fatal. The most characteristic symptoms are illness out of proportion to the physical signs, peculiar disturbances of the respiration and convulsions. These symptoms develop in the course of other diseases. The child is much sicker than it apparently ought to be. The respiration is usually very rapid, often gasping, and often associated with marked cyanosis. There is sometimes an incessant cough. The dyspnea is all out of proportion to the physical signs, which usually consist of a few scattered râles. It may be constant or occur in attacks. The dyspnea is not like that due to obstruction within the air passages or to pressure from the outside. It is more like that in acid intoxication, but is more rapid. When the dyspnea is constant, death usually occurs within forty-eight hours; when it is intermittent, it may be delayed for some days or weeks. The convulsions may occur at any time and are in no way characteristic.

Diseases of the Thyroid Gland

The internal secretion of the thyroid aids growth and stimulates the metabolic processes of the body, chiefly the katabolic. It is doubtful whether it acts directly on the tissues themselves or indirectly through the nervous system. Iodine apparently is of service as a catalyser in accelerating the metabolic processes. It is probable that the iodine is combined in some way in the thyroid into a compound suitable for the performance of this function.

When the thyroid is insufficient, the pituitary hypertrophies. The thyroid also has considerable influence on the growth and development of the reproductive organs in early life. It is possible that its secretion stimulates chromaphil tissue, and there may be, perhaps, some relation between the thyroid and the thymus and pancreas.

GOITER

Simple goiter, that is, enlargement of the thyroid gland, includes the varieties formerly known as endemic, sporadic and physiologic. The increase in size is due chiefly to an overgrowth of the colloid material, but an adenomatous form is not uncommon. It may be present at birth or develop at any time during childhood. It is much more common in girls than in boys and is especially likely to develop in them at about the time of puberty. The sporadic and endemic forms are due to an insufficiency of iodine in the food and water. Simple goiter is very common in children, especially in girls, in the country to the south of the Great Lakes and in that part of the Northwest where the water from the Cascade Mountains is used for drinking purposes. The cause of the physiologic form, occurring at puberty, is unknown. There are no symptoms of either an excess or a deficiency of the thyroid secretion. The tumor is the only manifestation.

Experience has shown that the administration of minute doses of iodine prevents the development of goiter in children in those districts in which it is common. Congenital goiter can also be prevented by giving iodine to the mother during the first half of pregnancy. It has been proved that the administration of thirty grains of sodium iodide, in divided doses, in two weeks, each spring and autumn, is more than sufficient to prevent the development of goiter in regions where it is endemic and that the same amount given during the first half of pregnancy prevents the development of goiter in the fetus. It is very important to give iodine in extremely small doses, when the object is to prevent the development of goiter. Iodine given in minute doses over a long period also cures simple goiter in children in the great majority of instances. The physiologic enlargement of the thyroid, which appears at about the time of puberty, requires no treatment, unless the child lives in a community in which goiter is endemic.

EXOPHTHALMIC GOITER

Exophthalmic goiter is extremely rare in childhood. The symptoms and physical signs are the same as in adult life. Drugs are useless, as in adults. The results from operation are better than those obtained with radium and the Roentgen ray.

CRETINISM

Cretinism is due to a marked insufficiency of the secretion of the thyroid gland. It may be either endemic or sporadic. The form seen in this country is almost invariably the sporadic. Endemic cretinism is usually associated with goiter. In sporadic cretinism the thyroid gland is usually almost, if not entirely, absent, although in rare instances there may be a goiter. Nothing is known as to the etiology of sporadic cretinism. It probably should be looked upon as simply an error in development.

It is usually said that the symptoms of sporadic cretinism do not appear until the second half of the first year. This is not true. They can be detected much earlier than this, if they are looked for. They are not as marked at first as they are later, because the infant is protected for a time by the thyroid secretion which it received from its mother. The earliest symptoms are dullness and delay in development, both mentally and physically. The babies do not show a normal interest in

their surroundings and are quieter than the average baby. In a short time they begin to show the physical evidences of the disease, which, of course, become more marked as they grow older.

The appearance of the fully developed cretin is very characteristic. It is short and thick. The head looks large. The anterior fontanelle is large, the bridge of the nose is sunken, the space between the eyes is wide, the mouth is open, the tongue is large and thick and sometimes fissured. It usually protrudes a little beyond the gums or teeth. Dentition is delayed and when the teeth have erupted they quickly decay. The hair is sparse, coarse and dry. The neck is short and thick. The thyroid is usually not palpable. Supraclavicular pads are uncommon in children, but are sometimes seen. The abdomen is considerably enlarged and pendulous. There is usually a very large umbilical hernia. The extremities are

FIG. 168.—Cretinism in infant.

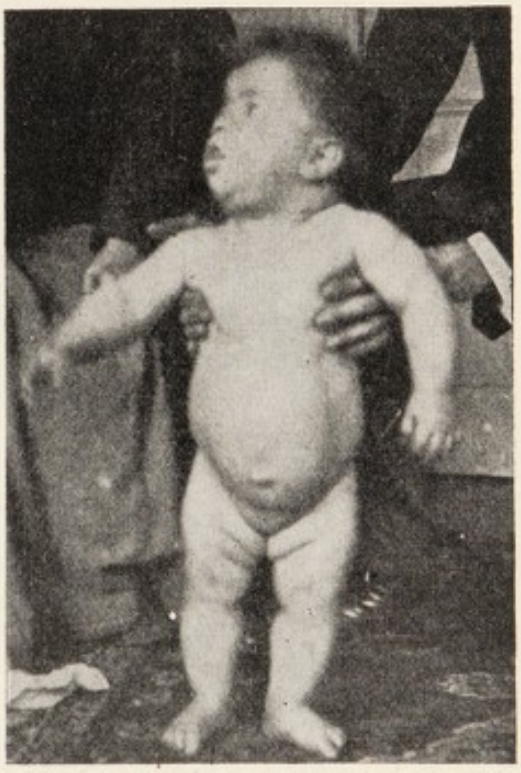

FIG. 169.—Cretin of two and one half years.

much shorter, compared with the body length, than normal. The hands and feet are short and thick. The fingers and toes are stubby. The skin is pale and dry. The skin and subcutaneous tissues are thickened and porky. They do not pit on pressure. The extremities are usually cold and often blue. The secretion of the sweat glands is markedly deficient. The temperature is subnormal and the pulse slow.

The Roentgen ray shows marked delay in the ossification of the bones at the epiphyses and at the centers of ossification. There is marked delay in growth at the epiphyseal lines.

Cretins are all feebleminded, usually to a marked degree. They are, however, usually happy and pleasant. Development is very slow. Walking may be delayed until the eighth or tenth year and the second dentition may not occur before adult life. The condition is not incompatible with long life.

Diagnosis.—The diagnosis of fully developed cretinism is very easy. No one who has ever seen a case, or even a picture of a case, can miss it. The diagnosis in the early months of life, however, is more difficult, but the condition should not be overlooked even then, if the possibility of its existence is kept in mind. It is sometimes confused with Mongolian idiocy and chondrodystrophy. In Mongolian idiocy, however, the enlargement of the tongue does not appear for several years. The eyes slant inward and downward. There is a marked epicanthic fold, the shortening of the extremities is much less and there are no evidences of myxedema. In chondrodystrophy the lack of growth is

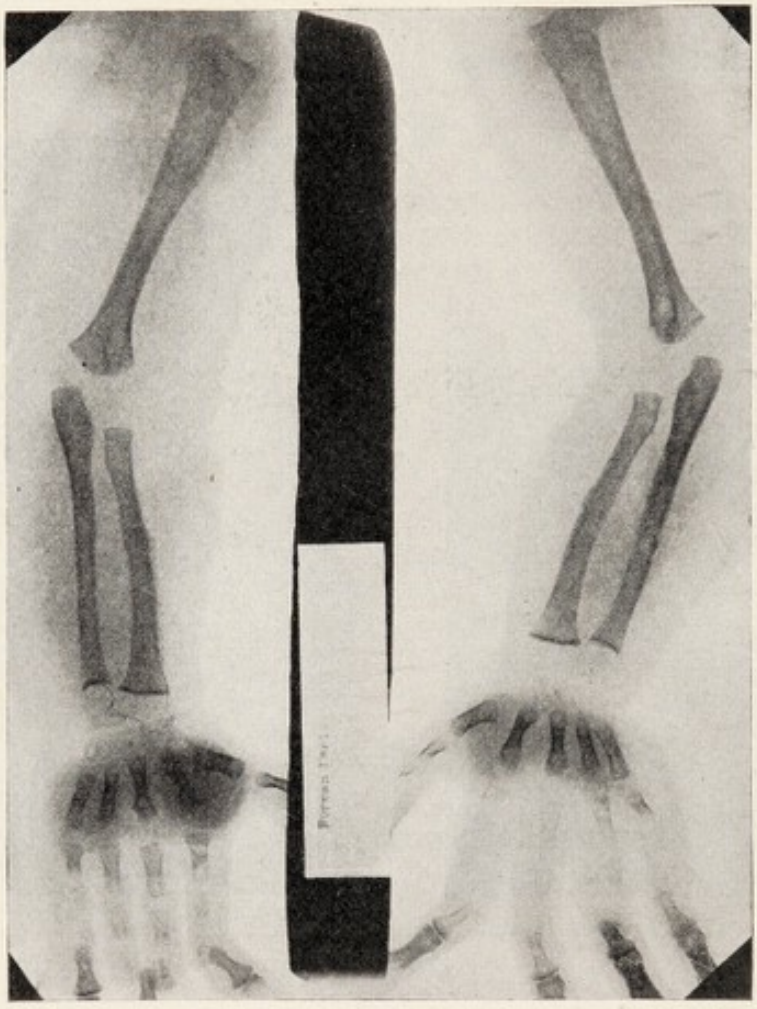

FIG. 170.—Bones of cretin of one year. Shows delay in ossification.

entirely in the extremities. The intelligence is normal, there are no evidences of myxedema, the tongue is normal and the condition in the bones, shown by the Roentgen ray, is entirely different from that in cretinism. There are many other minor differences between Mongolian idiocy and chondrodystrophy and cretinism, which might be mentioned here. Those just given are, however, enough to prevent mistakes in diagnosis.

Prognosis.—There is no hope of either improvement or recovery in cretinism, unless it is treated with thyroid extract. When it is, improvement is almost miraculous. Unless the treatment is begun before the baby is a year old, however, it must not be expected that the child will develop to be a normal adult. Even then, the chances are that it will

not be quite normal. I started treatment on a baby, six months old, twenty-seven years ago. He has had thorough treatment all these years. Physically he is short and thick set, but no one would recognize him as a cretin. He was unable to get beyond the grammar school, but played on both the school football and baseball teams. He is unable to do anything requiring more intelligence than manual labor. The other children in the family have been unusually intelligent mentally.

Treatment.—The initial dose of thyroid extract for a baby is one half grain, three times daily. The dose should be increased until the baby shows evidences of hyperthyroidism. These are restlessness, rapid pulse, fever and diarrhea. The dose should then be reduced to and kept at an amount just below that which produces toxic symptoms. It goes without saying, of course, that thyroid extract must be taken regularly as long as the patient lives.

FIG. 171.—Results of treatment in sporadic cretinism. The boy on the right is thirteen years old and the oldest of the three brothers. He has had thyroid treatment since he was six months old.

HYPOTHYROIDISM AND HYPERTHYROIDISM

If the thyroid gland is injured in any way during childhood so that the secretion is diminished, there is delay in growth and in mental development and the other manifestations of thyroid insufficiency, shown at their maximum in cretinism, appear to a greater or less degree, according to the degree of insufficiency. If there is little or no secretion, the condition is known as infantile myxedema. If there is but little insufficiency, it may be very difficult to recognize the symptoms. When there is a possibility that delay in growth and in development may be due to an insufficiency of the thyroid, thyroid extract should be given. If the symptoms are due to thyroid insufficiency, relief will be very marked and immediate. It must not be forgotten, however, that thyroid extract is a general metabolic stimulant and that, even when feeblemindedness is due to other causes, there may be a slight and temporary improvement when thyroid extract is given. Infantile myxedema responds to treatment with thyroid extract in the same way as myxedema in the adult.

The symptoms of hyperthyroidism are similar to those of exophthalmic goiter, but much less marked in degree. They are excitability,

nervousness and excessive perspiration. Sometimes the pulse is rapid and there is a tendency to looseness of the bowels. These symptoms are especially likely to appear in girls at about the time of puberty. The treatment consists in regulation of the life and hygiene. Drugs are useless.

Diseases of the Pituitary Gland

The pituitary body is composed of three parts, the anterior lobe, the intermediate portion and the posterior lobe. The anterior lobe is glandular in structure. The intermediate portion is usually grouped with the posterior lobe, which is nervous in origin. The functions of the lobes are entirely different and it is possible that that of the intermediate portion may be different from that of the posterior lobe, with which it is usually grouped. The anterior lobe is concerned with tissue growth, especially that of the skeleton and with sex development; the posterior lobe with the metabolic processes. It is possible that the pars intermedia may be especially concerned with the secretion of urine and the carbohydrate tolerance. It is also possible that the pituitary body may have nothing to do with these matters and that they are controlled by the tuber cinereum.

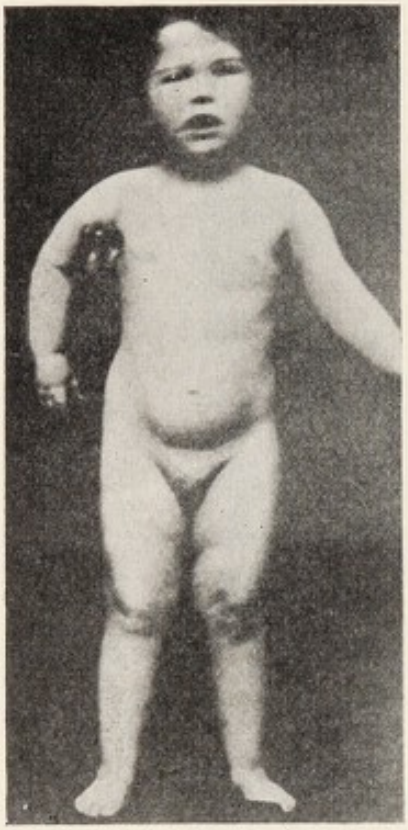

Fig. 172.—Hypopituitarism. Fröhlich's syndrome.

If the anterior lobe is overactive, there is an overgrowth of the body as a whole, most marked in the bones, and an excitatory affect on the reproductive organs, shown by an early and excessive development of the secondary sex characteristics. This is shown in early life by the development of various sorts of *gigantism*. The development is usually of the masculine type. The external genitals are normal or somewhat overdeveloped and the secondary sex characteristics are exaggerated.

