increased resonance, which may even be tympanitic, owing to emphysema which rapidly develops. If the foreign body remains impacted in one of the bronchi, it usually excites a localized inflammation, which extends to the surrounding lung and terminates in the formation of an abscess. This may result fatally, or there may follow a prolonged illness, with hectic symptoms resembling pulmonary tuberculosis; and finally, after weeks or months, the foreign body may be expelled by an attack of coughing, and the patient recover completely.

The *diagnosis* of a foreign body in the larynx is made by the suddenness of the attack and the violence of the early symptoms. In older children the body may be seen with the laryngoscope, but in young children this is very difficult. The prognosis is always doubtful, and depends upon the nature of the foreign body and the point at which it has been arrested.

Treatment.—The first thing to be tried is inversion of the patient. By this means, assisted by the cough, the foreign body is not infrequently expelled, even though it has passed below the larynx. The symptoms of laryngeal obstruction may call for immediate tracheotomy or laryngotomy, intubation not being applicable to these cases. If, after tracheotomy, the foreign body can be located in the larynx, but can not be extracted through the tracheal wound, the thyroid cartilage should be divided in the median line. The removal of a foreign body from the bronchi or the tracheal bifurcation should be attempted only by a skilled surgeon.

CHAPTER III.

DISEASES OF THE LUNGS.

THE PECULIARITIES OF THE LUNGS IN INFANCY AND EARLY CHILDHOOD.

Thorax.—The general shape of the thorax is somewhat cylindrical, the conical or dome-shape of the adult not being attained until puberty. The antero-posterior and the transverse diameters are nearly equal in the newly born, but after the third year the transverse diameter is always greater, the difference increasing steadily up to adult life. On account of the shape of the chest, the lungs are situated rather more posteriorly in the infant than in the adult.

The thoracic walls are very elastic and yielding, owing to the cartilaginous condition of a large part of the framework. They are relatively thinner than in the adult, chiefly owing to the imperfect development of the thoracic muscles. The greater part of the thickness of the thoracic walls is due to the deposit of fat, generally abundant in well-nourished infants; but where the fat is scanty the walls are extremely

thin. The capacity of the thorax is considerably encroached upon by the high position of the diaphragm, the large size of the thymus gland, and the frequent distention of the stomach and intestines.

Respiration.—According to Uffelmann, the rapidity of respiration during sleep at the different ages is as follows:

At birth	35 per minute.
At the end of the first year	27 " "
At two years	25 " "
At six years	22 " "
At twelve years	20 " "

During waking hours this rate is very materially increased, and from comparatively slight disturbance it may be nearly twice as rapid.

The type of respiration in infants is diaphragmatic, and it continues to be chiefly so until after the seventh year, when the costal element gradually becomes more and more prominent. The rhythm of respiration is easily disturbed. In very young infants the regular rhythm is seen only in sleep. The lungs do not always expand equally; at certain times and in certain positions respiration may be carried on for a few moments almost entirely with one lung. For some moments it may be very superficial, and then quite deep. The length of the interval between inspiration and expiration varies much at different times. Regular rhythmical respiration is not fully established before the end of the second year. After this time disturbances of rhythm are chiefly due to pulmonary or cerebral disease; but in infancy quite marked irregularity may have little or no significance. It is very common in all asthenic conditions.

Structure.—As compared with the adult, the trachea of the young child is larger; the bronchi are larger, more numerous, and occupy a greater space; the air cells are much smaller and occupy less space; and the interstitial tissue is much more abundant (Delafield).

Physical Examination.—This requires tact and time, but yields results which are quite as satisfactory as in adults. It should be undertaken only in a room having a temperature of about 72° F., or before an open fire.

Inspection.—This should be made with the chest bare. There should be noted, the shape of the chest, the presence of deformities from rickets, the want of symmetry in the two sides, bulging of the intercostal spaces, whether the two lungs expand equally or not, also variations in rhythm, and the presence and extent of any recession of the soft parts or bony walls as an indication of obstructive dyspnœa.

Palpation.—This also should be made upon the bare skin, always with the hand well warmed. Although we can not get the fremitus of the voice, we can get that of the cry. This is usually more intense than in adults, on account of the thinness of the chest walls. We frequently get a rhonchial fremitus—a vibration produced by mucus in the tubes. This may enable one to recognise bronchitis quite as positively as by the ear.

The position of the apex beat of the heart should be determined, it being remembered that in infancy this is normally in the mammary line, or just outside of it, and usually in the fourth intercostal space.

Percussion.—For the examination of the back, the child may be laid face downward upon the nurse's lap, or be seated upon her arm. For the front and the lateral regions of the chest, the child is most conveniently placed upon its side across a hard pillow. The percussion blow must be light, either with a single finger or a small percussion hammer, using a finger of the opposite hand as a pleximeter. Percussion should be made both during inspiration and expiration. The normal percussion note is somewhat tympanitic, this being due to the relatively large bronchi and the thin chest walls. This note is exaggerated in the interscapular region and beneath the clavicle, especially upon the right side. Here cracked-pot resonance may be obtained even in health. In early infancy the thymus gives dulness over the sternum as low as the third rib, sometimes even below this point, this gradually diminishing as age advances.

Auscultation.—This may be practised with the naked ear or with the stethoscope. A stethoscope is absolutely necessary for a thorough examination of the apices of the lungs in front and in the axillary regions. Most children are less frightened by the instrument than by the head of the physician during anterior auscultation. For the posterior part of the lungs, the stethoscope may be dispensed with. One with a small bell from half to three fourths of an inch in diameter is of great advantage. In auscultating with the ear it is not necessary to bare the skin. The physician should always auscultate the posterior part of the chest first, because he is most likely to find signs of disease there, and also because this is not so apt to frighten the infant. Every part of the chest should, however, be thoroughly auscultated, not omitting the high axillary regions. A convenient position for posterior auscultation is to have the child held over the nurse's shoulder.

The normal respiratory murmur of the infant is generally described as puerile. In quality this has been likened to the bronchial breathing of the adult, but the resemblance is not a very close one. It is rude, rather loud, and seems very near the ear. Its peculiar character is due to the fact that the tracheal and bronchial sounds are more distinct, because not transmitted through so thick a layer of lung and chest wall. It is especially loud in the regions where the bronchi are superficial, as between the shoulder-blades and beneath the clavicles, particularly of the right side. A careful comparison of the two sides of the chest will generally enable an observer to avoid errors. The irregularity of rhythm which occurs from slight causes should be remembered, and the infant's position changed several times during auscultation, to avoid the mistake of attaching too much importance to a feeble respiratory murmur of one side.

On account of the thinness of the chest walls, there is always great

difficulty in distinguishing between râles produced in the bronchi and pleuritic friction sounds. Before drawing any inference from the auscultatory signs, both lungs must be examined for several minutes, changing the child's position, and often inducing a cry or compelling a deep inspiration by other means, in order to bring out signs which otherwise may be overlooked. As auscultation is extremely difficult or impossible in a crying infant, this part of the physical examination should first be made if the child be quiet, since upon it we must chiefly depend for diagnosis. Inspection and percussion can be deferred until later.