If there is an insufficiency of the anterior lobe, skeletal growth and the development of the secondary sex characteristics are to a greater or less extent inhibited. The most characteristic manifestation of this insufficiency in early life is the *Lorain-Levi* type of *infantilism*. In this type the stature is below normal and the build is slight, but the proportions are like those of the adult. The external genitals are infantile and the secondary sex characteristics are not developed. There is also hypoplasia of the heart and arteries.

When there is an insufficiency of the secretion of the posterior lobe all the metabolic processes are slowed. There is a tendency to drowsiness. The pulse and respiration are slow, the blood pressure low and the temperature subnormal. The resulting condition is often described as hibernation. On account of the high carbohydrate tolerance, the fat tissue is increased. It is presumable that, if the posterior lobe is overactive, the metabolic processes are stimulated and an opposite set of symptoms produced.

It is reasonable to suppose that all sorts of combinations of increase and diminution in the activity of the two portions of the pituitary body may occur, giving rise to all sorts of combinations of symptoms. In general, however, the tendency in all disturbances of the secretions of the pituitary body is to finally lapse into insufficiency. The most characteristic example of insufficiency of both parts of the pituitary body in childhood is *Fröhlich's syndrome*—dystrophia or degeneratio adiposogenitalis. In this condition the child becomes very fat, the accumulation of fat taking place especially in the abdomen, buttocks and thighs. The genital organs are infantile and the secondary sex characteristics do not develop. The skin is thin, soft and smooth. There may or may not be an increased sugar tolerance. This disease is not due to overeating. In fact, the appetite is usually not very large. Dieting, moreover, has very little effect on the weight.

It is also reasonable to suppose that there is an interrelation between the secretions of the pituitary body and the other glands of internal secretion. The only gland with which such a connection has been definitely proven, however, is the thyroid.

Diagnosis.—It is reasonably safe to make a diagnosis of disturbance of the functions of the pituitary body when Fröhlich's syndrome is encountered and in certain cases of gigantism and infantilism, especially if of the Lorain-Levi type. It is very tempting to make a diagnosis of pituitary disease whenever a child is unusually fat or large, smaller than the average, late in sexual development or slow mentally. It must not be forgotten, however, that children are fat from other causes, that heredity has much to do with size and mental capacity and that there are many other reasons for delay in development, whether it be physical, mental or sexual. The size of the sella turcica is of no importance in the diagnosis of pituitary disease. It varies tremendously normally in childhood, as has been repeatedly shown. Furthermore, there is no relationship between the size of the pituitary body and the amount of its secretion, even if the size of the sella turcica indicates the size of the pituitary body, which is very doubtful. The sugar tolerance is very variable, not only when the pituitary body is diseased, but also under other conditions. Furthermore, it is doubtful if it is controlled by the pituitary body anyway. Finally, it has been shown clinically to be of little or no value in the diagnosis of pituitary disease. It is, therefore, in my opinion, unjustifiable to make a positive diagnosis of pituitary disease in childhood unless such characteristic syndromes as Fröhlich's and the Lorain-Levi type of infantilism are evident.

Prognosis and Treatment.—No physiologically active substances have been extracted from the anterior lobe. Extracts from the posterior lobe have a powerful physiologic action. None of the extracts from the glands of internal secretion have been proven to have any action when taken by the mouth, except that from the thyroid. The only preparation of the pituitary body which can reasonably be expected to do any good in the treatment of disease of the pituitary body is, therefore, the extract of the posterior lobe, pituitrin. This must be given subcutaneously and, moreover, can even then be expected to be of benefit only when there is a deficiency of the secretion of the posterior lobe. Pituitrin is worthy of a trial under these conditions. It is unwise, however, to expect much improvement from its use. Little more can be hoped for from surgery. The removal of an organ which is causing trouble because it is not doing its work, which is the usual difficulty with the

pituitary body, can do no good. The removal of a tumor which is preventing the pituitary from doing its work or of a portion of the gland, if it is hyperactive, may be of benefit. Operations on the pituitary glands are, however, very serious and often fatal. They should not be undertaken without careful consideration and should only be attempted by men especially trained in brain surgery.

DIABETES INSIPIDUS

Diabetes insipidus is a chronic disease characterized by the passage of large amounts of urine of low specific gravity.

Etiology.—This very rare disease is quite likely to begin in early life, sometimes even in infancy. It is more common in boys than in girls and is said to be often hereditary. The onset sometimes follows some acute disease or an injury to the head. It is not accompanied by any other evidences of disease of the kidneys. All these facts are of interest, but show nothing as to the real causation of the disease. In certain cases, however, polyuria of this type is associated with manifestations of disease of the pituitary body and disease of the pituitary body has been found at operation or at autopsy. The evidence is very conflicting as to what part, if any, of the pituitary body is at fault and whether the difficulty is hypersecretion or hyposecretion. At present what evidence there is seems to point to a hyposecretion of the pars intermedia. It is very possible, however, that the pituitary body has nothing to do with the etiology of diabetes insipidus and that it is due, as some believe, to disease of the tuber cinereum. However, as pituitrin apparently relieves the symptoms in some cases, apparently diabetes insipidus, it may be as well to describe this condition under diseases of the pituitary body as anywhere else.

Symptomatology and Prognosis.—The onset of the symptoms may be either slow or rapid, but is usually slow. The two marked symptoms are thirst and polyuria. The following extract from my records, concerning a girl of five years, shows how excessive the thirst may be:

"Gets up to drink two or three times in the night. Drinks two or three glasses at a time. Drinks every time she passes the faucet during the day, from two to four glasses at a time. Great distress, skin hot and dry, lips dry and cracked, if not allowed to drink. These symptoms will develop inside of two hours."

Another one of my patients, a boy of eight, passed as much as five quarts of urine, and another of thirteen, from twelve to fourteen quarts daily. In most instances the bladder becomes enlarged and considerable amounts of urine are retained without discomfort. A quart in the bladder of the boy of eight, already mentioned, did not annoy him at all. In some instances, however, there is incontinence. The skin is usually dry and the perspiration scant. The general condition is usually not much disturbed and gain in weight may be fairly normal. The appetite is not voracious. Digestion is usually not disturbed, but in some instances there are repeated upsets. The bowels are regular, if the child is allowed sufficient liquid. Sleep is disturbed because the patient has to get up repeatedly to pass water. In other instances, however, the general condition is disturbed, growth is not satisfactory and the child is below par nervously and physically. There is nothing about the condition incompatible with many years of life. Improvement and recovery occur in some cases. Death is usually due to some intercurrent disease.

The urine is pale, clear, of low specific gravity, 1.002 to 1.008, and contains neither albumin nor sugar. The sediment shows no evidences of disease of the kidneys. The total solids are normal. The kidneys are unable to concentrate the urine, that is, the specific gravity is not changed when salt and nitrogen are added to the diet.

In some instances there is an increased sugar tolerance, in others not. If there is an increased sugar tolerance, it is simply a coincidence, perhaps due to the same cause, not a symptom of the disease.

Diagnosis.—Diabetes insipidus, before the case is carefully studied, may be confused with diabetes mellitus, chronic interstitial nephritis and simple polyuria. In diabetes mellitus the specific gravity of the urine is high and the urine contains sugar, while in diabetes insipidus the specific gravity is low and the urine does not contain sugar. Chronic interstitial nephritis is very rare in childhood. The urine contains albumin and casts and the blood pressure is increased. In diabetes insipidus the urine does not contain albumin and casts and the blood pressure is low. In simple polyuria the amount of urine diminishes when the intake of liquids is diminished and there is no great discomfort from the lack of fluid. In diabetes insipidus the amount of urine continues large when the fluids are cut down and there is great suffering from the lack of fluids. There is no fixation of gravity in polyuria; there is in diabetes insipidus. It is useless to take Roentgenograms of the sella turcica or to try the sugar tolerance test, as both are unreliable and, furthermore, unnecessary.

Treatment.—It is usually possible to cut down the fluids a little without causing much discomfort. They should not be cut down, if discomfort is caused, because diminishing the amount of urine does no good, except to diminish the nuisance of passing urine so frequently. There are no special indications as to the diet. It should be one suited to the age and digestive capacity of the individual child. The general hygiene should, of course, be looked after. There are no drugs, with the possible exception of pituitrin, which have any effect on the symptoms. In some instances pituitrin diminishes the amount of urine and, hence, diminishes the thirst and improves the general condition. In other instances it has no effect whatever. It probably has no curative action. It must be given regularly, at least twice daily, and, of course, subcutaneously. Most children think the remedy worse than the disease. The beginning dose for young children is one minim and for older children two minims. The dose for young children should be increased one half minim and that for older children one minim daily, watching the results of the treatment carefully. The earliest symptom of an overdose is usually a movement of the bowels within a few minutes. This is generally preceded and accompanied by cramps and colic. Other more marked symptoms are rapid pulse, faintness, excitement and marked pallor. If any of these symptoms are produced, the dose should be diminished enough so that they are not. If there is no improvement in the polyria after two or three weeks, it is useless to continue the drug.

If the symptoms are due to a hyposecretion of the pars intermedia, it hardly seems reasonable to operate or to use the Roentgen ray or radium in the treatment, all of which measures, if affective, diminish rather than increase the secretion.

DISEASES OF THE ADRENAL BODIES

Anatomically the adrenal bodies are made up of two distinct portions, the cortex and the medulla. In all probability these two portions are

not related functionally. The medulla is made up of chromaphil cells, which are the same as the chromaphil cells which are distributed in similar but smaller masses along the sympathetic nervous system at other levels. There is no satisfactory evidence to prove that the secretion of this chromaphil tissue is of any use during normal conditions, but it is possible that it may be of service in emergencies. It has been supposed that disturbances of the function of the medulla of the adrenal bodies was the cause of *Addison's disease*. There is no positive proof of this assumption, however, and there are many points about the disease which seem to show that it is not justified. It is, however, of little importance whether this assumption is true or not, because Addison's disease does not occur in childhood.

There is considerable evidence to show that the cortex is of importance in connection with the development of the reproductive organs. At any rate, tumors of the cortex are often associated with sex abnormalities. This is especially likely to be the case if the tumors are adenomata. It is presumable that when tumors of the cortex produce sexual changes it is because they are associated with hypersecretion. The manifestations of disease of the adrenal cortex vary according to the age at which it develops.

If it develops early in intrauterine life, pseudohermaphroditism results. The internal organs are feminine, but the infant has the appearance of a cryptorchid boy, with a well formed penis, sometimes with a little hypospadias, the scrotum being empty but not cleft. The vagina narrows at its lower part and connects at the level of the neck of the bladder with a well developed prostate and opens into the prostatic portion of the urethra at the level of the summit of the verumontanum by a rather small orifice. The prostate and urethra are of the masculine type. If the disease develops later in intrauterine life, there is no question as to the sex of the infant, but there is atrophy of the uterus and ovaries, enlargement of the clitoris and premature hypertrichosis.

When the disease develops before puberty, there is a general overgrowth of the body, often marked obesity, excessive development of the sexual organs and hypertrichosis. Intellectual development is usually impaired. This condition occurs much more often in females than in males. It is characteristic that when the disease occurs in females it results in a diminution of the female and an increase of the male primary and secondary sex characteristics, while, when it develops in males, there is an increase in the male but no appearance of female characteristics.

If only one adrenal body is involved, removal of the tumor is possible. Collett (American Journal Diseases of Children, 1924, XXVII, 204) has reported the successful removal of the tumor in a child a little over two years old, with marked diminution of the sexual manifestations.

DISEASES OF THE PINEAL BODY

The secretion of the pineal gland is probably not of great importance. Symptoms of disturbance of its functions are confined to early life, because it involutes at puberty. The pineal body is not essential to the maintenance of life. Certain authorities state that its removal leads to precocity of development. Others state that no changes result. On the other hand, other observers have found that the administration of pineal substance hastens growth and sexual maturity.

Tumors of the pineal body, usually adenomata and, therefore, probably associated with an increase in secretion, sometimes cause marked

changes in sexual development. Strangely enough these symptoms occur almost entirely in boys. There is a marked overdevelopment of the skeleton and of the body as a whole. The ossification of the bones is far in advance of the age. There is also a marked premature development of the sexual organs and of the secondary sex characteristics. The distribution of the hair is masculine and the voice is masculine. The intelligence corresponds to the age and not to the physical development. Associated with these changes are usually evidences of increased intracranial pressure or of pressure on those parts of the brain near the pineal body. There is, of course, no treatment for this condition. Removal of the pineal body in man is out of the question.

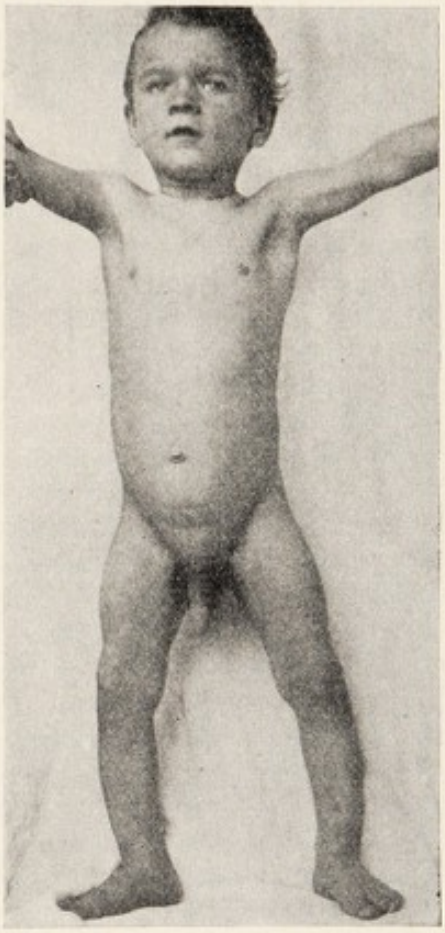

FIG. 173.—Pineal tumor. Age, twenty-three months.

DISEASES OF THE PANCREAS

Tumors and cysts may develop in the pancreas in early as in adult life. The pancreas may also be involved in congenital syphilis and in chronic diffuse tuberculosis. The secretions of the pancreas may presumably also be diminished. This probably happens very seldom in early life, however, and, when it does, it is practically impossible to recognize it. Acute pancreatitis may also occur in early life, but it occurs so seldom that it is simply a pathologic curiosity. The pancreas may be involved in mumps. When it is, there is tenderness and muscular spasm over the pancreas. The prognosis is good.

DIABETES MELLITUS

The only important disease of the pancreas in childhood is diabetes mellitus, which is due to an insufficiency of the internal secretion of the pancreas. This insufficiency may be relative, but is usually the result of disease or destruction of the islands of Langerhans. Diabetes mellitus is far more common in early life than was formerly supposed, from 2% to 3% of all cases being in children under ten years of age. It occurs about twice as frequently from five to ten years as from birth to five years. It may occur even in infancy. I have seen a number of cases, the youngest being eleven months old. It is, as in later life, more common in boys than in girls and is definitely familial and hereditary. Several children in the same family are not infrequently affected. The prediposition of the Jewish race to this disease is less marked in early life than later. It apparently may follow continued excessive indulgence in sweets.

A possible explanation for the lesser incidence of diabetes in childhood may be the somewhat greater sugar capacity at this age, the glucose capacity per unit of weight being greater in childhood than in adult life. The child is better equipped for sugar retention than the adult, the liver substance being greater in proportion to the bulk, 4.4% at four years, 2.7% in the adult.

Symptomatology.—The symptoms in childhood are essentially the same as in the adult—polyuria, polydipsia, emaciation and constipation. Polyphagia is perhaps more common. Symptoms of gastric disturbance are also rather more common in the child. Disturbances of the skin, eyes and nervous system are less frequent. Marked changes in the disposition are, however, very common, the child often becoming quiet, morose and sad. Enuresis is often the first symptom to call attention to the trouble, while in others the first thing noticed is the presence of flecks of sugar on the clothing, vessel or floor. Another early and suspicious symptom is irritation of the external genitals.

The onset is usually described as acute in childhood. It is, however, probably less often so than is ordinarily supposed, because what is taken for the onset is undoubtedly not infrequently the change from the mild to the severe type of the disease. Another reason why the onset seems acute in children is that children do not complain of slight symptoms and do not notice symptoms which would trouble an adult. Nevertheless, in certain instances the onset is extremely acute. The mild and severe types of the disease are about equally common in early life, but there is a much greater tendency for the mild to change to the severe type in early than in later life.

Prognosis.—The prognosis in childhood is almost uniformly bad. Von Noorden's statement that "with few exceptions diabetes in childhood knows no cure, no matter how mild it may appear in the beginning, nor how gradual its development in the first months or even years," is unquestionably essentially correct. The duration in children under ten years of age is seldom longer than one and one half or two years and in the second decade it seldom exceeds from two to four years. Nevertheless, there are a certain number of cases on record in children in which recovery has taken place. I have had one such case. It is possible that recovery may occur more often and certian that the duration of the disease will be prolonged under the newer methods of treatment.

Diagnosis.—Diabetes should be recognized at once in those cases in which the symptoms are typical. It should also be thought of in all cases in which there is gradual failure or a condition of malnutrition which is not otherwise accounted for. Irritation of the external genitals, especially in girls, and the appearance of enuresis in children previously continent should always suggest it. The finding of sugar in the urine under such circumstances confirms the diagnosis. The finding of sugar in the urine in childhood without other symptoms of diabetes does not, however, justify the diagnosis of this disease, because a temporary glycosuria, as the result of overstepping the limit of tolerance, is not uncommon in childhood. This is especially likely to occur when the indiscretion is accompanied by a disturbance of the digestion. Even the persistent appearance of sugar whenever a definite amount of carbohydrates is overstepped, although this amount is below the usual normal limit, does not necessarily justify a diagnosis of diabetes, as has been shown by Von Noorden.

It is also important to remember that in occasional instances, even in childhood, glycosuria may be a manifestation of the so-called *renal diabetes* which is apparently a condition distinct from true diabetes mellitus. In this condition the excretion of sugar occurs with a blood sugar content which is normal or even lower than normal, while in diabetes mellitus the glycosuria occurs only when the blood sugar is above normal. Reduction of the carbohydrates in the diet has no effect in this condition,

while it diminishes or stops the glycosuria in diabetes mellitus. It is impossible to build up a tolerance for carbohydrates in renal diabetes, while it can be built up in diabetes mellitus. There are no other symptoms in renal diabetes, many in diabetes mellitus. There is no treatment and no cure for renal diabetes. None are necessary, as it is a harmless condition. Withdrawal of the carbohydrates may, however, bring on acidosis, which otherwise never develops.

Under normal conditions sugar rarely appears in the urine of breast-fed infants, although the breast-fed infant gets from eleven to eight grammes of lactose per kg. of weight daily. If it does, it is in the form of lactose or galactose. The assimilation limits are lower in the new-born and in premature infants. They vary, moreover, quite widely in infants of the same age. Glycosuria is not uncommon in premature breast-fed infants and is not of pathologic significance. Temporary glycosuria is not at all uncommon in the acute digestive disturbances of infancy, in which conditions the assimilation limit is not infrequently lowered. If the sugar in the food is lactose, the sugar in the urine is in the form of lactose or galactose.

Both dextrose and lactose precipitate Fehling's solution on boiling. Dextrose precipitates it immediately, lactose after a longer time. This difference in time is, however, not sufficient to justify any conclusion as to the form of sugar. If a positive test for sugar is obtained with Fehling's solution, the urine should then be boiled for three minutes with an equal amount of a 10% solution of sodium hydrate. Hot Fehling's solution should then be added and the mixture again boiled. A positive reaction then means the presence of lactose.

Treatment.—The treatment of diabetes in early life is, of course, the same as in later life and consists essentially in regulation of the diet. This is not, as a rule, any more difficult in childhood than in adult life. It is, however, often very difficult in infancy, when the diet normally is made up chiefly of milk and carbohydrates. The main principles of the dietetic treatment of diabetes are simple. They are to keep the intake of carbohydrates below the carbohydrate tolerance of the individual and make up the deficiency in calories with proteins and fats. It must be remembered, however, that one half of the protein molecule may serve as a source of sugar for the organism and that, unless the fat is protected by a certain proportion of carbohydrates, acid intoxication results. There are, however, great differences of opinion as to what are the proper proportions of the different food elements in the diet. The teachings of the specialists are different and each one has changed his teachings several times during the last twenty years. Allen's starvation treatment has modified our ideas very materially and the discovery of insulin has introduced an entirely new factor.

When a child with a severe type of diabetes comes under treatment, it is probably wiser not to starve it at once, because of the danger of bringing on an acid intoxication, although it is well known that when acid intoxication is present starvation diminishes or stops it instead of increasing it. It is better to first cut out the fat entirely for a few days, then to cut out the protein, at the same time gradually diminishing the carbohydrates, and finally to stop them entirely. When the case is of the mild type, it is safe to stop food entirely at once. It is important, when food is stopped, to give large amounts of water. It is allowable to give a certain amount of clear soups and beef tea. Older children may be given tea or coffee, if desired. There is no danger in giving alcohol in

the form of whiskey or brandy, as it is burned up and does not serve as a source of sugar. Very little is gained, however, by using it. It is unnecessary and probably inadvisable to give bicarbonate of soda.

Starvation should be continued until the urine is sugar free. It is not necessary for the practitioner to estimate the blood sugar, although such estimations are of help in hospitals where they can be easily and accurately done. In my experience, it is wiser in children to skip the stage of 5% vegetables, as most children prefer starvation to the vegetables. Other carbohydrates should, therefore, be given at once. There is much difference of opinion as to whether fats or proteins should be first given or a combination of the two. There is, of course, considerable danger of acid intoxication, if fats are given too freely. My own feeling is, however, that many of the specialists are more afraid of fat than they need to be. The carbohydrates must, of course, be kept below the limit of tolerance and the calories made up by proteins and fats. If at any time sugar appears, a starvation day must be given at once. It hardly seems necessary or advisable to go into the details of feeding in diabetes in this place, because there are so many books devoted entirely to the treatment of this disease. It must be remembered, however, that a child must have at least one gramme of protein, and preferably two grammes, daily per kg. of weight.

Allen's dictum that the diabetic patient's weight should be kept down by limitation of the diet to the point which his pancreas is able to take care of is presumably correct in the case of adults. It is not strictly applicable, however, in the case of children, because they must be given enough not only to hold their weight at a minimum but to enable them to grow in height and to increase enough in weight to keep up with the increase in height.

In the vast majority of cases of diabetes in early life insulin is neither necessary nor advisable in the treatment, except, perhaps, temporarily. If, however, the child has acid intoxication when it comes for treatment, develops it later or is unable to take sufficient carbohydrates to thrive, insulin must be used as in the adult.

If acidosis is marked, all food should be stopped, but definite amounts of glucose or glucose-producing substances, such as cane sugar or orange juice, given by the mouth. One unit of insulin should be given per three grammes of carbohydrate. It is very important not to give an excess of insulin. The earliest symptoms of an excess of insulin are sudden, marked hunger, sudden weakness and a peculiar restlessness or nervousness. These symptoms may be difficult of recognition in a child. In them pallor or flushing of the face, dilated pupils and an increase in the pulse rate are symptoms objectively recognizable. More marked evidences of an overdose are sweating, tremor and finally convulsions.

If the child is in coma, pure dextrose should be given either subcutaneously or intravenously, preferably intravenously. A 5% solution may be given subcutaneously and a 10% solution intravenously. Insulin should be given subcutaneously at the same time, in the proportion of one unit to three grammes of dextrose. The urine should be examined under these conditions every one or two hours for the presence of sugar and the acetone bodies.

When insulin is used because of low sugar tolerance, it should be given one half hour before a feeding. It may be given once, twice or three times daily, as is necessary. The general rule is that half as many units of insulin are required daily as there are grammes of sugar excreted

in twenty-four hours. If the amount indicated is five units or less, this may be given all at once before breakfast. If between five and thirty units are required, they may be given half before breakfast and half before supper. If more than thirty units are required, one third should be given before each of the three meals. The object in the treatment, of course, is to use as little insulin as is necessary and to stop it as soon as possible.

SECTION XVIII

UNCLASSIFIED DISEASES

ACUTE INFECTIOUS ARTHRITIS

RHEUMATISM

It will be a great advance in medicine when the term "rheumatism" is dropped. It interferes with clear thinking, is the cause of many errors in diagnosis and leads to much foolish and thoughtless therapy. The lesion in and about the joints, whether acute or chronic, is due either to the action of some toxic substance in the circulating blood or to micro-organisms brought by the blood. A far better name, when the symptoms are acute, is acute infectious arthritis, although this term also is open to certain objections.

Etiology.—Acute infectious arthritis in childhood is almost certainly due in the vast majority of instances to the presence of a microörganism in the joint or the tissues about it. It is possible, however, that the symptoms may be due to toxic products formed elsewhere. It is almost certain that the causative organism is a coccus. It is uncertain, however, whether it is the large coccus, described by Poynton and Paine, the streptococcus described by Rosenow or some other form of coccus. It is generally believed, probably correctly, that in the vast majority of instances the causative organism enters through the tissues of the fauces and nasopharynx. In fact, in most cases the arthritis is preceded by a more or less marked inflammation of the tonsils, nose or nasopharynx. In some instances infection apparently comes from disease of or about the teeth. It seems to me irrational to lay so much stress as is usually done on predisposition and heredity in the etiology of "rheumatism." As I see it, the only reasonable explanation of a familial or inherited tendency or predisposition to "rheumatism" is the inheritance of a tendency to overgrowth of adenoid tissue in the fauces and nasopharynx or of a mucous membrane of low resistance. Bad hygienic surroundings and lack of sunlight and fresh air play a part in the etiology only in so far as they lower the general resistance and thus predispose to diseases of the tonsils and nasopharynx. It is idle to believe that any special form of diet can have anything to do with the etiology of a disease caused by a microörganism, which presumably enters through the throat.

Symptomatology.—The symptomatology of acute infectious arthritis in early life is materially different from that in late childhood and adult life. In the first place, the disease almost never occurs in infancy and is very uncommon before four years. The adult picture of "acute articular rheumatism" is almost never seen in early life. When it is, the appearance of the joints and the general symptoms are the same as in the adult. In many instances the only symptoms are indefinite pains in the extremities, often not located by the child in the joints, a little stiffness of the joints or lameness and a slight elevation of temperature. The temperature often never goes above 100° F. These symptoms may

vary in duration from one or two days to a week. In other instances the pains are more severe and are definitely located in some joint or joints and the disability is a little more marked. The temperature is, however, never high. In still more marked instances there may be a little swelling and sometimes a little redness about one or more joints with still more disability and a little higher temperature. In most instances, however, the child does not feel sick enough or uncomfortable enough to want to stay in bed. The duration of the symptoms may be from a few days to one or two weeks. General constitutional symptoms are slight. There is no sweating. Unfortunately, these mild cases are just as likely to be complicated by infection of the endocardium and pericardium as are the more severe manifestations of the disease seen in late childhood and adult life.

The blood shows a moderate polynuclear leucocytosis and often the changes of a mild secondary anemia. The urine is usually concentrated and highly acid, but seldom shows the evidences of acute degenerative nephritis.

Prognosis and Complications.—Acute infectious arthritis in early life is of itself an unimportant disease. The infection in and about the joints never goes on to suppuration and the disease is never fatal. The danger in acute infectious arthritis in early life is in its complications. The most serious are those involving the heart. It is questionable, however, whether these complications should not properly be looked upon as other manifestations of the infection which caused the "rheumatism" rather than as complications. I have occasionally seen acute nephritis in connection with acute infectious arthritis and in a few instances have seen pleuritis develop. I have, however, never seen peritonitis, meningitis or even meningismus. I have very often seen chorea precede, follow or accompany "rheumatism." Neither can properly, however, be looked upon as the cause of the other. They are both simply manifestations of either a common or a similar infection. Unless the focus of infection can be found and removed, repeated attacks of acute infectious arthritis are likely to occur.

Diagnosis.—If a baby is thought to have "rheumatism," it is safe to assume that it has not. New-born and young babies occasionally have an acute suppurative arthritis. No one, however, can mistake that for what is commonly called "rheumatism." The symptoms are too severe. In general, what is though to be "rheumatism" in infancy is scurvy. In scurvy the tenderness is along the shafts of the bones rather than about the joints, the swelling is primarily over the shafts, there is no redness and there are almost always other signs of scurvy, such as hemorrhages into the skin, swollen and purplish gums and hematuria. In childhood tuberculous disease of the hip, spine or knee may be confused with acute infectious arthritis. In these conditions, however, the onset is slower, the symptoms more sharply localized and the duration longer. Physical examination, moreover, shows the limitation of motion characteristic of the joint involved. Periosteitis and osteomyelitis are sometimes in the beginning confused with acute infectious arthritis. In these diseases, however, the pathologic process is almost always limited to one bone or one joint. The onset is acute. The symptoms are marked from the first and increase rapidly in severity. They are more marked than they ever are in acute infectious arthritis at this age. The temperature is high and usually irregular. There is a very marked polynuclear leucocytosis and the patient is manifestly extremely ill.