Peculiarities in Disease.—There are several peculiarities connected with the respiratory organs in infancy and early childhood which must be constantly borne in mind in studying their diseases. The muscular development of the thoracic wall is feeble. The soft, yielding character of the thoracic framework causes the chest to sink in readily from atmospheric pressure whenever there is obstructive dyspnœa. On account of the small size of the air vesicles, acute congestion may interfere with their function almost as completely as does consolidation. Because of the delicate walls of the air vesicles, emphysema is readily produced in obstructive dyspnœa, but it is rarely permanent. There is a tendency to collapse, either on the part of lobules or groups of lobules, but very rarely of an entire lobe. This is a much less important factor in the production of symptoms in acute pulmonary disease than many writers would lead us to suppose. The tendency of inflammation to spread from the large to the small bronchi is very much greater than in adults. In all forms of pulmonary disease the rapidity of respiration is much greater than in adults, on account of the rapid metabolism of the child. Areas of consolidation often exist without appreciable changes in the percussion note, because they are superficial and are surrounded by healthy or emphysematous lung. Flatness should always suggest the presence of fluid. Disease is often overlooked, from a failure to examine the whole chest.

Probably the most common mistakes are to confound bronchial râles with friction sounds, exaggerated puerile breathing with bronchial breathing, and to overlook the existence of fluid because of the presence of bronchial breathing.

ACUTE CATARRHAL BRONCHITIS.

Acute catarrhal bronchitis is one of the most frequent conditions for which the physician is called upon to prescribe in children. It occurs at all ages, from early infancy up to puberty. Its frequency, however, diminishes steadily after the second year. The predisposition to acute bronchitis exists with the same constitutional conditions, and is acquired in the same manner as the predisposition to the acute catarrhal inflammations of the upper respiratory tract. (See Acute Rhinitis). Bronchitis is

very common in children who are suffering from rickets and malnutrition. It is much more frequent in the cold months, especially in the late winter and early spring, when there are sudden atmospheric changes and high winds.

Bronchitis may be a primary or a secondary disease. The primary form is excited by cold, exposure with insufficient clothing in severe weather, wetting of the feet, or chilling of the surface in any manner. Under these conditions it may occur alone, or be associated with or preceded by acute catarrh of the nose, pharynx, or larynx. In rare cases it is caused by the inhalation of irritants. Bronchitis is an almost invariable accompaniment of measles and influenza. It is very common in pertussis, in scarlet and typhoid fevers and diphtheria, and may occur in any acute infectious disease; it also complicates pneumonia and pleurisy. The relation of micro-organisms to the other etiological factors is the same as in the other acute catarrhs. (See Rhinitis).

Lesions.—Acute catarrhal bronchitis is an inflammation of the mucous membrane of the bronchi. As a rule it is bilateral, both sides being involved to the same degree. Localized bronchitis is secondary to some other pathological process in the lungs, usually tuberculosis or pneumonia. In acute bronchitis only the larger tubes may be affected, this usually being complicated with inflammation of the trachea (ordinary tracheo-bronchitis); or, in addition, the process may extend to the medium-sized tubes (severe bronchitis); or, in infants especially, it may extend to the smallest tubes (capillary bronchitis). In the last form there are invariably changes in the zones of air vesicles surrounding the bronchi, and these cases are therefore more properly classed as broncho-pneumonia. In the first form the inflammation is superficial, and affects only the mucous membrane of the bronchi. In the second form it may involve the entire thickness of the bronchial wall, and in the third form it does so regularly.

The pathological changes consist in congestion and swelling of the mucous membrane, desquamation of the epithelium, and an exudation of mucus and pus-cells. At autopsy the injection of the mucous membrane is usually distinct; pus and mucus line the walls of the larger bronchi, and by pressure ooze from the cut extremities of the smaller tubes. The chief lesion of the walls of the bronchi consists in an infiltration with leucocytes. In infants dying from bronchitis, the lungs are much more frequently emphysematous than collapsed. There is swelling of the lymph glands at the root of the lung, which in most of the acute cases is slight, but in protracted cases, and after recurring attacks, may be quite marked.

Symptoms.—It is convenient to consider separately the symptoms in infants and in older children.

The bronchitis of infants.—1. The mild form (bronchitis of the larger tubes).—The onset is generally gradual, and the symptoms of bronchitis may be preceded by those of catarrh of the nose, pharynx, or larynx. The

change in the character of the cough, the slightly accelerated breathing, and a further rise in temperature, indicate an extension to the bronchi. The cough may be constant and severe, or very slight. There is no expectoration. The secretions are usually coughed up into the mouth or pharynx, and swallowed. This sometimes excites vomiting. At other times the mucus is coughed only into the trachea or larynx, and aspirated again into the lungs. The respirations are from 40 to 50 a minute, and often accompanied by a rattling sound, due to mucus in the large bronchi or trachea. The general symptoms are not severe, and unless the infant is very young or very delicate no apprehension need be felt as to the outcome. The temperature is generally from 100° to 102° F. for two or three days, then below 100° F. There are a moderate amount of restlessness dependent upon the severity of the cough, usually anorexia, and sometimes vomiting and diarrhœa.

The physical signs in the first stage are dry, sonorous râles over the whole chest. A little later these give place to coarse mucous râles heard everywhere, but especially distinct between the scapulæ and in the infraclavicular regions. On palpation there is usually a marked rhonchial fremitus. Often there is not enough dyspnœa to cause recession of the soft parts of the chest. Unless the disease extends to the smaller bronchi and the air vesicles, the illness usually lasts about a week. Coarse râles in the chest may remain for some time after the symptoms have subsided. Relapses are exceedingly common. In a delicate or susceptible child, or in one whose surroundings are bad, one attack is likely to be followed by a succession of others, so that the child may not be really well until warm weather comes. The general health may suffer from the prolonged confinement to the house, although the patient may never have been seriously ill.

2. The severe form (bronchitis of the smaller tubes).—This differs from the preceding variety mainly in the greater severity of all its symptoms. The onset may be like that just described, the severe symptoms not appearing until the patient has been sick two or three days, or they may be severe from the outset. If the latter, it is indistinguishable from that of broncho-pneumonia. There are cough, dyspnœa, accelerated breathing, fever, and moderate, sometimes severe, prostration. The cough is tighter, and more frequently of a short, teasing character than severe and paroxysmal. There is difficulty in nursing. Dyspnœa may be quite marked and is shown by the active dilatation of the alæ nasi and the recession of all the soft parts of the chest on inspiration. The respirations as a rule are from 50 to 80 a minute. The temperature for the first day or two is usually 101° or 102°, but it may be 103° or 104° F. So high a temperature does not continue unless pneumonia develops. The prostration is in most cases more closely related to the dyspnœa and the rapidity of respiration than to the temperature. Often there is slight cyanosis.

In the beginning the chest is filled with sibilant and sonorous râles, many of them of a musical character. In twelve or twenty-four hours these are replaced by moist râles—coarse or fine, according as they are produced in the large or medium-sized tubes. There are often loud, wheezing râles on expiration. The respiratory murmur is feeble; the resonance on percussion is normal or slightly exaggerated. As the case progresses toward recovery, the finer râles are the first to disappear. The râles are always best heard behind, but they are present all over the chest.

At the onset of such a case it is impossible to say whether the disease will be limited to the medium-sized bronchi or will extend to the smallest bronchi and air vesicles. In young or very delicate infants, and during measles, it is very common for the disease to spread rapidly to the air vesicles. In other cases, usually in infants under six months old, there may develop attacks of respiratory failure or suffocation. These may occur in a severe case at any time, and, because of the infant's inability to empty the tubes of secretion, the dyspnœa steadily increases until the respiratory muscles are exhausted, the inspiratory force being too feeble to overcome the obstruction in the tubes. The symptoms which follow are usually ascribed to pulmonary collapse. I am, however, by no means certain that this is the correct explanation, for in autopsies made in such cases I have usually found the lungs to be the seat of acute emphysema. The clinical picture is a clear one. There is no disposition to cough or cry; the pulse is feeble; the respiration very rapid, superficial, often irregular; the skin cyanotic, and often clammy. Finally, there may be added to the others signs of carbonic-acid poisoning—dulness, apathy, and stupor. Such attacks may come on quite suddenly even in robust infants, and unless the treatment is energetic, even heroic, death often follows in a few hours, being frequently preceded by convulsions.

The usual course of the disease in infants previously in good health is that the severe symptoms continue for two or three days only, after which the temperature falls to 100° or 100·5° F., and gradually becomes normal. The constitutional symptoms usually decline with the temperature, and, except during the first thirty-six hours, they rarely give cause for anxiety. Recovery almost invariably occurs unless the disease extends to the finer bronchi.

Bronchitis is principally to be distinguished from broncho-pneumonia. The differential diagnosis is more fully considered under that disease. The most important points are that in pneumonia the temperature is higher and more prolonged, the prostration greater, the râles very often localized —being heard only behind, often over only one lung—the duration is more protracted, and all the symptoms are more severe.

The bronchitis of older children.—This is not nearly so serious as in infants, because the same danger does not exist of extension of the inflammation to the finer bronchi and air cells.

1. The mild form.—This is very common. The constitutional symptoms are slight, and often entirely absent after the first day. The patient is never sick enough to go to bed. The first symptoms are cough and soreness or a sense of oppression beneath the sternum. The cough is always worse at night. It is at first tight, hard, and racking; later it is loose, and in children over five years old there is usually expectoration—first of white, frothy mucus, but after a few days it becomes more abundant, and of a yellow or yellowish-green colour, from the presence of pus. The physical signs are only coarse râles, at first dry, and later moist, but heard over both sides of the chest, in front and behind. There may be some disturbance of digestion, anorexia, constipation, or diarrhœa. The usual duration of the attack is from one to two weeks. If the patient is not kept indoors the disease may pass into a subacute form, lasting for several weeks as a protracted "winter cough," but without any other important symptoms.

2. The severe form.—The onset is abrupt, with fever, chill, pains in the back, headache, cough, and sometimes pain in the chest. There is a feeling of tightness or constriction beneath the sternum. The onset resembles pneumonia, except that the symptoms are less severe. The temperature for the first two or three days ranges between 100° and 103° F. It is generally highest in the first twenty-four hours. The cough resembles that of the mild form, but it is usually more severe. The expectoration is more profuse, and occasionally, in the early stage, it may be streaked with blood.

The coarse râles of the mild form are present, and in addition there are finer râles—at first dry, and later moist—heard all over the chest. Frequently, wheezing râles are heard on expiration. The duration of the attack is ordinarily from two to three weeks, the patient being sick enough to be confined to bed for three or four days only. There is frequently a cough for some time after all physical signs have disappeared. Relapses are easily excited by any indiscretion before the patient has quite recovered.

The prognosis in the primary cases is good, such almost invariably terminating in recovery, and very exceptionally passing into broncho-pneumonia; but this not infrequently happens when the attack complicates measles or pertussis.

Treatment of Bronchitis. *Prophylaxis.*—To remove the predisposition to bronchitis the same means should be employed as those mentioned in acute rhinitis (page 430). General measures also should be adopted to build up the health of delicate infants. Those with tuberculous antecedents, and those who are especially prone to pulmonary disease, should if possible spend the winter in a warm climate. In all such patients the systematic administration of cod-liver oil should be continued throughout every cold season. The sleeping apartments of susceptible infants should not be too cold—never below 60° F.—but they must be

well ventilated, best by an open fire. Such children should sleep in flannel night clothes, care being taken to see that the feet are always warm. While bronchitis of the large tubes is not *per se* a serious disease, it may become so by extension to the smaller tubes. It is consequently very important in infants and young children that these apparently mild attacks should not be neglected.

General management.—Every young child who has an acute catarrh of the nose, pharynx, larynx, or bronchi should be kept indoors. In every such catarrh accompanied by fever the child should be kept in bed while the fever lasts, even if the temperature does not go above 100·5° F., and is accompanied by no other constitutional symptoms. In infants and young children, many cases of bronchitis result from an extension of an acute rhinitis or laryngitis, hence this precaution is of more importance than everything else in preventing the extension downward of a catarrhal inflammation. A very large number of the cases will recover promptly when no other treatment is employed than to keep the child in bed. The temperature of the room should be about 70° or 72° F. It should be well ventilated and frequently aired, the child being removed to another room while this is done. Infants should not be allowed to lie for hours in the same position as there is a great advantage in changing from the crib to the nurse's arms. Careful attention should be given to feeding (page 190) and to the condition of the bowels. A cathartic, preferably castor oil, should be administered at the outset. Distention of the stomach and bowels with gas adds greatly to the discomfort of the patient, and may cause serious symptoms.

Abortive measures are rarely successful, for, by the time the physician is summoned, the disease is generally so well established that they are futile. Mild cases may sometimes be cut short by a hot foot-bath, free catharsis, and diaphoresis, especially by the use of phenacetine and Dover's powder (phenacetine three grains, Dover's powder one grain, to a child of three years).

Local applications.—Poultices are objectionable on account of their weight and the difficulty in getting them properly applied. For infants the oiled-silk jacket (page 59) is decidedly preferable. This should be applied in the beginning, and may be worn throughout the attack. It accomplishes all that a poultice does, with much less disturbance to the patient. Counter-irritation is very valuable. In infants the best results are obtained by the frequent use of a mustard paste (page 52). It should be large enough to envelop the chest, and covered by a towel, so as not to soil the oiled-silk jacket or the clothing. The paste is removed as soon as the skin is thoroughly reddened, which will be in from five to ten minutes, according to the strength of the mustard and the condition of the child's skin. The skin should then be dried and the oiled-silk jacket again pinned snugly about the chest. This may be repeated, according to

indications, from two to eight times a day. If properly used, it may be continued for a week without causing any soreness of the skin.

Inhalations.—The value of these is not sufficiently appreciated. They may in the great majority of cases take the place of the administration of drugs by the mouth, a very great advantage in infants. They may be used by means of the croup kettle or vapourizer (pages 58 and 59), the child always being placed in a tent. In the early part of the disease relaxing inhalations, like simple watery vapour or limewater, may be used. Later turpentine, creosote, terebene, or eucalyptol may be added. Of these, creosote has given me the most satisfaction. Inhalations are to be used for ten or fifteen minutes from four to twelve times a day.

Expectorants.—In infancy this class of drugs may usually be advantageously dispensed with. For older children the relaxing expectorants, especially antimony and ipecac in combination, may be used in the first stage. When the secretion is more abundant, either the alkaline or the stimulating expectorants may be given. Of the former, the best are liquor potassæ, citrate of potassium, and muriate of ammonia; of the latter, creosote, turpentine, terebene, and squills. Small, frequently repeated doses usually give the best results.

Opium.—This should be given very cautiously to young infants, as it is capable of doing great harm. The dry, harassing cough of the early stage sometimes yields to nothing so quickly as to small doses of Dover's powder (e. g., one tenth of a grain every two hours to a child of one year). In the case of infants, late in the disease, and especially in severe cases, opium should be withheld altogether. It disturbs the stomach, constipates the bowels, and, most of all, it greatly depresses the respiration.