There are a number of other conditions, associated with pain in the extremities, which are sometimes confused with "rheumatism." The most common of these is "growing pains." There are, of course, no such things as "growing pains." Nevertheless, children, like adults, when they have played very hard, may have pains and aches in the muscles in the same way as adults. These are neither "growing pains" nor "rheumatism," but are often called both. Children occasionally have severe pains in the shins at night, usually two or three hours after going to sleep. I do not know the explanation of these pains. They are certainly not due to infection and never lead to anything else. They are most easily relieved by heat externally. Children also have pains in the neighborhood of the joints, which are presumably due to mild mechanical injury at the actively growing epiphyseal lines as the result of overexertion and overuse. These pains are most marked after exercise and disappear with rest. There are no other manifestations of disease.

Treatment.—The most important part of the treatment of acute infectious arthritis is the preventive. Adenoids and chronically diseased tonsils should be removed before they have a chance to serve as portals of entry for the microörganism which causes the disease. Tonsils which are repeatedly acutely inflamed should be removed for the same reason. Everything should be done to prevent infections of the nasopharynx. The teeth should be kept clean and filled or removed, if they become decayed. It is not possible to prevent entirely the development of acute infectious arthritis in this way, but its frequency can be much diminished. It is to a considerable extent a preventable disease. It is useless to attempt to prevent it by cutting red meats out of the diet or by any other special dietary regulations, as the disease is not due to a disturbance of the metabolism but to infection by a microörganism.

A child with acute infectious arthritis, no matter how mild the symptoms, should be put to bed and kept there until all the symptoms have ceased. It is usually advisable to keep it there several days longer in order to prevent their recurrence. The diet should be regulated by the temperature, there being no contraindication to any single foods or types of foods. Water should be given freely. The bowels should be kept open. If the symptoms are at all severe, it is advisable to have something rather than cotton or linen next to the skin. Heat externally relieves the discomfort to a certain extent. Immobilization of the extremity does more. The application of cotton dressings on which oil of wintergreen or the old fashioned lead and opium wash have been sprinkled sometimes seems to afford relief. It is difficult to understand how these applications can do any good, except in so far as the cotton keeps the joint warm, because little or none of the drugs can be absorbed. They do no harm, however, and the parents feel that something is being done. The smell of the wintergreen proves it.

It is very unlikely that salicylic acid has any effect on the course of acute infectious arthritis, as it is impossible to get a sufficient concentration of the drug in the blood, and hence in the joints, to even limit, much less inhibit, bacterial activity. It is also probable that it neither increases nor diminishes the frequency of cardiac complications. It does, however, undoubtedly lower the temperature and make the patient more comfortable. The source of the drug is of no importance. The salts of salicylic acid are preferable to the drug itself, because they are more soluble. It makes no difference whether salicylate of sodium, strontium or lithium, or a combination, is used. They are less likely to disturb

the stomach and are seemingly more effective, if an alkali is given at the same time. Bicarbonate of soda is the one of choice. As many grains of this as of the salicylate should be given or, better, enough to make the urine alkaline. The salicylate may be given in powdered form or in a mixture with some simple syrup. It is rather foolish to combine it with a syrup, however, as it is itself so sweet that it is sickish. Acetylsalicylic acid—aspirin—is less likely to disturb the stomach than the salicylates and is, therefore, usually preferable for children. It is not necessary to use any form of salicylic acid in most cases of acute infectious arthritis in childhood, however, as the adult type of acute articular rheumatism is seldom seen at this time. If the salicylates are used, enough should be given to produce the physiologic symptoms, that is, profuse sweating and ringing in the ears, unless vomiting makes it necessary to stop the drug sooner. Five grains of salicylate of soda every three hours for a child of five years is an average dose. Larger doses should be given to older children. It is advisable, however, to give somewhat smaller doses of acetylsalicylic acid.

The pain is seldom severe enough to require anything stronger than acetylsalicylic acid or antipyrine to relieve it. There is no objection, however, to giving small doses of morphine, if necessary.

It is inadvisable, as a rule, to remove the tonsils or adenoids, whether or not acutely inflamed, during an attack of acute infectious arthritis. In general, it is wiser to wait until the attack is over. They should then be removed in order to prevent, as far as possible, recurrence of the arthritis.

CHRONIC INFECTIOUS ARTHRITIS

This disease, also called chronic toxic arthritis and sometimes chronic non-tuberculous arthritis, is the usual type of chronic arthritis seen in early life. It is unquestionably secondary to some focus of infection elsewhere in the body. In the vast majority of instances there is probably no direct invasion of the tissues, of and about the joints, by bacteria, the lesions being due to the action of toxic substances brought by the blood.

Pathology.—The joints show marked thickening of the capsule and of the connective tissues just outside of it. There is also thickening and vascularization of the synovial membrane. Fibrous adhesions are sometimes present. The cartilages may show pitting at their margins from the pressure of little processes of the thickened synovial membrane, but are otherwise healthy. There are no bony changes.

When the lymph nodes are enlarged, they appear normal on section, or sometimes show ecchymoses in their substance. Microscopically they show simple hyperplasia. When the spleen is enlarged, it is firm and of normal appearance. Microscopically it shows hyperplasia of the lymphatic tissue of the Malpighian corpuscles.

Symptomatology.—The onset is in most instances insidious and the progress slow. The first symptom is usually stiffness, which is soon followed by swelling. Pain is ordinarily not as marked a symptom as stiffness and is seldom troublesome, unless the attempt is made to move the joint. The joints earliest affected are those of the hands, wrists and cervical spine, the ankles, elbows and other joints being subsequently involved. All the joints may be eventually attacked. Involvement of the joints of the jaw is quite characteristic. As a rule, the affection of the joints is symmetrical. The swelling looks and feels more like a

general thickening of the tissues around the bones than a bony enlargement. It is smooth, general and fusiform. There is no tenderness or redness, except during acute exacerbations. There is no tendency to suppuration or to bony anchylosis. There is, however, marked limita-

FIG. 174.—Hands in chronic infectious arthritis.

tion of motion. The muscles which move the diseased joints show early and marked wasting, but there are no changes in the electrical reactions. Marked deformities of the extremities are likely to develop as the result

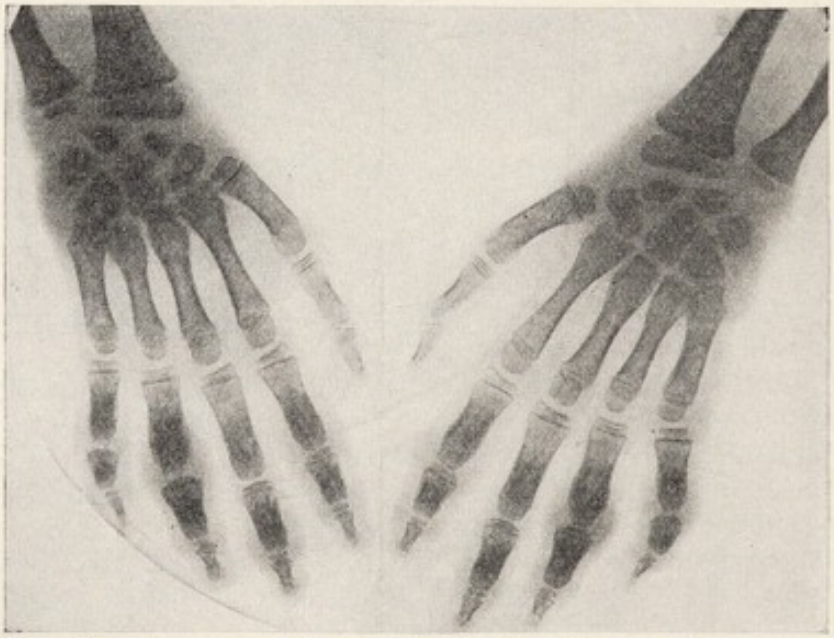

FIG. 175.—Roentgenogram of same hands (Fig. 174) showing normal bones.

of contractures of the muscles. These are usually of the flexors and adductors, that is, of the stronger muscles. There is usually considerable loss of weight. The course is slow and, unless the source of infection can be found and removed, steadily progressive. Periods of spontaneous

improvement are, however, not uncommon. It is not a fatal disease, death, when it occurs, being due to complications, usually in the digestive tract.

Enlargement of the peripheral lymph nodes is not uncommon. It attacks primarily and chiefly those related to the affected joints, but is not infrequently general. The lymph nodes are discrete, hard, not tender and show no tendency to break down. Their size varies with the acuteness of the symptoms in the joints. Enlargement of the spleen is not at all uncommon and may be slight or marked. There is no definite relation between the severity of the joint manifestations, the enlargement of the lymph nodes and the size of the spleen. The blood shows the changes characteristic of mild secondary anemia. There may or may not be a polynuclear leukocytosis. This is more likely to be present during exacerbations of the joint symptoms and may be quite marked. It bears no relation to the size of the lymph nodes and spleen. It is unquestionably simply an evidence of the toxemia which is the cause of the manifestations in the joints.

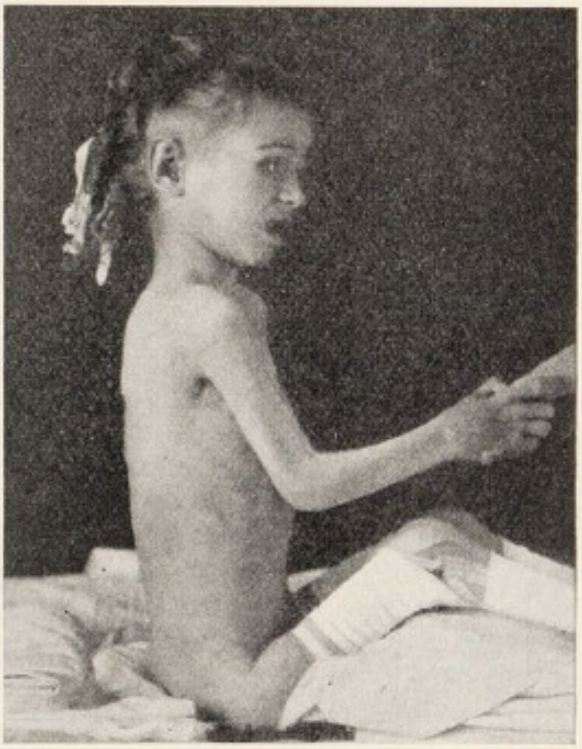

Fig. 176.—Chronic infectious arthritis. Note position of jaw.

The temperature presents two types. One shows periods of pyrexia, lasting only a few days, and followed by longer periods of apyrexia. The other shows more or less continuous slight pyrexia. The pyrexial attacks may or may not be associated with clinically demonstrable exacerbations of the trouble in the joints.

The urine usually shows nothing abnormal, but sometimes during exacerbations it may show the changes characteristic of acute degenerative nephritis of a very mild type. There is no tendency to involvement of the heart. If heart lesions develop they are accidental, not a part of nor a complication of the disease.

In some of the patients the eyes seem quite prominent. This appearance is probably not due to exophthalmos, but to the emaciation and rigid position of the jaw.

Chronic infectious arthritis is not infrequently called *Still's Disease*. This term, if used at all, should, however, be applied only to the group of cases originally described by Still, that is, those cases of chronic infectious arthritis in which there happens to be enlargement of the lymph nodes and spleen, anemia and polynuclear leukocytosis, as well as involvement of the joints. It is wiser, however, to drop it from our nomenclature.

Diagnosis.—The onset and symptomatology of chronic infectious arthritis are so characteristic that it is not likely to be confused with other conditions. Tuberculous arthritis is usually limited to a single joint, generally one of the larger ones, the enlargement of the joints is not

as symmetrical, there is more often redness, and, in time, evidences of breaking down of the tissues develop.

The Roentgen Ray shows involvement of the bones in tuberculous arthritis, while they are not involved in chronic infectious arthritis.

Treatment.—The treatment consists primarily in the discovery and removal of the focus of infection which is the cause of the disease. No other treatment can be more than palliative. If the focus of infection is found and removed, complete recovery, even in the most severe cases, is, however, the rule. In most instances it is in the tonsils or in some of the adnexa of the nose and nasopharynx. It must not be forgotten that even in early childhood there may be a chronic infection of the ethmoid cells or of the maxillary antra. The focus of infection is seldom located in the teeth and jaws in early childhood, but often is in late childhood. In early, as in adult life, it is impossible to determine whether there is suppuration about the teeth or not without the help of the Roentgen ray. The focus may be a pyelitis, which is not infrequently of a mild or remittent type. The source of the intoxication may be anywhere. I have known it to be chronic bronchitis, with or without bronchiectasis, a small encapsulated empyema and a latent abscess of the lung.

When no focus of infection can be found elsewhere, the digestive tract should be carefully studied, because the changes about the joints may be caused by the toxic products formed as the result of abnormal fermentative and putrefactive processes in the contents of this tract. These abnormal processes are most easily discovered by a careful macroscopic and microscopic examination of the stools. I have been but little helped by examination of the urine for evidences of putrefaction. The treatment consists in the regulation of the diet to correct the abnormal fermentative processes. The principles to be followed are the same as in the treatment of disturbances of the digestion associated with abnormal fermentation. It is foolish to give cultures of the lactic acid forming organisms or of the bacillus acidophilus as a routine in such cases, because it is impossible to permanently implant any organisms in the intestines by giving them by the mouth. It is true that the intestinla bacterial flora may be temporarily somewhat modified, if the bacteria are given continuously. The character of the flora can only be changed, however, by changing the composition of the food, that is, by changing the medium on which the bacteria grow. Furthermore, if bacterial cultures are given as a routine, without careful study of the stools, they are just as likely to do harm as good.

There are no drugs which have any specific action in this disease. The salicylates and other so-called "rheumatic" remedies cannot be expected to do any good, and may by their depressing action do much harm. Treatment with stock vaccines is illogical. If it is possible to make an autogenous vaccine, it is possible to remove the focus of infection and cure the disease. I am very sceptical as to the value of non-specific protein therapy.

Local applications are useless, except possibly for the temporary relief of discomfort. The regular application of heat, however, to the affected joints, unquestionably does some good. The joints may be "baked" or, better, treated by electric lights. Regular massage is also of value. The object of both heat and masssage is the same, namely, to improve the circulation. Passive motions of the joints and proper apparatus to prevent and overcome contractures are also of value. Much harm may be done, however, unless they are skilfully used.

Fresh air, sunlight and an abundance of suitable food improve the general condition and are of importance, but are in no way curative. Cod-liver oil, iron and other drugs may be used for the relief of certain symptoms and complications, but do not prevent the onset or affect the course of the underlying process.

GLANDULAR FEVER

(PFEIFFER'S DISEASE)

This condition, first described by Pfeiffer in 1889, is probably simply a symptom complex, not a disease entity. The picture which it presents is, however, characteristic enough to warrant description.

Etiology.—The etiology is unknown. It is, however, undoubtedly bacterial in origin. The symptomatology suggests that the causative organism may be some form of streptococcus. It is most common between two and eight years, but may occur in both older and younger children. Several members of the same family are often affected. It occurs most often in the autumn and winter. The period of incubation is unknown. In West's series it apparently was most often seven days.

Symptomatology.—The onset may be acute with high temperature or gradual with an increasing temperature. It is accompanied by the usual symptoms of an acute, febrile, infectious disease. Swelling of the glands develops in two or three days. It almost always appears first on one side of the neck and then, from one to three days later, on the other. The swelling is situated behind the upper half of the sternomastoid muscle. It appears smooth and uniform, but palpation shows that it is made up of several discrete glands. The swelling is tender and often painful. It is often accompanied by stiffness of the neck. The swelling lasts from one to four weeks. The inflammatory process almost never goes on to suppuration. The axillary and inguinal lymph nodes are enlarged in about three quarters of the cases, and the mesenteric in about one third. The liver and spleen are moderately enlarged in about half of the cases.

The pharynx and fauces are moderately reddened in many of the cases. There are, however, no symptoms of coryza. Hoarseness is a not uncommon symptom. There is almost always a feeling of tightness, or choking, in the throat. The breath is foul and the tongue much coated. There is marked anorexia. Nausea is not uncommon, but there is no vomiting. There is usually a tendency to constipation. Not infrequently there is a green diarrhea just before improvement begins. Pain in the abdomen is not uncommon. It is presumably due to involvement of the mesenteric lymph nodes. The rate of the pulse is increased out of proportion to the rise in temperature. Debility is more marked than would be expected from the fever and other symptoms. The temperature drops to normal by lysis and the symptoms gradually improve. The duration of the acute stage varies from three or four days to two weeks. The enlargement of the glands persists into convalescence.

Diagnosis.—This disease is sometimes mistaken for mumps. It should not be, however, because the swelling is not in the location of the parotid gland. Furthermore, there is no reddening about the opening of Steno's duct, while in mumps there is no enlargement of the inguinal, axillary and mesenteric lymph nodes or of the liver and spleen. In cervical adenitis due to inflammation of the tonsils, the lymph nodes first affected are about the angle of the jaw and the evidences of inflammation of the fauces are more marked. Enlargement of the other periph-

eral lymph nodes, of those of the mesentery and of the liver and spleen does not occur in ordinary cervical adenitis. It must be admitted, however, that the differential diagnosis between glandular fever and ordinary cervical adenitis is often very difficult. In fact, it is not improbable that they are both due to the same cause and that the peculiar symptom complex in glandular fever is simply due to some peculiarity in the causative organism or to the amount of absorption from the primary focus.

Prognosis.—The prognosis is good. Recovery always takes place. The inflammation in the glands almost never goes on to suppuration. Convalescence is, however, often slow. Acute nephritis is an occasional complication.

Treatment.—The treatment is entirely symptomatic and along the same lines as that of acute tonsilitis and cervical adenitis.

ACRODYNIA

This peculiar syndrome, which is also called "The Pink Disease," "Erythroedema" and several other names, is either a new disease in this country or has been recognized only during the last few years. It is probably not the same condition that was described under this name in Europe about a century ago. It occurs most often in infancy and seldom develops after five years.

Symptomatology.—The onset is very gradual, several weeks or months usually elapsing before the baby is brought to a physician. The early symptoms are slowly increasing irritability, restlessness and sleeplessness, with loss of appetite, strength and weight. After a few weeks or months more characteristic symptoms develop. The hands and feet become swollen, purplish-red and cold. The discoloration is most marked at the tips of the fingers and gradually fades out until it disappears entirely at or near the wrist. In many instances there is desquamation in the affected areas, which is most marked at the tips of the fingers and toes. Occasionally papules or pustules form on the fingers. In rare instances the nails are involved. Occasionally a similar discoloration appears on the cheeks and the tip of the nose, while in other instances there are scattered areas of erythema on the body.

Associated with, and often preceding, the discoloration of the skin is marked paresthesia of the extremities, which is often most distressing. The patients are constantly rubbing, scratching and chewing the extremities. In one reported instance the child had eaten off the ends of several fingers and toes. There is probably some diminution of cutaneous sensibility in these cases. It is difficult to determine this, however, because of the age of the patients. The intense itching causes much restlessness and sleeplessness in many cases. In other cases, however, there is marked dullness and apathy. In marked cases the babies either lie on the side rubbing the hands or feet or crouch down with the head burrowed into the pillow. I know of no disease in which the picture of abject misery is so marked. The reflexes are usually diminished.

The hair often falls out and in other instances is pulled out by the patients. Another peculiar manifestation is loosening and falling out of the teeth without any evident disease of the gums.

Marked anorexia is a striking symptom in almost every case. On account of the failure to take food emaciation is almost always very marked. Vomiting is unusual and constipation the rule.

Frequent urination is common. The urine seldom contains albumin, although there is often an excess of polynuclear leucocytes. The blood shows a moderate leucocytosis, which is sometimes due to an increase in the polynuclear neutrophiles and sometimes to an increase in all the cells. The spinal fluid is essentially negative. The Wasserman test is negative and nothing abnormal has been found in the gastric secretions in the few cases in which they have been examined. The temperature is usually normal. If not, it is more often subnormal than elevated.

Recovery usually takes place after several months to a year, although occasionally a patient dies.

Etiology.—Judging from the symptomatology, this syndrome is due to some disease of the peripheral nervous system, involving especially the sensory nerves. There are, however, no pathologic data to verify this assumption. It seems at first glance as if it is a deficiency disease. There is no evidence, however, that this is the case. In fact, what evidence there is, is against it. Furthermore, there is no evidence to show that it is due to either an excess or a lack of any of the food elements in the diet. There is a certain amount of evidence to show that it may be the result of previous respiratory infection or to focal infection in the upper respiratory tract. At any rate, there is a history of a respiratory infection preceding the onset of the disease in many cases and recovery has apparently been hastened by the removal of adenoids and tonsils in other instances.

Diagnosis.—It is easy to recognize this disease when it is fully developed, if the possibility of its occurrence is borne in mind. It may be suspected whenever loss of appetite and paresthesia occur without any other definite explanation. It may be distinguished from pellagra by the lack of a sharp line of demarkation of the skin lesion, the age of the patient, the absence of pellagra in the family and in adults in that region, the absence of marked digestive symptoms, the history of a preceding respiratory infection and the duration of the symptoms.

Treatment.—The most important element in treatment is to force feeding. The baby should be fed with a tube, if it will not take the proper amount of food otherwise. Improvement usually begins at once when the proper amount of food is taken. If there are evidences of infection in the upper respiratory tract, treatment for the infection should be instituted. Adenoids and tonsils should be removed, if they appear to be diseased. Rodda has reported some striking results after their removal. Otherwise the treatment is entirely symptomatic; soothing washes for the itching, hypnotics for the restlessness and sleeplessness, stimulants if indicated.

CHONDRODYSTROPHY FETALIS

In this condition the trunk is of normal size while the extremities are short. The head is usually of normal size, but sometimes a little enlarged. The bridge of the nose is usually markedly sunken. The shape of the hand is characteristic. The fingers are short, of nearly the same length and separated at the second joint. This hand is often spoken of as the "trident" hand. The mentality is usually normal, but sometimes is slightly defective. The muscular power is diminished during early life but later is often greater than normal. The development of the external genitals is normal, as is also the sexual power.

Prognosis.—In the majority of instances death occurs in utero or during the first few days after birth. Many others die during infancy, but, if infancy is passed, the prognosis as to life is as good as the average.

Etiology.—Nothing is known as to the essential cause of this disease. As in most of the cases the pathologic changes have evidently taken place

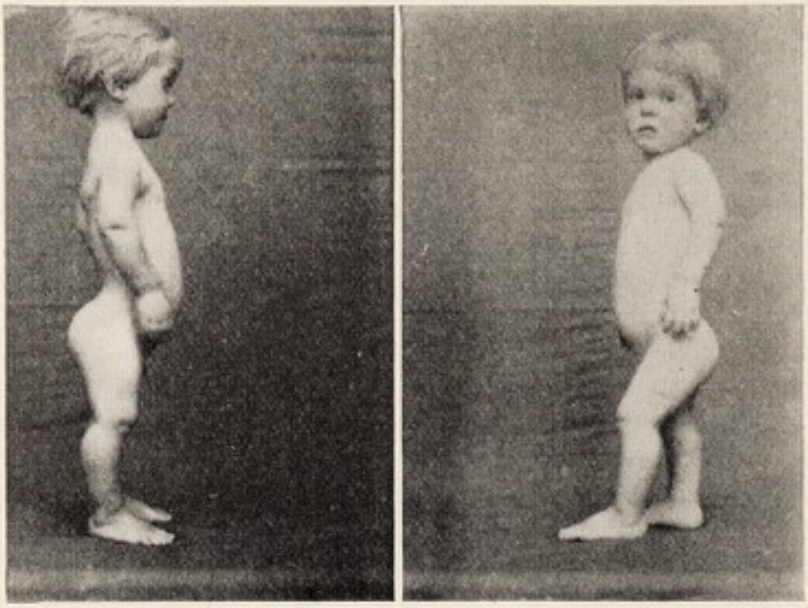

FIG. 177.—Chondrodystrophy.

at the time of the ossification of the primitive cartilages and have come to a stop before birth, the disease must be active between the third and sixth months. As it is often associated with other defects of development, it must be due to some general cause affecting the nutrition of the organism as a whole. It is possible that it may be due to some change in the placenta, but is more likely due to some morbid process, perhaps of an

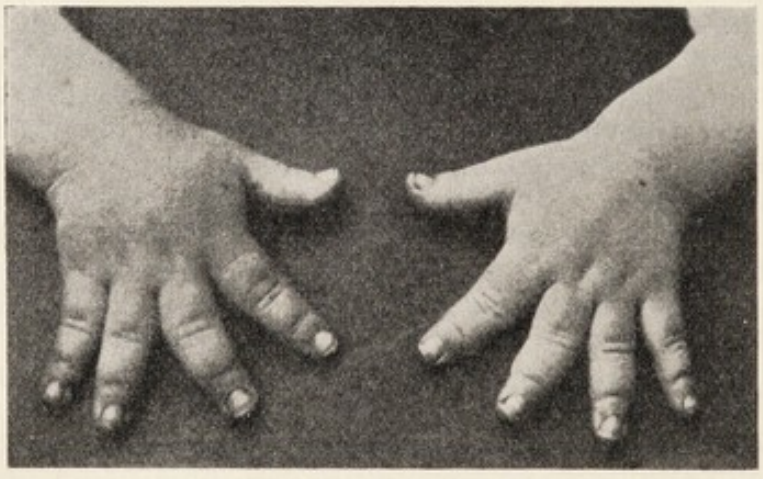

FIG. 178.—"Trident hand" in chondrodystrophy.

infectious nature, attacking the ovum as a whole. In a considerable number of cases adults afflicted with this condition have borne children of the same kind. In other cases, however, their children have been normal. In the majority of cases, however, the condition has not been present in the parents. Heredity, while it plays a part in the production of the disease, is probably not, therefore, of great importance.

Pathology.—The pathologic process is, in brief, a disturbance of the normal process of ossification of the primary cartilage. It takes place early in fetal life, probably most often between the third and sixth months, and has ceased at birth. As the pathologic process usually affects endochondral ossification and is active only during the earlier

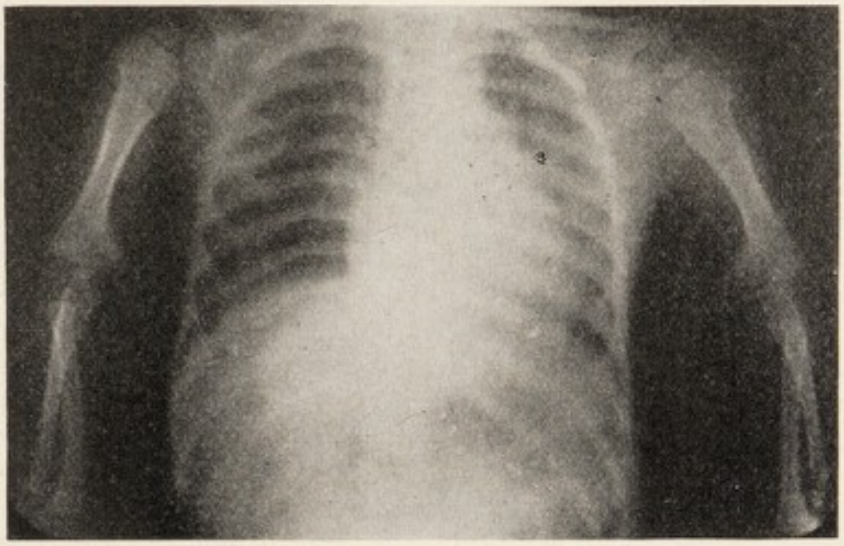

FIG. 179.—Chondrodystrophy.

months of fetal life, only those bones are involved in which this method of ossification normally takes place at this time. In them, however, the process is most definite and universal. Those bones which are formed entirely in membrane and those which, though formed in cartilage, remain entirely or almost enrtiely cartilaginous until the latter part of intrauterine life, are almost never involved. The periosteal formation of bone goes on normally or more actively than usual. These facts explain the distribution and to a certain extent the character of the deformities seen in these cases. It is for these reasons that the bones of the upper part of the cranium, of the trunk (except the ribs) and of the carpus and tarsus are normal, while those of the base of the skull and of the pelvis, as well as the ribs and the long bones of the extremities, are deformed.

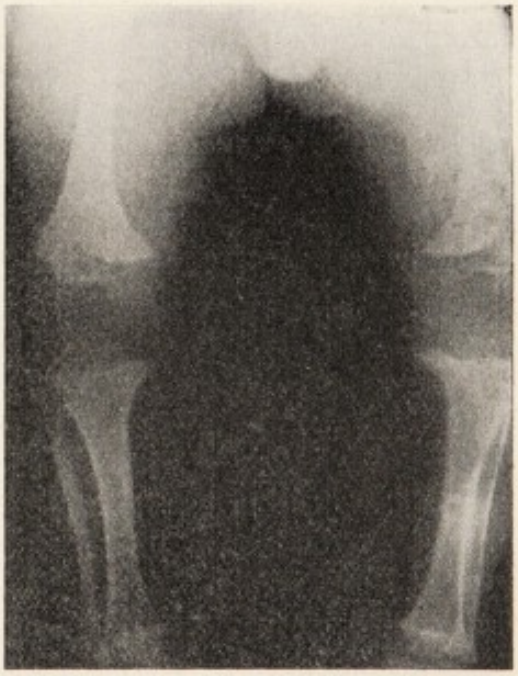

FIG. 180.—Chondrodystrophy.