Emetics may sometimes be used with advantage when the secretion is very abundant and the cough feeble, but they should be avoided with weak pulse, great prostration, and slight stupor. Syrup of ipecac is the best emetic under these conditions.

Cardiac stimulants.—These are required in most of the severe cases. The best is alcohol. It should be begun as soon as indicated by weak pulse and general prostration. For a child a year old, from half an ounce to one ounce of brandy, diluted with from six to eight parts of water, should be given in each twenty-four hours, in small doses at short intervals.

Respiratory stimulants.—The most valuable drugs are strychnine and atropine. To an infant of six months $\frac{1}{400}$ grain of strychnine and $\frac{1}{1200}$ grain of atropine may be given every two hours. For a short time twice these doses may be used. They are needed only in the most severe cases, and may be used in combination or alternately. An important respiratory stimulant is counter-irritation over the entire body by the mustard paste or hot mustard bath.

The management of mild cases in infants.—In the great majority of cases the disease is self-limited, tending to spontaneous recovery. Often

no treatment is needed, except the hygienic measures mentioned. An oiled-silk jacket should be applied. If the cough is excessive, inhalations of creosote or turpentine three or four times a day may be used, or small doses of Dover's powder or phenacetine. The oppression which often comes on toward evening may be relieved by a mustard paste at bedtime. Stimulants are not required. All other drugs may be advantageously omitted, but during convalescence cod-liver oil should be given.

The management of severe cases in infants.—These must be treated very much like cases of broncho-pneumonia. The temperature is rarely high enough to require interference, but the chief danger is due to the inability of the child to get rid of the secretion by the cough. In my experience the two most valuable means of treatment have been the use of inhalations and counter-irritation. The former should be repeated for ten or fifteen minutes every two hours, and for a short period may often be given with advantage every hour. Early in the disease, vapour of plain water or limewater may be used ; later, creosote is best. Counter-irritation by the mustard paste should be repeated every three hours, and the oiled-silk jacket worn continuously. Alcoholic stimulants are usually needed in delicate children, and in secondary bronchitis accompanying the infectious diseases. In most of the cases the medication should consist only of cardiac and respiratory stimulants. In strong children the occasional use of an emetic at bedtime is admissible.

Attacks of suffocation and respiratory failure.—The indications here are to get as much blood as possible to the surface and to the extremities, in order to relieve the overloaded right heart, and to compel the child to make full and deep inspiratory efforts. One plan of treatment (Jacobi's) is to induce frequent crying by flagellation or spanking, this being kept up for several hours. Another (H. C. Wood's) is to use alternately hot and cold douches to the chest until some reaction is obtained, and then to follow up this by the occasional use, for a few moments, of a very hot bath (120° F.). Both these means, but especially the first mentioned, are of great value, as I have had abundant opportunity to verify. Another useful measure is the hot mustard bath, or the hot mustard pack applied to the entire body. In conjunction with the above means, both heart and respiratory stimulants should be given in full doses. If possible, oxygen should be administered. As these symptoms are liable to recur every few hours for a day or two, a repetition of the treatment will be needed, and if possible the physician should remain with the patient.

If a young infant can be tided over these critical attacks, recovery is probable. After this danger is past, the treatment previously indicated may be pursued. The use of expectorants, particularly the composite cough mixtures containing opium, can not be too strongly condemned in all severe cases of infantile bronchitis.

The management of cases in older children.—In the non-febrile cases

confinement in bed is unnecessary, but children should be kept indoors. In the early stage, with hard, dry cough, one of the best remedies is brown mixture (the mistura glycyrrhizæ composita of the U. S. P.). It will be found advantageous in most cases to have the formula made up with one half the usual amount of opium. When the cough is especially hard and dry, a single inhalation may be used at bedtime. In the second stage, muriate of ammonia may be added to the mixture; or terebene, two or three drops upon sugar, may be given four or five times a day. Inhalations of creosote or turpentine should be used.

In the more severe cases accompanied by fever the patients should be kept in bed and an oiled-silk jacket applied. In the beginning the liquor ammoniæ acetatis and spiritus ætheris nitrosi may be given for their effect upon the skin and kidneys. For the general discomfort, pain, headache, etc., nothing is better than phenacetine and Dover's powder (three grains of the former to one grain of the latter to a child of five years), repeated every three to six hours. For the cough the same remedies may be used as in the mild cases. All patients should be kept in bed as long as the temperature is above normal. Subsequently, the cases may be managed as in the milder form of the disease.

The protracted cough of convalescence.—It often happens, both in infants and in older children, that after all physical signs and constitutional symptoms have disappeared, a cough continues sometimes for weeks. Expectoration is scanty, or is wanting altogether; the cough is hard, dry, often paroxysmal, and in some cases occurs at night only. For this condition the best remedies are quinine, cod-liver oil, and creosote. The last named may easily be given to young infants as well as to older children, in combination with liquid beef peptonoids.* It may be also used in pill form or by inhalation. These measures may be tried alternately or in combination. Where they are not effective a change of climate should be advised.

FIBRINOUS BRONCHITIS (BRONCHIAL CROUP).

Fibrinous bronchitis is seen in diphtheria, usually as an extension from the larynx or trachea. There is, however, another form of bronchitis attended by a fibrinous exudate, which occurs as a primary disease. This is very rare in children. Weil has, however, collected twenty cases of the primary form. The etiology is obscure. It is seen at all ages, from infancy up to puberty, and it may be either acute or chronic. From the cases thus far reported it would appear that the acute form is relatively more common in children than in adults. The disease may be confined to certain branches of the bronchial tree, or it may affect all the bronchi, even to the minute subdivisions. The fibrinous membrane is found loose in

* A preparation put up by the Arlington Chemical Company, and a very palatable way of giving creosote.

the tubes or adherent. There are generally associated other pulmonary changes, such as emphysema, areas of atelectasis or of broncho-pneumonia.

The acute form somewhat resembles ordinary catarrhal bronchitis. The diagnostic features are the severity of the dyspnœa and the expectoration of tube casts from the larger bronchi, or elongated cylinders from the smaller ones, the former resembling macaroni, the latter vermicelli. The expectorated masses are often in balls or plugs, and their peculiar character is not recognised until they are placed in water. The casts are dissolved by alkalies, especially by limewater. After the expulsion of a large cast, improvement in all the symptoms occurs. These, however, return as the exudate reappears. The ordinary duration of acute cases is from one to three weeks.

In the chronic form there are no constitutional symptoms, but only dyspnœa and cough, often recurring in paroxysms, with the expectoration of fibrinous casts. The patient may have these attacks at intervals of a few days or weeks, extending over a period of months, or even years. There are no characteristic physical signs. The diagnosis rests upon the peculiar character of the expectoration. The prognosis in acute cases is unfavourable, the mortality being 75 per cent (Weil). Chronic cases are not dangerous to life.

Treatment.—This is quite unsatisfactory. To loosen the membrane and facilitate its expulsion, the most efficient means are inhalations of the vapour of limewater and the internal administration of pilocarpine. Occasionally emetics are of value. Improvement in some of the chronic cases has resulted from the use of iodide of potassium.

CHRONIC BRONCHITIS.