The flattening of the bridge of the nose is due to a shortening of the base of the skull, which may be due either to premature tribasilar synostosis or to imperfect development of the basal cartilages. The flattening of the sides of the chest, which is seen in some cases, is due to the arrested development of the ribs. The beading of the ribs is due either to periosteal overgrowth at the end of the diaphysis, forming a cup-shaped mass around the epiphysis, or to the displacement forward of either cartilage or rib by an asymmetrical development, resulting from the abnormal folding in of the periosteum. The pelvis is contracted

and flattened because of abnormal ossification of the innominate bones. The growth of the sacrum and coccyx is not interrupted.

The long bones of the extermities depend upon cartilaginous ossification at the junction of the epiphyses and diaphysis for their growth in length during intra- as well as extrauterine life. An arrest of this process results, therefore, in shortening of the extremities. In chondrodystrophy the chief pathologic changes are at the line of ossification. The most important of these are in the columnar zone, which is absent or defective. Growth in length is thus prevented. There is in addition in some cases a fibrous ingrowth of the periosteum between the epiphyseal cartilage and the diaphysis. This prevents, of course, any further growth in length at this point. The shafts of the long bones are almost entirely composed of periosteal bone. This is often excessive and not infrequently abnormally hard. The medullary canal is sometimes diminished in size or even replaced by hard bone. At the ends of the shaft the periosteal overgrowth may extend peripherally so as to form a cup about the cartilaginous extremity. Bowing of the long bones is common. The pull of the muscles accounts for this when the bones are softened. When they are hard, the bowing is probably due to local intrusions of the periosteum, which prevent regular growth, or to a resistance of the thick cartilages at the epiphyses to the growth of the diaphysis.

Diagnosis.—This condition is so characteristic that it ought not to be mistaken for anything else. It is, however, sometimes confused with rickets, cretinism and osteogenesis imperfecta. Rickets is a disease which occurs after, not before birth. The extremities are not shortened, the bridge of the nose is not sunken and there is no deformity of the hands. The appearances of the bones with the Roentgen ray are diametrically opposite. In cretinism the fontanelle is widely open, the tongue is large and protruding, the skin is thickened and the baby is mentally deficient. There are many other differences which it is not necessary to describe. The pictures of the bones shown with the Roentgen ray are also radically different. The diagnosis between chondrodystrophy and osteogenesis imperfecta is considered under the latter disease.

Treatment.—There is no treatment.

OSTEOGENESIS IMPERFECTA

Nothing whatever is known as to the etiology of this condition, which is also sometimes called osteopsathyrosis and fragilitas ossium. It is certainly not due to an insufficiency of any of the glands of internal secretion or to syphilis. In a certain number of cases it is familial and in others hereditary. It is equally common in the two sexes and may be transmitted through both sexes. In the great majority of cases the manifestations of the disease are present at birth. It is generally believed, moreover, that even when the symptoms do not appear until some months or years after birth, it is, nevertheless, congenital in origin.

Pathology.—The pathologic process, as the name implies, is an imperfect ossification of the bones. All the bones, whether formed from membrane or cartilage, are involved. The cartilage itself is, however, normal. There is, therefore, no interference with the growth of the bones in length. Ossification in the cartilages is, however, abnormal and the formation of bone from the periosteum and in the marrow is imperfect. The osteoblasts are everywhere diminished in number and the osteoclasts are much too numerous. As the result of these changes the bones are either abnormally soft or very brittle. The blue sclerotics, which are

often present in this disease, are not due to any inherent coloring of the sclerotics but to an increased transmission of the color of the choroid. Very few observations have been made as to the metabolism in this disease and the results of these observations have not been consistent.

Symptomatology and Prognosis.—The characteristic symptom is multiple fractures of the bones. These fractures are almost invariably intraperiosteal. They may occur before, during and after birth. When they have occurred in utero, they may or may not have healed at the time of birth. If they have healed, they may have caused marked deformities. When they occur after birth, they are said to cause less pain than under normal conditions. They may result from very slight trauma or from simple muscular exertion. The callus is usually larger than normal. In some instances healing occurs more quickly than usual, but in others it is delayed. All sorts of deformities follow as the result of these fractures. The extremities, therefore, usually seem shorter than normal. The tendency to fractures may persist through life, but is said to diminish after puberty, if the children survive. Many cases are born dead. Infants with this condition are usually small and poorly nourished and die early. The head is usually very soft, often being merely a membranous bag with isolated bony plates. In other instances, however, the ossification of the head at birth is normal. If the babies live, the head is usually abnormal in shape because of the yielding of the bones to pressure. The hair is said to be fine and luxuriant. It is said that these children are usually below par mentally. Those whom I have seen have not been. This peculiar condition of the bones is often associated with blue sclerotics. This association is especially common in the familial form. Blue sclerotics may be present, however, without imperfect ossification of the bones.

The Roentgen ray shows multiple, mostly intraperiosteal, fractures, excessive callus formation, lack of ossification in all the bones, slenderness of the long bones, an irregular and usually thin cortex and enlargement of the medullary cavity. The epiphyseal cartilages and the centers of ossification are enlarged. The epiphyseal lines are straight.

Diagnosis.—There ought not to be any trouble in the diagnosis of this disease. Very severe cases of rickets with multiple green stick fractures may suggest it. In rickets, however, the characteristic bony lesions of rickets, enlargement of the centers of ossification of the bones of the cranium, rosary and enlargement of the epiphyses of the long bones, are present, while in osteogenesis imperfecta these lesions are absent and the deformities are all in the shafts of the bones. The pictures shown by the Roentgen ray are, moreover, radically different. At first glance osteogenesis imperfecta may be mistaken for chondrodystrophy fetalis. In this condition, however, the bridge of the nose is sunken, the head is not soft, the trunk is not involved and the extremities are short because of failure of the bones to grow in length, not because of fractures and deformities. There is marked thickening of the long bones of the extremities from excessive periosteal bone formation and marked widening of the bones in the region of the epiphyseal lines. The trident hand is characteristic and the sclerotics are never blue. The findings with the Roentgen ray are diametrically opposite.

Treatment.—There is no specific treatment for this disease. Phosphorus, cod liver oil and the calcium salts are all highly recommended. I have never seen any of them do any good, but have seen phosphorus given in large doses do much harm by disturbing the digestion and the

general condition. All that can be done for these babies is to feed them carefully and give them plenty of fresh air and sunlight. Extreme care must be exercised in handling them in order to prevent breaking of the bones. In infancy it is necessary to be constantly on the watch for fractures, as in many instances the babies show no subjective symptoms of injury. When fractures occur, great care must be taken to prevent the development of deformities.

SECTION XIX

DISEASES OF THE SKIN

MILIARIA

Miliaria is the result of obstruction of the sweat glands, which may or may not be accompanied by inflammation. When there is simple obstruction of the sweat glands from an accumulation of epithelial cells, the result is the appearance of large numbers of very small vesicles, none of them larger than the head of a pin. This eruption is called *sudamina*. It appears most often in the course of febrile diseases. It is of no importance, causes no discomfort and requires no treatment.

When there is inflammation about the sweat glands which causes a retention of their secretion, an eruption appears, which, according to its severity, is described either as the *red gum* (m. rubra) or *prickly heat* (m. papulosa). When it is slight, it consists of small, scattered, red papules, many of which are surmounted by a tiny vesicle. It causes little or no discomfort, except occasionally a little itching. When it is marked, the eruption is more diffuse and thickly set. Small pustules may take the place of the vesicles. The eruption is usually accompanied by much itching. The scratching which this causes often causes a secondary inflammation of the skin or leads to eczema. If the lesions are not scratched, they dry up and disappear in a few days, provided the cause is removed.

Etiology.—The cause of miliaria is overheating of the surface of the body. This may be due in summer simply to atmospheric conditions. In winter it is often the result of overheated houses. At all seasons its development is favored by an excess of clothing and by wool in the underclothes. Miliaria is much more common in infancy than in childhood.

Diagnosis.—The only condition with which the red gum and prickly heat are likely to be confused are eczema. Miliaria develops quickly and eczema slowly. The lesions of miliaria persist but a short time, unless they are scratched; those of eczema are of long duration. Miliaria appears on wide areas at once; eczema spreads slowly.

Treatment.—Miliaria can to a large extent be prevented by taking care not to dress babies and young children too thickly and by keeping the temperature of the houses in which they live within reasonable limits. If they have an especially delicate skin, their underclothing should contain little or no wool.

If miliaria develops, silk, linen or cotton should be worn next to the skin. Bathing with cool water allays the irritation and itching. Bicarbonate of soda, in the proportion of one rounded teaspoonful to a quart, may be added to the water. A starch bath also often relieves the itching. Half a cupful of starch is added to the water in the bath, which should be at the temperature at which the child usually has its bath, and the child bathed for several minutes. Care must be taken not to irritate the skin while drying. After bathing, some good dusting powder should be

liberally applied. Almost any of the dusting powders is suitable. One of the simplest and best is equal parts of powdered zinc oxide and starch. Boric acid powder, in the proportion of ten grains to the ounce, or salicylic acid, in the proportion of five grains to the ounce, may be added, if desired. Salicyclic acid, however, sometimes increases the irritation of the skin. Ointments and salves should not be used.

URTICARIA

(HIVES)

The characteristic lesions of this condition are wheals or lumps. They vary much in size and in their distribution. There may be only one or two spots or very many. They appear suddenly and disappear quickly, leaving no evidences of their presence, unless they have been scratched. In some instances the lesion is a papule with a reddish areola. Occasionally the papule is surmounted by a vesicle and, in rare instances, vesicles develop without papules.

Urticaria is always accompanied by itching, which may be intense. As the result of the consequent scratching, secondary lesions of the skin, such as dermatitis, often develop. If infection occurs, impetigo and various pustular lesions may result. The itching often causes much irritability and sleeplessness. Other symptoms accompanying urticaria, such as fever, nausea, vomiting and diarrhea, are usually due to the pathologic condition which causes the urticaria.

Etiology.—Urticaria is a vasomotor neurosis involving the blood vessels of the skin. The cause of the vasomotor disturbance is undoubtedly some toxic substance. This may come from without or from within. When it comes from without, it may be from the bite of some insect, usually a mosquito, from contact with a jellyfish, or from the sting of certain plants, such as the nettle. Hence the name sometimes applied to it, "nettle rash." When the cause of the disturbance comes from within the blood, it may be some drug or some special article of food to which the child is sensitized. The toxic substance may also originate in the digestive tract as the result of disturbance of the digestion, no special article of food being at fault.

The drugs which are most likely to cause urticaria are quinine, arsenic and antipyrin. The foods to which children are most likely to be sensitive are eggs, oatmeal, strawberries and shellfish. Protein sensitization may develop to any form of protein, however, and manifest itself in the form of urticaria. One of the common causes of urticaria from protein sensitization is horse serum. When urticaria is due to the absorption of toxic substances from the digestive tract it is likely to be associated with fever, nausea or vomiting, and diarrhea or constipation. All of these symptoms may be absent, however, even when the cause of the trouble is apparently in the digestive tract.

Urticaria is usually an acute condition which disappears promptly when the cause is removed. In some instances, however, the lesions keep recurring in spite of every effort to find and remove the cause and also when every apparent cause has been eliminated. Sometimes an individual lesion may be very large. It is then spoken of as giant urticaria or *angioneurotic edema*. If it involves the tongue, lips or eyelids, the swelling may be very large. If the tongue is enlarged, it may interfere with talking, swallowing and even with breathing. It is probable that similar lesions may develop in the walls of the stomach and intestines, causing pain, vomiting and diarrhea.

Treatment.—The most important points in the treatment of urticaria are the removal of the cause and the prevention of the recurrence of the disease. This is easy when the cause is some drug which can be stopped. It is not difficult when the cause is external or some special article of food, like strawberries or tomatoes, which is not an essential part of the diet and can, consequently, be easily eliminated. When the cause is sensitization to some important article of food, such as milk, eggs or one of the cereals, the problem is more difficult. It is often very hard to cut out everything which contains this food, especially if it is eggs, milk or wheat. Fortunately, in most instances the sensitization gradually diminishes and finally disappears as the child grows older. In marked cases, however, it is advisable to attempt to desensitize the child by giving minute amounts of the food which do not cause a reaction and gradually increasing them. When there are evidences of disturbance of the digestion in the chronic cases, the diet should be regulated so as to correct them.

In an acute attack, unless it is due to some external cause, drug or animal serum, it is advisable to give a cathartic to clean out the digestive tract. There is nothing better than castor oil for this purpose. From two teaspoonfuls to two tablespoonfuls should be given, according to age. If preferred, one of the preparations of senna or a saline may be given instead. The diet should be limited to milk and starch, unless the patient is sensitive to milk or some special form of starch. If such is the case, they should not be given, but broths and other starches used. Water should be given in considerable quantities. It does no harm and perhaps does some good to give one or two teaspoonfuls of bicarbonate of soda in it each twenty-four hours. Lactate of calcium has not been helpful in my experience. There is nothing which relieves the symptoms so quickly in the severe cases, which do not yield to simple measures, as epinephrin. This should be given subcutaneously in doses of from two to ten minims of the 1:1000 solution, according to the age of the child. Relief is usually almost immediate and often lasts many hours.

The various antipruritics should be tried for the relief of the itching. The simplest are a solution of bicarbonate of soda, one rounded teaspoonful to a quart of water, equal parts of alcohol and water, and hamamelis. "White wash," which is carbolic acid, 0.5 c.cm., powdered zinc oxide, 20 grams, and lime water, 100 c.cm., helps in many cases, as do many other similar washes. Bromides and other sedatives should be used at night, if the itching disturbs the sleep. It should go without saying that the clothes next the skin should be cotton, linen or silk.

DERMATITIS

Several varieties of dermatitis are recognized on the basis of the etiology. *Dermatitis calorica*, or sunburn, and *dermatitis traumatica* are relatively unimportant. *Dermatitis medicamentosa*, which results from the ingestion of certain drugs, is more important. Everyone knows that belladonna flushes the skin, but few remember that arsenic may cause a great variety of eruptions. Physicians are also likely to forget that an eruption of pimples and pustules may be due to the iodides, while the bizarre lesions which the bromides sometimes cause are quite likely to be attributed to syphilis. Among other drugs which occasionally cause dermatitis are quinine, the salicylates and antipyrin. The treatment of dermatitis medicamentosa is, of course, the removal of the cause, that is, the discontinuance of the drug.