Chronic bronchitis is not a common disease in children, particularly in young children, one reason being that chronic emphysema, so frequently an associated condition in adults, is rare in early life. Chronic bronchitis always accompanies chronic pulmonary tuberculosis and chronic interstitial pneumonia, with or without the occurrence of bronchiectasis. It is seen in chronic cardiac disease, especially with lesions of the mitral valve. It may occur as a late symptom of hereditary syphilis. Excluding the varieties mentioned, it usually follows attacks of acute bronchitis, the process becoming chronic because of the patient's constitutional condition or his unhygienic surroundings. The acute attack may be primary, but it often follows measles and whooping-cough. Rickets, general malnutrition, and lymphatism are the constitutional conditions in which acute bronchitis is most likely to pass into the chronic form. Deformities of the chest, the result either of rickets or of Pott's disease, are occasionally a cause.

Symptoms.—The only constant symptom is cough, which is persistent, obstinate, and nearly always worse at night or early in the morning. It often occurs in paroxysms strongly suggestive of pertussis. Expectora-

tion is not generally abundant, but in older children there is usually some expectoration present, and in a few cases it is profuse. A copious morning expectoration of fetid pus or muco-pus indicates bronchiectasis. There is no fever, little or no dyspnœa, and although the patients are thin they are not emaciated, and in many cases the general health is not much affected. There may be coarse mucous râles, or no physical signs whatever. The duration of the disease is indefinite, depending upon the cause. All these patients are better in summer and worse in winter, and suffer frequently from exacerbations of acute or subacute bronchitis.

The diagnosis is to be made mainly from pertussis and tuberculosis. From mild attacks of pertussis the diagnosis may be impossible except by the course of the disease. Tuberculosis may be suspected if the thermometer shows regularly a slight evening rise of temperature, if there is much anæmia, and steady loss of flesh. A positive diagnosis can be made only by the discovery of tubercle bacilli in the sputum.

Treatment.—The first indication is to treat the primary disease. In cardiac cases digitalis is the best remedy, and all sedatives are to be avoided. Attention should be directed to the general condition—rickets, malnutrition, and lymphatism each receiving its appropriate treatment. In most cases a general tonic plan of treatment is best, particularly the continuous use of cod-liver oil. In many cases a change of climate is the only thing which is really curative. For the relief of cough, opiates are to be avoided as much as possible. The main reliance should be upon potassium iodide, creosote and terebene, given both by mouth and by inhalation.

REFLEX COUGH—NERVOUS COUGH.

Strictly speaking, all cough is reflex and of nervous origin. The term "reflex cough" is, however, commonly used to denote that which occurs without any evidence of disease in the larynx, trachea, bronchi, lungs, or pleura. On account of the close nervous connection through the vagus and its branches between the mouth, ear, throat, stomach, and thoracic organs, it is possible for cough to be produced by many forms of irritation in these organs or cavities. Clinically, the following varieties of nervous cough are observed:

1. That dependent upon pharyngeal irritation. One cause of this is an elongated uvula. This cough occurs usually at night, and is tickling, hacking, or hemming in character. A similar irritation may be produced by the trickling of mucus into the lower pharynx from the nose or rhino-pharynx.

2. That due to aural irritation. This is rare, and may be associated with chronic otitis of any variety. It has no special characteristics.

3. That due to gastric irritation—the "stomach cough." This is much more frequent than the other forms. It is usually associated with chronic indigestion and occurs both in infants and in older children.

4. That due to dental irritation. The cough of dentition is often spoken of, although I have never seen a case which could fairly be ascribed to it.

5. Cardiac cough. This is usually associated with mitral disease, and due to pulmonary congestion. The cough is dry, hard, and often severe.

6. The variety which occurs usually about the time of puberty, and often associated with anæmia, chorea, or spinal irritation. It is a short, hacking, or teasing cough, sometimes very distressing, and it seems to be a manifestation of extreme nervous irritability.

7. The periodical night cough, which is generally ascribed to irritation of the vagus or its branches by enlarged, sometimes caseous, lymph nodes of the tracheo-bronchial group. This often occurs in severe paroxysms, the character of which is very much like pertussis. The attacks are apt to come on about the middle of the night and last for several hours. Vomiting is rare. The cough may recur regularly every night for months. On account of the loss of sleep the patient's general health may be considerably undermined.

8. A very similar cough may occur in connection with abscesses in the posterior mediastinum due to Pott's disease.

Symptoms and Diagnosis.—These cases are not common in infants, but are quite frequent in older children. In nearly all the varieties the cough is worse at night, and in many it may be confined to that time. The influence of habit is often seen, the attacks coming on regularly at certain periods. The general health may not be affected, except from the disturbance of sleep. The diagnosis between the different forms is often very difficult. The precise cause in a given case is discovered only by a careful examination of the ear, nose, pharynx, heart, stomach, lungs, and a consideration of the patient's general condition. The existence of enlarged or tuberculous bronchial glands may be suspected in patients of tuberculous antecedents, in those who have previously suffered from measles, pertussis, or repeated attacks of bronchitis, and when the cough is very severe and paroxysmal. A similar group of symptoms may exist with abscesses from Pott's disease. In either of these conditions there may be attacks of suffocation.

Treatment.—Opium and expectorants are not indicated, and inhalations are of little value. The only successful treatment is that which is directed to the cause of the disease. If no cause can be found, and the cough appears to be of purely nervous origin, the best results follow the use of the bromides or the administration of antipyrine at bedtime.

ASTHMA.

Asthma may be defined as a vaso-motor neurosis of the respiratory tract. It is characterized by attacks of severe spasmodic dyspnœa, which

may be preceded, accompanied, or followed by bronchial catarrh of greater or less severity. In the asthmatic attacks of infancy the catarrhal element is very prominent, and these cases present quite a different clinical picture from the disease as seen in older children, which differs in no essential points from the asthma of adults.

Writers differ very much in their statements regarding the frequency of asthma in early life, mainly because of a want of agreement in regard to what shall be included under this term. The asthmatic attacks of infants are considered by some as a stage of bronchitis, by others as distinct from that disease. Typical attacks resembling those of adult life are rare in children, and extremely so before the seventh year. However, of 225 cases of asthma reported by Hyde Salter, the disease began before the tenth year in nearly one third the number.

Etiology.—The general or constitutional causes are the same in children as in adults. Asthma may be hereditary. It occurs especially in children whose antecedents have suffered from gout or from other neuroses. The local cause may be any form of irritation in the nose or pharynx—hypertrophic rhinitis, adenoid growths of the pharynx, hypertrophied tonsils, or elongated uvula—or in the bronchial mucous membrane, as a result of previous attacks of acute bronchitis. It is probable that it may also be caused by the irritation of enlarged bronchial glands. In susceptible persons a paroxysm may be excited by cold or damp air, indigestion, constipation, or the inhalation of various irritating substances, such as dust, the pollen of certain plants, etc. First attacks of asthma in children are apt to follow bronchitis.

Symptoms.—Four quite distinct clinical types of asthma are seen in children: (1.) Cases which in their onset simulate attacks of capillary bronchitis. (2.) Those in which asthmatic symptoms follow an attack of bronchitis, continuing for weeks or months, but not necessarily recurring. (3.) Hay fever, or the periodical form which occurs every summer. (4.) That which resembles the ordinary adult asthma, with the nervous element predominating. The prominence of the catarrhal symptoms is characteristic of all asthma of children, the first two varieties being peculiar to early life.

Attacks resembling capillary bronchitis.—These cases are rare, but may be seen even in infants. The onset is sudden, with moderate fever, incessant cough, severe dyspnœa, and sometimes symptoms of suffocation —cyanosis, prostration, and cold extremities. The chest is filled with sonorous, sibilant, and soon with subcrepitant râles. Instead of running the usual course of bronchitis of the finer tubes, the symptoms may pass away very rapidly, and in forty-eight, sometimes in twenty-four, hours the patient may be quite well. It is only by the course of the disease and by recurring attacks that their true nature can be recognised. In infants this form may be fatal.

Cases following attacks of bronchitis—Catarrhal asthma.—This form is not uncommon, though it is frequently designated by some other term than asthma—sometimes as spasmodic bronchitis, or catarrhal spasm of the bronchi. The symptoms are, however, indistinguishable from asthma, and they evidently belong in the same category. This form is usually seen in infants, being rare after the third year. Many of the patients are rachitic; others have large tonsils, or adenoid growths of the pharynx; while in still others there is every reason to suspect the presence of large bronchial glands. Usually there is nothing peculiar about the antecedent bronchitis; in most cases it is not especially severe, and is limited to the larger tubes. The febrile symptoms subside in a few days, but the cough continues, as do also the dyspnœa and wheezing. When the symptoms are fairly established they are very uniform and characteristic. The respiration is accelerated, usually to 50 or 60, sometimes to 70 or 80, a minute. The temperature from time to time may be very slightly elevated, or it may remain normal. The respiration is noisy, laboured, and accompanied by distinct wheezing, which can sometimes be heard all over the room.

On auscultation, there is prolonged expiration accompanied by loud, wheezing râles, either sonorous, sibilant, or musical, and occasionally moist râles are present. In cases which have lasted some time a moderate amount of emphysema can be inferred from prominence of the infraclavicular regions, and exaggerated resonance over the chest in front.

These symptoms and signs may continue for three or four weeks only, and gradually wear off, or they may last as many months—if they begin in the winter or spring, often continuing until the middle of the summer. While they are constantly present, they vary in intensity from time to time, being usually much worse at night. The symptoms are always increased by exposure to a cold, damp atmosphere, by any fresh accession of bronchitis, and often by trivial digestive disturbances. The usual duration of the cases I have seen has been two to six weeks. The cough is not usually severe, and expectoration in most cases is absent. The general health is often but little affected. With recovery from the asthmatic symptoms the emphysema usually disappears gradually, although I have seen one severe case in which it persisted.

What proportion of these children afterward develop ordinary asthma, from personal experience I am unable to say. Some undoubtedly do, but in others which I have been able to follow, recovery has seemed to be permanent. This would appear more likely in those cases closely associated with rickets, or with other causes which disappear spontaneously with time or as a result of treatment.

Hay fever.—This is very rare before the seventh, and but few well-marked cases are seen before the tenth year. In its clinical aspects it does not differ essentially from the disease as seen in adults, except possibly by the greater prominence of the bronchial catarrh.

Ordinary attacks of the adult type.—These usually occur at intervals of a few weeks or months, depending upon the nature of the exciting cause. The beginning is usually at night, with dyspnœa, a short, dry cough, and loud, wheezing respiration. Deep recession of the soft parts of the chest is seen, as in laryngeal stenosis. There is prolonged expiration, accompanied by loud, sonorous, sibilant and wheezing râles, and the vesicular murmur is very feeble. Later, moist râles may be heard. After many attacks emphysema is present. This occurs more rapidly than in adults, and may be extreme, giving rise in marked cases to serious thoracic deformity. On account of the loss of sleep and interference with nutrition, the general health may become seriously impaired.

Diagnosis.—Typical attacks of asthma are easily recognised. Some of the catarrhal forms seen in infancy, however, present great difficulty, and a positive diagnosis may be impossible except by the progress of the case.

Prognosis.—This is best in the cases of catarrhal asthma in infants, and in older patients when it depends upon some local cause which can be removed, as when the disease is due to reflex nasal or pharyngeal irritation. In the majority of other cases, asthma is likely to become chronic unless the child is removed to some climate in which the attacks do not occur. The younger the child, the shorter the duration of the disease, and the less marked the hereditary tendency, the better the prognosis.

Treatment.—The nose and the rhino-pharynx should be carefully examined in every case of asthma, and any pathological condition there present should be removed as the first step in the treatment. Special importance, in children, should be attached to the removal of adenoid growths of the pharynx. During attacks, the best means of relieving the symptoms is the inhalation of fumes of nitre paper or stramonium leaves. Most of the proprietary remedies (Papier de Fruneau, Himrod's cure, and Kidder's pastilles) contain these ingredients. The two preparations last mentioned are by most children particularly well tolerated. The sleeping room may be filled with the fumes from these substances, or the child may be placed in a tent into which the fumes are introduced. Emetics should be employed when the attack is brought on by indigestion. Lobelia is the most satisfactory remedy for this purpose. To prevent the recurrence of night attacks, nothing in my experience has been so valuable as a full dose of antipyrine at bedtime—four grains at five years and six grains at ten years. Between the attacks the main reliance should be upon the syrup of hydriodic acid and potassium iodide, which are to be given for a long time in full doses. Tonics are to be used in nearly all cases. Those especially valuable in asthmatic patients are cinchonidia and arsenic.

In the cases of catarrhal asthma following bronchitis, expectorants and ordinary cough remedies are useless. Cod-liver oil and the iodide of potassium are valuable in some of the cases. Others get much relief

from the regular use of creosote inhalations several times a day, with a nightly dose of antipyrine. The fumes of nitre and stramonium often afford no relief, and sometimes the cases are made distinctly worse by them. The best of all measures is to send the child at once to a warm, dry climate.

For all children who have had repeated attacks, whether in the form of hay fever or the ordinary variety, the most important thing is removal to a place where they do not have the disease, and a residence there long enough to break up the tendency to recurrence. This will usually require at least three or four years. The region best suited to most asthmatics is one which is high, dry, and moderately warm. Patients often suffer less in cities than in the country. If taken early, asthma in children is frequently curable by these means; if neglected, the disease is almost sure to continue until adult life.

CHAPTER IV.

DISEASES OF THE LUNGS.—(Continued.)

PNEUMONIA.

In early life the lungs are more frequently the seat of organic disease than any other organs in the body. Pneumonia is very common as a primary disease, and ranks first as a complication of the various forms of acute infectious disease of children. It is one of the most important factors in the mortality of infancy and childhood (page 39).

Cases of acute pneumonia are divided, from an anatomical point of view, into two principal groups: (1.) Broncho-pneumonia, also known as catarrhal and as lobular pneumonia. (2.) Lobar pneumonia, also known as croupous and as fibrinous pneumonia. These differ from each other as to the products of inflammation, the distribution of the disease in the lung, and somewhat as to the parts involved and the nature of the changes in them.

In broncho-pneumonia the large bronchi are the seat of a superficial inflammation, while in those of small size the entire bronchial wall is affected; the exudation into the air vesicles is mainly cellular, being made up of epithelial cells, leucocytes, and red blood-cells (Fig. 72), fibrin being either absent, or present only in small amount. In many cases there are marked changes both in the alveolar septa and in the interstitial tissue of the lung; resolution is often imperfect, and there is a strong tendency of the inflammation to pass into a chronic form, involving the connective-tissue framework of the lung. The lesion is widely and often irregularly distributed, usually being most marked in

the vicinity of the small bronchi, from which the inflammation spreads, and in the most superficial lobules of the lung.