Dermatitis venenata is the most important form in early life and is due to the application of or contact with irritating substances. There are a large number of drugs which, when applied externally, may irritate the delicate skin of a baby or young child. Among those most commonly used are carbolic acid, iodine, mustard, mercury and turpentine. There are many plants which cause dermatitis when they come in contact with the skin. The most common in New England are the rhus toxicodendron, poison ivy, and rhus venenata, dogwood, or poison sumach. The rhus diversiloba, or poison oak, does not grow in New England. The lesions in *ivy poisoning* do not appear for one or two days after the exposure. They vary from an erythema with a few vesicles and pustules to an inflammation of erysipelatous type accompanied by marked edema and serous exudation. If there is a secondary infection, the lesions may become pustular. The lesions are situated primarily on the exposed portions of the body. The disease may be transferred to other portions of the body by the hands or clothing. The duration of the disease varies from one to four weeks. There is much itching, which is very annoying and may be intense enough to prevent sleep. When the lesions are wide-spread, there is often some fever and constitutional disturbance.

Ivy poisoning is not likely to be confused with anything but eczema. The onset is seldom as acute in eczema and the lesions are not as likely to be limited to exposed portions of the body. Eczema may occur at any time of the year. Ivy poisoning is very rare except in the summer and autumn. There is, moreover, often a history of previous attacks and a known susceptibility in ivy poisoning.

Treatment.—The active principle of the plants which cause ivy poisoning is a non-volatile oil. It is evident, therefore, that direct contact is necessary to produce a dermatitis. This oil is very sticky and clings persistently to parts which have been contaminated with it. It is evident that the first object in treatment is to get rid of this oil. This can be done best by thoroughly washing and scrubbing the affected and exposed parts with soap, water and a scrubbing brush. This treatment seems harsh, but is very effective. The irritating oil is soluble in alcohol. Washing the skin thoroughly with alcohol will, therefore, also remove it. Great care must be taken, however, that the alcohol, which contains the oil in solution, does not get on the normal skin and spread the inflammation. Ointments should never be used on the skin in ivy poisoning, because they absorb the poisonous oil and spread the disease. If the lesions are small and scattered, one of the best methods of treatment is to paint them with collodion, keeping them covered until they have dried up. If they are larger, they should be thickly powdered with boric acid powder and covered with old linen or cotton cloth. A gauze dressing should never be used, as it always sticks to the surface, delays healing and spreads the inflammation. The inflamed areas should be dressed once or twice daily, preferably but once. Soap and water may be used, as the oil is not soluble in water. Care must be taken not to wash the secretions on to the normal skin, and the affected area must be thoroughly dried before the powder is applied. Exposure to sunlight or to the ultraviolet rays for a time each day has seemed to me to hasten healing.

INTERTRIGO

This condition is described by some authors under the head of erythema, by others under that of dermatitis, and by still others as a form

of eczema. The probable explanation is that this term is loosely applied to a number of different lesions. However this may be, everyone who sees many babies knows what is meant by the term.

Intertrigo in the folds of the neck, axillae and elbows almost always occurs in fat babies with delicate skins that are kept too warm. It never becomes serious, but is often hard to cure. The babies should not be dressed too warmly and the folds should be kept well powdered. Healing is facilitated by keeping the irritated surfaces apart by putting a piece of cloth in the folds. In some instances some simple ointment, like the officinal boric acid or zinc oxide ointment, may be used on the cloth instead of powder. It is important not to apply it too thickly.

Intertrigo behind the ears occurs in thin as well as in fat babies. It is sometimes one of the evidences of malnutrition, but is more often due to lack of cleanliness. It is often necessary to put a piece of cloth between the ears and scalp and keep it in place with a cap before the skin will heal.

Intertrigo in the folds of the groins may have the same etiology as that in those of the neck and axillae. If so, it is usually mild in type and yields readily to treatment. When it spreads over the lower abdomen, genitals, buttocks and thighs, it is almost always due to wet diapers. The distribution of the irritation corresponds exactly to the area covered by the diapers. When the irritation is due simply to wet diapers without any chemical changes in the urine, there is seldom anything more than redness of the skin. The treatment is simple and consists mainly in the removal of the cause. The baby must be kept as dry as possible. The diapers should be changed as soon as they are wet. The skin should be thoroughly dried and well powdered every time that the baby is changed. If this treatment is ineffective, various measures may be tried. One helps in one case, another in another. A mixture of equal parts of lime water and water may be used instead of water to wash the parts and stearate of zinc powder applied after they are dry. Sopping on black wash (lotio nigra) once or twice daily and letting it dry sometimes helps. Another method of treatment is to wash the parts with sweet oil and to keep them anointed with sweet oil, or, after washing with sweet oil, lanoline or some other simple ointment may be applied. In some cases a mixture of equal parts of castor oil and zinc oxide ointment is very useful. One of the best methods of treatment is by exposure to warm dry air. This may be accomplished by simply removing the diapers and uncovering the parts in a warm room. A more effectual method is to box in the crib with cloth and stretch a sheet tightly across the top. The baby is confined on the sheet with the affected parts bare. An electric light in the crib underneath supplies the necessary heat.

In some instances the irritation of the skin is caused by loose, excessively acid stools. The lesions are then often similar to those caused by abnormalities in the urine. They are limited to the area which the stools reach. The treatment is, of course, to remove the cause by regulation of the diet. While this is being done the local measures detailed above should be tried.

Occasionally the irritation is due to hyperacidity of the urine. The treatment consists in diminishing the acidity of the urine by increasing the amount of water ingested and by the administration of an alkali.

The most severe cases of intertrigo are due, however, to ammoniacal urine. The urine is sometimes ammoniacal when it is passed. This is unusual, however, the ammoniacal decomposition usually occurring after

it is passed. In these cases the reddening of the skin is very marked. The epidermis often comes off, leaving an oozing surface. In other instances blisters are formed, which, when the tops come off, leave shallow erosions. These may go on to ulceration. Inflammation of the glans penis is not uncommon, especially in circumcised babies. This leads to ulceration at the meatus, which, in turn, causes painful micturition and sometimes retention of the urine. The heels, where they touch the wet diapers, may show a similar condition. These more marked lesions are sometimes mistaken for those of syphilis. The lesions are, however, confined to the diaper area and vary from day to day, while those of syphilis are not so limited and are steadily progressive. There are no lesions on the palms and soles. The color has not the characteristic copper color of syphilis. There are none of the other symptoms of syphilis, such as snuffles and fissures about the mouth.

There has been much discussion as to the etiology of ammoniacal urine and the "*ammoniacal diaper.*" When the urine is ammoniacal when it is passed, the ammonia is apparently the result of a disturbance of the metabolism from an excessive amount of fat in the food, a wrong combination of food elements in the food or an excessive amount of milk. Correction of the formula or a reduction in the amount of milk will usually correct the disturbance. The administration of alkalis by the mouth relieves the system of the necessity of calling on ammonia to combine with the excessive amounts of acid formed.

The etiology of the ammoniacal diaper due to decomposition of the urine after it is passed seems to be different. It was thought at one time that the decomposition was due to the presence of alkalis in the diapers, which had not been properly washed and rinsed. In such instances the alkalis come from the soap used in washing the diapers. This is undoubtedly the explanation in a small proportion of the cases. The apparent explanation in most instances is the presence in the stools of a microorganism which splits urea to form ammonia. Cooke (American Journal of Children's Diseases, 1921, XXII, 481) has found such an organism in the stools, to which he has given the name of *bacillus ammoniagenes*. Other organisms, however, may do the same thing. He found that if the diapers, after being thoroughly washed, were wrung out in either a 1:5000 solution of corrosive sublimate or a 1:20 solution of boric acid the action of the bacteria on the urine was prevented. The cause of the intertrigo being removed in this way, recovery quickly occurs. I have found this method of treatment most useful in some cases. It has also been found that the administration of hexamethylenamin by the mouth will prevent the decomposition of the urine, even in the presence of bacteria which break up urea. Doses of from one to three grains three times daily are necessary. As always, hexamethylenamin can do no good unless it is broken up. Burnham's test should be used, therefore, if there is no improvement in the local condition, to determine whether the hexamethylenamin is being broken up or not. If it is not, acid sodium phosphate should be given at the same time.

ECZEMA

This is by far the most common and the most troublesome of the diseases of the skin in early life. It seldom appears during the first month of life and, fortunately for the reputation of most physicians, there is a strong tendency to spontaneous recovery after the first year.

It may, however, persist into childhood or recur at intervals throughout childhood. It seldom appears first during childhood.

Etiology.—The most important etiologic factor in eczema in early life is the delicacy of the skin of the infant and young child. It not only reacts to external irritants which have no effect on the tougher skin of the older child and adult, but reacts excessively. External irritation is, therefore, one of the causes of eczema in infancy. This irritation may be due to many causes, such as heat, perspiration, scratching, rough clothing, vermin, other diseases of the skin, and so on.

Eczema may be caused by too much food, too rich food or an excess of one of the food elements. Infants with eczema from one of these causes are usually fat and healthy in other ways. When the baby is on the breast, the difficulty is usually either an excessive amount of milk or too high a percentage of fat. When the baby is fed artificially, the difficulty with the food may be an excessive amount of food or too high a percentage of fat or sugar. In older children an excessive amount of starch is the rule.

Eczema may also be the result of protein sensitization. This is seldom the case in the breast-fed. It is possible that the sensitization may be due to the transmission of a foreign protein through the milk. The evidence as to whether this happens or not is conflicting. Sensitization in the artificially-fed is most often to the proteins of cow's milk, eggs or one of the grains. When eczema is due to protein sensitization, the babies are often poorly nourished.

Eczema is often associated with disturbances of the digestion, which apparently are not directly due to overfeeding or to a large excess of one of the food elements. In such instances the cause is presumably some toxic substance absorbed from the intestinal tract.

I am extremely doubtful as to whether reflex irritation or a neurotic inheritance can play any part in the etiology of eczema. It is probable, however, that babies may inherit a delicate skin. I am also very sceptical as to the part which the various "diatheses" play in the etiology of eczema. In fact, I am very sceptical as to the existence of "diatheses" anyway. They seem to me to be merely high-sounding names to cover up ignorance.

Symptomatology.—I do not feel competent to describe in detail the lesions of the skin in eczema in early life. In a general way I divide them, for my own convenience and as a basis for treatment, into several main types. In the first type, which is most common in fat babies and in which the lesions are always most marked on the face, the skin is raw and oozing, but not much thickened. The exudation dries and forms crusts. When these are removed, the raw, oozing skin is revealed beneath. In the next type, which may occur anywhere on the body, the skin is red, thickened and scaly. The eruption is often papular. This type is often combined with the oozing type. In another type the skin is dry, thickened and scaly, but not very red. This type is more common in older children than in babies and is likely to be localized at the bends of the elbows and knees or on the hands. Patches may occur, however, anywhere.

The first two types are accompanied by intense itching and are often complicated by secondary infections as the result of scratching. In the third type itching is less troublesome and secondary infections unusual. Enlargement of the peripheral lymph nodes connected with the affected areas is not uncommon.

Diagnosis.—After having made the diagnosis of eczema, the next thing to do is to try to find out the cause. Every detail of the patient's life must be investigated—clothing, hygiene and surroundings—in order to discover all possible causes of external irritation of the skin.

If babies are on the breast, they should be weighed before and after nursing for twenty-four hours to determine how much milk they are getting. The milk should than be examined. In this way only is it possible to determine whether the baby is getting too much to eat or not and whether there is an excess of one of the food elements. The examination of the stools will aid in determining whether there is an excess of one of these elements and also as to whether there is a disturbance of the digestion from it. When babies are artificially-fed it is easy to find out whether they are getting too much food and whether there is an excessive amount of one or more of the food elements. The examination of the stools gives valuable information as to the condition of the baby's digestion and as to whether it has an intolerance for one of the food elements.

If no cause for the eczema is found in the surroundings or food and there are no evidences of disturbance of the digestion, skin tests should be done for protein sensitization (see asthma). All the proteins which the baby has had in its food should be tried. If it is breast-fed, it is advisable to try some of the other more common proteins on the possibility that it is getting them through the milk. It is also advisable to try some of the proteins which the baby is likely to get soon in its food in order to know in advance if it is sensitive to them. Protein sensitization is more often a cause of the dry, patchy eczema of later childhood than of eczema in babies.

Prognosis.—If the cause of the eczema can be found and removed, recovery is usually rapid. If it can not be found, as it usually can not, the duration of the disease depends on the skill of the physician, the intelligence and carefulness of the mother or nurse, the severity of the individual case and the age of the baby. The older the baby, the sooner it is likely to get well. It will not improve rapidly, no matter how good the treatment ordered, unless the mother or nurse is attentive to every detail of it. It will not do well, unless the physician understands and explains every step of the treatment in detail. It is never advisable to give a definite prognosis as to the duration of the disease in an individual case. It is safe to say, however, that it will not prove fatal to the baby, even if it does half kill the mother, and that it will eventually stop, either because of or in spite of the treatment.

Treatment.—The first thing to be done in the treatment of eczema is to find out the cause; the next thing is to remove it. It is usually easy to eliminate external causes of irritation of the skin. It is easy to cut down the quantity of breast milk which the baby takes by limiting the duration of single nursings or by cutting down the number of nursings. The strength of the milk as a whole can be diminished by lengthening the intervals between nursings and by giving water at the time of nursing. What can be done to vary the individual food elements in human milk is discussed under breast feeding. An artificial food can, of course, be varied to meet any requirements. The amount and strength as a whole can be changed at will, as can also the relation of the individual food elements. As a general rule, it is almost never advisable to take a baby off the breast and put it on an artificial food because of eczema. If it is weaned, the last state of that baby is likely to be worse then the first.

I have never known a baby with eczema to be sensitized to the proteins of human milk. If it is, it should be weaned. If a baby is sensitized to the proteins of cow's milk, it should have a wet nurse or be put on goat's milk. Incidentally, the eczema in some babies that are not sensitive to cow's milk seems, nevertheless, to be due to the casein of cow's milk. They can sometimes take cow's milk in powdered form or condensed without trouble, although they can not take it raw or boiled. When the sensitization is to the proteins of other foods, it is a simple matter to eliminate them from the diet.

While the cause of the eczema is being found and removed, the lesions of the skin must be treated. When the cause can not be found and removed, all that can be done is to treat the local lesions. The most important elements in the treatment of the skin are the prevention of scratching and minute attention to details. No treatment can do any good, if the baby is allowed to scratch, and no treatment will do as much good as it should, unless it is carefully carried out.

The only way to prevent scratching is to confine the hands so that the baby can not get at the affected areas. It is useless to put on aluminum mitts and have the arms free, because a baby can damage the skin almost as much with the mitts as with the fingers. Pasteboard cuffs are more useful. They prevent the baby from bending its elbows and hence from scratching its face, but they do not prevent it from rubbing its face with its arms, unless the arms are confined in some other way. These cuffs are made from stiff pasteboard and covered with cotton flannel. They should reach from just below the axilla to just above the wrist. They are bound on with webbing straps and kept from slipping down by being pinned at the top to the clothing. Another way to prevent scratching is to tie the baby down with a modification of a camisole. A belt reaching from the axillae to the crest of the ilia is kept from slipping down by shoulder straps and is fastened in the back. Strips of cloth of the same width as the belt are sewed to the sides of the belt. These may be pinned to the mattress or tied to the sides of the crib. The sleeves of the dress are then pinned to the mattress or tied to the sides of the crib. Another way is to put all of the baby but its head in a bag or pillow case and tie it down. Still another more elaborate method is to make a slit twenty inches from the top in a sheet large enough to cover the crib and tuck in well both at ends and sides. The slit is made either at a right angle or at an angle of 45° in this way:  It is six inches in width and four inches in length. After the baby's head is put through the slit the flap is fastened about the neck with tape. The sheet is then tucked in like any top sheet, leaving the baby's head sticking out. In the worst cases the baby may be put on a Bradford frame.

Whatever ointment is used must be protected, so that it cannot be rubbed off. When the lesions are anywhere except on the head it is easy to cover the parts with a cloth and keep the cloth on with bandages. When they are on the face a mask should be made of old cotton or linen cloth and, at any rate at first, worn constantly. The mask is made from a strip of cloth wide enough to reach from two inches above the hair line on the forehead to the neck under the chin, and long enough to overlap a few inches at the back of the head. Holes should be cut for the eyes, nose and mouth. The ends of the piece of cloth should then be slit lengthwise to make a many-tailed bandage. These tails are brought

together behind and sewed, so that the mask fits tightly both above and below and can not slip. As an additional safeguard, it is advisable, if babies are very restless, to have the baby wear a light cap in addition. The cap is pinned to the mask in front and at the sides. When the lesions are on the scalp, it is easy to keep it covered with a cap.

The ointment should always be spread on the cloth or on the mask and the cloth or mask then applied to the skin. This is much better than spreading the ointment on the skin and then putting the cloth or mask over it. Plenty of ointment should be used. It is always a mistake to put it on thinly. This is one of the times when it is always wise to send a man, not a boy. Furthermore, the ointment should be kept on all the time. It is seldom necessary to change the dressing oftener than once in twenty-four hours; it should never be changed more than twice. Water should not be used in removing the ointment. It is, of course, one of the fundamental principles in the treatment of eczema that no water shall touch the affected areas. It is easy to remove the surplus ointment with the help of sweet oil, albolene, or some of the other mineral oils used for constipation. It is also very important not to try to get the skin too clean. It does no harm to leave some of the ointment on it, while the rubbing necessary to get it all off may do much harm by irritating the skin. Great care must be taken to prevent the baby from scratching while the dressing is being changed, as exposure to the air always causes itching. Contrariwise, there is nothing which relieves the itching so much as covering the inflamed surface thickly with ointment. What the ointment is makes relatively little difference, provided it is bland and unirritating.

The best treatment for the oozing type of eczema is crude coal tar. This is not the same thing as wood tar, but is a by-product obtained in the manufacture of coal gas. It is inky black and very thick. It should be smooth and contain no granules. The results obtained with it are not satisfactory, unless a good specimen is used. It should not be used when the skin is infected or the lesions are on the scalp. It should be painted on the oozing surface with a cotton swab twice daily until the oozing has stopped. It should not be cleaned off with oil, but allowed to come off itself. After it has come off, the skin is smooth and red, but not oozing.

The skin should then be thickly covered with some bland, unirritating ointment. A very good one is the so-called "bismuth ointment," which is:

Bismuth subnitrate or subcarbonate
Lime water
Anhydrous lanoline āā ℥ i

Enough sweet oil should be added to make a smooth ointment.

Another good one is:

Starch
Zinc oxide powder āā ʒ i
Vaseline ℥ i

If there is a recurrence of the oozing, the crude coal tar should be used again for a few days.

When the skin is red, thickened and acutely inflamed, but not oozing, it is better not to apply the crude tar plain but to use it in the form of an ointment. The following combinations are the usual ones:

Crude coal tar ʒ ss – ʒi
Starch
Zinc oxide powder āā ʒ i – ʒii
Vaseline ℥ ii

As the skin improves, the crude coal tar may be left out of the ointment or bismuth ointment or lanoline used instead.

Both crude coal tar and ointments containing it stain everything which come in contact with them. It is said that this stain can be prevented from becoming permanent by rubbing the clothes thoroughly with lard before they are washed with soap and water. It is safer, however, to use old clothes and bedding which can be destroyed after the eczema is cured.

When the skin is dry, thickened and red, crude coal tar does not, as a rule, help. Bismuth ointment or a combination of one or two drachms each of powdered zinc oxide and starch with an ounce of vaseline, if applied thickly and continuously, are helpful. If there is much thickening of the skin, the addition of five or ten grains of salicylic acid to the ounce of one of the combinations given above is often helpful.

In the acute stage, when babies are very restless and fretful, it may be necessary to give them paregoric or one of the bromides to quiet them.

SEBORRHOEA CAPITIS

This condition, commonly known as "*cradle cap*" or "*milk crust*," is due to a mixture of the secretion of the sebaceous glands of the scalp, which secrete profusely at this age, epithelial scales and dirt. A crust is formed which may cover any part or the whole of the scalp. It may be thin or thick, according to the amount of secretion and the care which has been taken in the individual case. In neglected cases it may have a very disagreeable odor. If no eczematous inflammation has developed under it, the skin appears normal when the crust is removed.

Treatment.—The first thing to do is to remove the crusts. This can often be done with soap and water. If this is not possible, the scalp should be thoroughly anointed with sweet oil or vaseline at night and covered with a cap. In the morning, with the help of soap and water, the crusts can be removed. Mothers are often afraid to rub hard enough, especially over the anterior fontanelle, to get them off. They must be told, therefore, to use a reasonable amount of force and, if necessary, a fine-toothed comb. In some instances it is necessary to grease the head several successive nights before the crusts can be entirely removed. The hair should be cut short either before or after the removal of the crusts.

After the scalp is cleaned, it should be kept a little greasy with the officinal boric acid ointment. The scalp should be washed in the same way as that of any baby.

IMPETIGO CONTAGIOSA

There is much difference of opinion as to whether this disease is caused by staphylococci, streptococci or both. It is probably that streptococci are more often the cause than staphylococci. It is more common in poorly nourished children than in the healthy, and among the dirty than in the clean. It is contagious. Being contagious, it is likely to occur in groups of closely associated children.

Symptomatology.—The earliest lesion which is ordinarily seen is a lax, partially filled vesicle or bleb, usually varying in diameter from one eighth to one quarter of an inch. In rare instances they may be larger. Ordinarily there is no areola. The contents of these vesicles become purulent in a few hours. The covering of these vesicles being very thin, they are soon ruptured and yellow, superficial crusts are formed. If they are not scratched, they dry up and fall off in a few days, leaving a

reddish spot which fades out in a week or so. The true skin is not involved. Consequently, there is no scar. These spots appear first, in most instances, about the mouth. They may show themselves first on any of the exposed areas of the skin. The infection may be transferred to any part of the body by scratching. Naturally it appears most often on the parts which are the easiest to scratch. The lesions often coalesce and, if they are spread by scratching, may cover considerable areas.

There is, as a rule, not much itching. There are no constitutional symptoms.

Treatment.—In the first place, a child must be prevented from scratching and spreading the disease to other parts of the body. This may be accomplished either by covering the lesions or by confining the hands. The crusts are to be first removed with soap and water. If they do not come off easily, they should first be softened with olive oil or vaseline kept on over night. After the crusts are removed, the affected areas should be kept covered with some simple, mildly antiseptic ointment. I ordinarily use an ointment made up of one part of the officinal white precipitate ointment (ung. hydrargyri ammoniati) and three parts of vaseline. If preferred, from 5 to 20 grains of ammoniated mercury may be mixed with one ounce of vaseline. The officinal boric acid ointment may also be used. If scratching is prevented, recovery takes place in a few days.

FURUNCULOSIS

Furuncles, or boils, are caused by an infection of the subuctaneous cellular tissue by staphylococci, usually of the aureus variety. Infection usually takes place about a hair follicle, but sometimes about a sweat or sebaceous gland. There is nothing characteristic about the individual furuncle in early life. At that time, however, they are likely to be smaller and to occur in larger numbers than in later life. They are most common in feeble and debilitated infants and in children who are below par, although they sometimes develop in healthy infants and children. In babies, they occur most often on the scalp, shoulders and back; in children, on the buttocks and thighs. Styes are also not uncommon in children. In both babies and children boils are likely to occur in groups. In rare instances, in feeble babies, the spots become gangrenous.

Treatment.—As always, the best treatment is the preventive. If babies and children are so fed and cared for that they are well and strong, they seldom have boils. Infection occurs much more easily when the skin is moist and hot than when it is dry and not too hot. Keeping the skin dry and at a proper temperature by dressing babies and children so that they are not too hot, by using hair instead of feather pillows for babies, by changing their diapers frequently and drying and powdering the parts freely, usually prevents the development of boils. Children who use their eyes for close work only with proper light and who wear glasses, if they need them, seldom have styes.

Before a furuncle has completely developed, it may sometimes be aborted by painting it with tincture of iodine. After it has developed, the contents, if it is small, can be squeezed out by pressure on the sides. If it is large, it should be incised. After the boil is opened, a wet dressing of a 1:5000 solution of corrosive sublimate or of a saturated solution of boric acid should be applied. Wet dressings should not be kept up for more than one or two days, however, as keeping the skin moist favors further infection. They should be stopped as soon as possible.

If the scalp is involved, the hair should be cut short. The skin should be kept clean, just as when there are no boils. In the affected region it should be washed daily with alcohol, carefully dried and thoroughly powdered with boric acid powder. When the infection is about the buttocks, genitals and thighs of babies, these parts must be kept clean and dry and powdered with boric acid powder. Exposing these parts to warm, dry air, as in the treatment of severe intertrigo in babies, also helps to prevent infection. In older children, exposure to the ultraviolet rays seems to help. They should not, of course, be used on the scalp. Ointments should not be used, as they macerate the skin and hence favor reinfection.

It must never be forgotten that the discharges from furuncles are contagious. Everything about the patient that can be contaminated by them must, therefore, be either destroyed or thoroughly disinfected. The best way to disinfect the clothing, as well as the sheets and pillowcases, is by boiling.

In my opinion there are no drugs which, given internally, have any effect either on the course of or in the prevention of furunculosis. In obstinate cases, however, vaccines sometimes do much good. An autogenous vaccine is preferable to mixed or stock vaccines, but these also may be useful. The initial dose should be 50,000,000 organisms. Another dose of 100,000,000 should be given from four to six days later. It is usually not necessary to increase the dose farther. It should be repeated at intervals of from four to six days. If improvement is not manifest after four or five doses, it is useless to keep on. I have had no experience in the use of yeast.

In addition to the local treatment of the lesions everything possible should be done to get the patient into the best possible general condition. The diet should be carefully regulated and digestive disturbances corrected. Babies must be given an abundance of sunlight and fresh air. The daily life of children must be so regulated that they are not overfatigued and get enough sleep and rest.

ERYTHEMA NODOSUM

This disease is not at all a common one. It is said to occur more often at about ten years than at any other period of life. Very little is really known about it. Some think that it is simply a marked type of erythema multiforme, others that is is a distinct disease. However that may be, the symptomatology points strongly to its being a constitutional disease with peculiar manifestations in the skin rather than a primary disease of the skin. Many believe it to be a manifestation of tuberculosis. The evidence in favor of this belief, however, is not convincing. For many years it has been thought to be a form of rheumatism. Rheumatism being an indefinite term, very loosely applied, this explanation does not help much. It is presumably bacterial in origin. Whether the peculiar skin lesions are due to the direct action of bacteria or are toxic in origin is not clear. It is not known, moreover, if the disease is bacterial in origin, whether it is caused by a specific organism or whether the symptom complex may be produced by a variety of organisms. It is said to be contagious. If this is true, the contagiousness must be very slight, for even a specialist in the diseases of children sees but few cases in the course of his life. All the cases which I have seen have been single. No other children in the family have been attacked and, when treated in the hospital without special precautions, no other cases have developed.

Symptomatology.—The appearance of the characteristic nodules is usually preceded for several days or a week by more or less marked constitutional symptoms. Occasionally the rash is the first manifestation of the disease. Not very infrequently the symptoms develop after an attack of tonsilitis. The early symptoms are moderate fever, malaise, anorexia and disturbance of the digestion. There is often pain in the back and extremities. Sometimes the onset is quite acute and the symptoms quite serious.

The characteristic nodes usually appear on the legs before they do on the arms. They are most numerous in the region of the knees and elbows and are thicker on the extensor than on the flexor surfaces. They occasionally develop on the trunk and face. There are seldom more than a dozen or two spots. The spots are raised above the skin and vary from one half inch to one inch in diameter. They are round or oval. If oval, the long axis is parallel with that of the extremity. They are smooth, glossy, bright red, tender and painful. They do not itch or suppurate. The color gradually changes like that of a bruise. The swelling disappears in a few days or one or two weeks. The nodules are likely to appear in groups at intervals of a few days. The skin often desquamates over the affected areas.

During the acute stage of the eruption the temperature remains elevated. It usually reaches about 101° F. or 102° F., but may be higher. There is ordinarily marked anorexia and a badly coated tongue. Pains in the extremities and joints are often troublesome. Sometimes the joints are swollen and reddened. Complications in the heart and kidneys occasionally develop. Secondary anemia is common and lasts well into convalescence. The average duration of the illness is about three weeks.

Treatment.—The child should be put to bed and kept there until it is well. The basis of the diet should be milk. Water should be given freely. The bowels should be kept open with saline laxatives. There are no drugs which have any effect on the course of the disease. Acetylsalicylic acid, in doses of from two and a half to five grains, or similar doses of a combination of equal parts of phenacetine and salol may be given for the relief of pain and discomfort. The application of heat externally helps the pain in the extremities. Oil of wintergreen or lead and opium wash may be used as in acute infectious arthritis, if desired.

As soon as the patient is convalescent, foci of infection should be looked for and, if found, removed. They are most likely to be located in the fauces and about the teeth.

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