In lobar pneumonia, bronchitis, when present, is usually superficial, the walls of the bronchi being very slightly or not at all affected; the same is true of the alveolar septa. The principal product of the inflammation is fibrin (Fig. 73), which fills the alveoli and the terminal bronchi, the cells being relatively few and chiefly leucocytes. The process is usually sharply circumscribed, involving an entire lobe or a part of a lobe. In most cases it clears up rapidly and completely, there being but little tendency to involve the framework of the lung in a chronic process.

While in typical cases the two forms of inflammation are quite distinct, there are seen many intermediate forms which partake of the characters of both, and one may be in doubt, even after a microscopical examination, into which group to place a case. It not infrequently happens

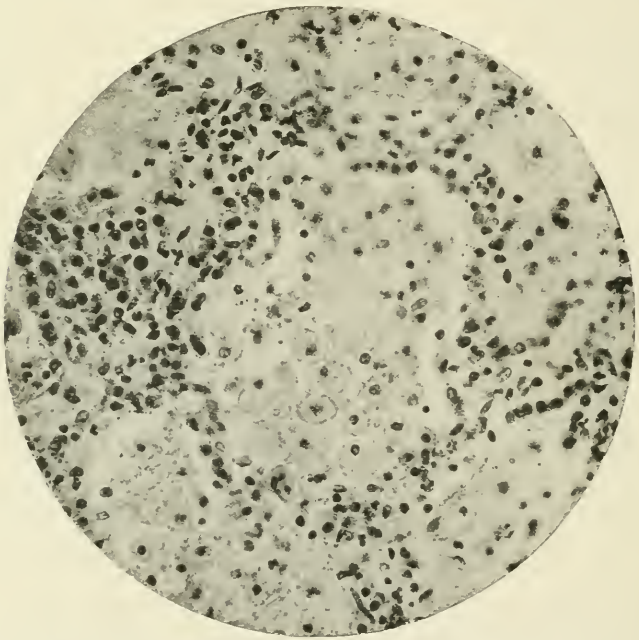


Fig. 72.—Broncho-pneumonia. The picture shows at its centre one entire air vesicle, and at its margin parts of four or five other vesicles; they are filled with large epithelial cells having small nuclei. There are also seen leucocytes with intensely black nuclei and narrow protoplasm. Between the cells is a finely granular material, which is the exudation fluid coagulated during the hardening process. The alveolar septa are somewhat infiltrated.—From Karg and Schmorl.

that both varieties of pneumonia are present in different parts of the same lung or in opposite lungs at the same time. These mixed forms are especially frequent during the second and third years; but during the first year, and after the third, the types are usually well marked.

The following table shows the relative frequency of lobar and broncho-pneumonia in three hundred and seventy cases,* nearly all taken from

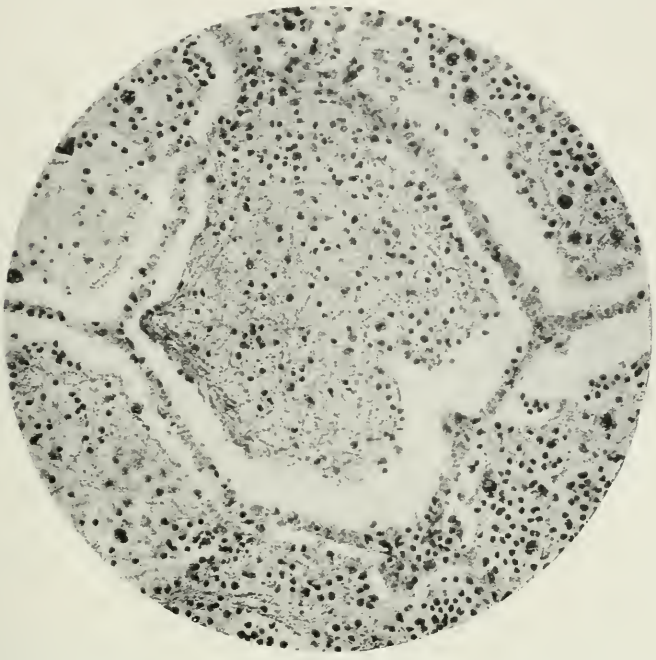


Fig. 73.—Lobar pneumonia. In the air vesicle shown in the picture there is a firm, close network of fibrin, in the meshes of which are leucocytes. At the lower part the exudation has contracted away from the wall in consequence of the process of hardening.—From Karg and Schmorl.

one institution (New York Infant Asylum). There are included all the cases of acute primary pneumonia occurring during a period of seven years:

Under six months,	broncho-pneumonia,	73	cases;	lobar	pneumonia,	11	cases.
Six to twelve "	"	96	"	"	"	29	"
Second year,	"	73	"	"	"	40	"
Third "	"	19	"	"	"	23	"
Fourth "	"	0	"	"	"	6	"
Totals,	"	261	"	"	"	109	"

Thus it will be seen that, of the cases of acute pneumonia occurring during the first two years, 25 per cent were lobar and 75 per cent were broncho-pneumonia.

When we come to a consideration of the micro-organisms with which the different forms of pneumonia are associated, we find that they do not

* The division was here made according to the predominant clinical or pathological features. Most of the doubtful cases were classed as broncho-pneumonia.

correspond to the anatomical varieties. Lobar pneumonia is regularly associated with the presence of the pneumococcus (micrococcus lanceolatus), which in most cases is found pure. In broncho-pneumonia no one form is always present. In the primary cases the pneumococcus is usually found, and in many cases it is alone. In the secondary cases there is almost always mixed infection. In measles and diphtheria the streptococcus is the principal form, such cases being usually of the worst type. In other secondary cases there are found the staphylococcus, and sometimes Friedländer's bacillus. Each of these varieties of bacteria may be found alone, but they are often associated, and with any of them may be found the pneumococcus, or other specific germs, most frequently the bacillus of influenza, diphtheria, or tuberculosis.

The reason why the same cause—the pneumococcus—in one case produces broncho-pneumonia and in another lobar pneumonia, is in part owing to the difference in the structure of the lung at the different ages—that of infancy being more bronchial, and that of older children more vesicular (page 460). Another reason is to be found in the constitution of the patient: in the very young and in feeble and delicate children, the process tends to become diffuse and the products are chiefly cellular; in those who are older and more vigorous it is likely to be circumscribed, with fibrin as its chief product; in the intermediate ages and intermediate conditions the types are often mingled.

Etiologically as well as clinically, lobar pneumonia is a single disease, usually running a regular self-limited course. Broncho-pneumonia, on the other hand, includes a number of quite distinct diseases, which are not only etiologically but clinically different. Sometimes when it is due to the pneumococcus it has more features in common with lobar pneumonia than with cases of broncho-pneumonia due to another kind of infection, such as the streptococcus.

The immediate source of infection of the lungs is the mouth, the nose, or the pharynx. All the forms of bacteria found in pneumonia are found in these cavities, some of them constantly, others only at certain times, especially during an attack of any of the acute infectious diseases. What part direct contagion plays in the spread of pneumonia can not be settled without fuller data than at present exist. There seems to be no doubt, from clinical observations alone, that the secondary forms, especially those complicating measles and diphtheria, are sometimes communicated in this way. This is probably not often true of primary cases, except in hospitals for infants where the rapid development of case after case in the same ward can not be well explained on any other hypothesis.

The different forms of pneumonia which will be considered are: (1) Acute broncho-pneumonia. (2) Acute fibrinous pneumonia. (3) Acute pleuro-pneumonia. (4) Hypostatic pneumonia. (5) Chronic broncho-pneumonia.

Tuberculous broncho-pneumonia will be discussed in the chapter devoted to Tuberculosis.

ACUTE BRONCHO-PNEUMONIA.

Synonyms: Catarrhal pneumonia, lobular pneumonia, capillary bronchitis.

This is essentially the pneumonia of infancy. Under two years, the great majority of the cases of primary pneumonia are of this variety, and throughout childhood nearly all the cases of secondary pneumonia. The term broncho-pneumonia describes a lesion rather than a disease, several quite distinct forms of infection being included under this head. Its mortality is high, because of the tender age of the patients in which the primary cases occur, and also because when secondary it complicates the most severe forms of the acute infectious diseases of children.

Etiology.—*Age.*—The 426 cases of broncho-pneumonia of which I have notes occurred as follows:

During the first year	224	cases, or	53 per cent.
" " second year	142	" "	33 " "
" " third "	46	" "	11 " "
" " fourth "	10	" "	2 " "
" " fifth "	4	" "	1 " "
	426		100

After four years broncho-pneumonia is very infrequent as a primary disease, although it is seen throughout childhood as a complication of the infectious diseases.

Sex.—In the primary cases males are more frequently affected than females, the proportion being five to four. In the secondary cases the sexes are about equally affected.

Season.—Of the cases referred to, 38 per cent occurred during the winter months, 31 per cent during the spring, 13 per cent during the summer, and 18 per cent during the autumn. While, therefore, nearly 70 per cent of the cases occurred in the cold months, broncho-pneumonia is seen throughout the year.

Previous condition.—Broncho-pneumonia affects all classes, but is most frequent in children having poor hygienic surroundings, especially in inmates of institutions, and in those previously debilitated by constitutional or local disease. In 246 consecutive cases of primary pneumonia, 110 were in good condition prior to the attack, and 126 were delicate, rachitic, or syphilitic.

Previous disease.—The following table gives a good idea of the conditions with which acute broncho-pneumonia is most frequently seen; 443 cases were classed as follows:

Primary *	164
Secondary to bronchitis of the large tubes	41
Complicating measles	89
" pertussis	66
" diphtheria	47
" acute ileo-colitis	19
" scarlet fever	7
" influenza	6
" varicella	2
" erysipelas	2
	443

A large number of the patients had previously suffered from one or more attacks of bronchitis, and fifteen previously had broncho-pneumonia.

As an exciting cause, exposure to cold must still be classed among the potent factors of primary pneumonia.

Bacteriology.—Much light has already been thrown upon broncho-pneumonia by bacteriology, but many points still remain to be settled. In 1889 Prudden and Northrup † showed that the broncho-pneumonia of diphtheria was usually due to the streptococcus. In 1891 Mosny ‡ published a report upon 17 cases of broncho-pneumonia: 4 were primary, 7 were secondary to measles, 3 to diphtheria, and 1 to scarlet fever. In the 4 primary cases, the pneumococcus was found alone in 3, and the streptococcus alone in 1. In the 11 secondary cases, the pneumococcus was found in 3; in one of these, a case of measles, it was alone. The streptococcus was found in 10 cases—alone in 5, with the pneumococcus in 1, with the pneumococcus and Loeffler's bacillus in 1, with the staphylococcus in 2, with Friedländer's bacillus in 1; in one case Friedländer's bacillus was found alone, and in one case a peculiar streptococcus.

In 1892 Netter # published a report upon 42 cases. He has not separated the primary and secondary cases. Of 25 cases in which but one form of bacteria was found, the pneumococcus was present in 10, the streptococcus in 8, the staphylococcus in 5, and Friedländer's bacillus in 2. In the 17 cases of mixed infection, the streptococcus was present in 15, the pneumococcus in 9, the staphylococcus in 8, and Friedländer's bacillus in 4.

I am indebted to Dr. Martha Wollstein, Pathologist to the Babies' Hospital, for permission to include here the results of observations made by her but not yet published. I had the opportunity of observing most of the cases clinically, they having been treated in my wards. Thus

* It is probable that a number of cases complicating influenza were included among these primary cases.

† American Journal of the Medical Sciences, June, 1889.

‡ Étude sur la Broncho-Pneumonie, Paris, 1891.

Archives de Médecine expérimentale, January, 1892.

far 33 cases have been studied, 19 of which were primary and 14 secondary. Of the secondary cases, 2 complicated measles, 3 diphtheria, 3 marasmus, and 6 tuberculosis. The pneumococcus was found in 17 of the 19 primary cases, occurring alone in 9, with the streptococcus in 7, and with the staphylococcus in 1. Of the two remaining primary cases, the streptococcus was found alone in one, and with the staphylococcus in the other. Of the 14 secondary cases, the pneumococcus was present in 11, and alone in 2, both of these being cases of measles. The pneumococcus was associated with the streptococcus in 1 (a case of diphtheria), with the staphylococcus in 2 (both marasmus cases), with the tubercle bacillus in 2, with the tubercle bacillus and streptococcus in 3, with the tubercle bacillus and the staphylococcus in 1. Of the three cases in which the pneumococcus was absent, all showed the streptococcus—once alone, once with the staphylococcus, and once with the tubercle bacillus.

Our present knowledge of the bacteriology of broncho-pneumonia may be summarized as follows: In the primary cases the pneumococcus is nearly always present, and in a large proportion of the cases it occurs alone. In cases of mixed infection it is most frequently associated with the streptococcus. The secondary cases are usually due to a mixed infection. The pneumococcus is found in a large number of these cases, but plays a much less important part than the streptococcus, particularly in cases complicating measles, diphtheria, and scarlet fever. The staphylococcus is next in point of frequency in the mixed cases, and it may occur alone. Still less important is the part taken by Friedländer's bacillus both in primary and secondary cases. The association of the pneumococcus in all of the six tuberculous cases studied by Dr. Wollstein is of special interest, as it explains what is so often observed clinically, that in cases of tuberculous broncho-pneumonia the symptoms are indistinguishable from the simple form. Three of these cases ran the course of simple acute broncho-pneumonia, and were so diagnosticated during life.

We have not yet sufficient data definitely to connect the different forms of infection either with any set of lesions or with any group of clinical symptoms. The cases due to streptococcus infection are usually the worst forms, and are apt to show widely disseminated lesions. The cases in which the onset and clinical history resemble lobar pneumonia, and where there are found extensive areas of consolidation, and often excessive pleurisy, are usually due to the pneumococcus.

Lesions.—The term broncho-pneumonia is now generally adopted as a generic one, and it is to be preferred either to lobular or catarrhal pneumonia, as it gives prominence to the bronchial element in the inflammation. The process may begin in the larger tubes and gradually extend to those of smaller calibre, finally involving the pulmonary lobules in which these tubes terminate; or it may extend to the air vesicles which surround the tube in its course through the lung, so that in whatever

direction the lung is cut, there are seen surrounding the small bronchi, zones of pneumonia (Fig. 74). In other cases the process seems to begin almost at the same time in the small bronchi and the air vesicles, as both are found involved, even when death occurs within a few hours of the first symptoms.

There are, however, cases in which the parts of the lung affected bear no relation to the bronchi—where there are found simply smaller or larger

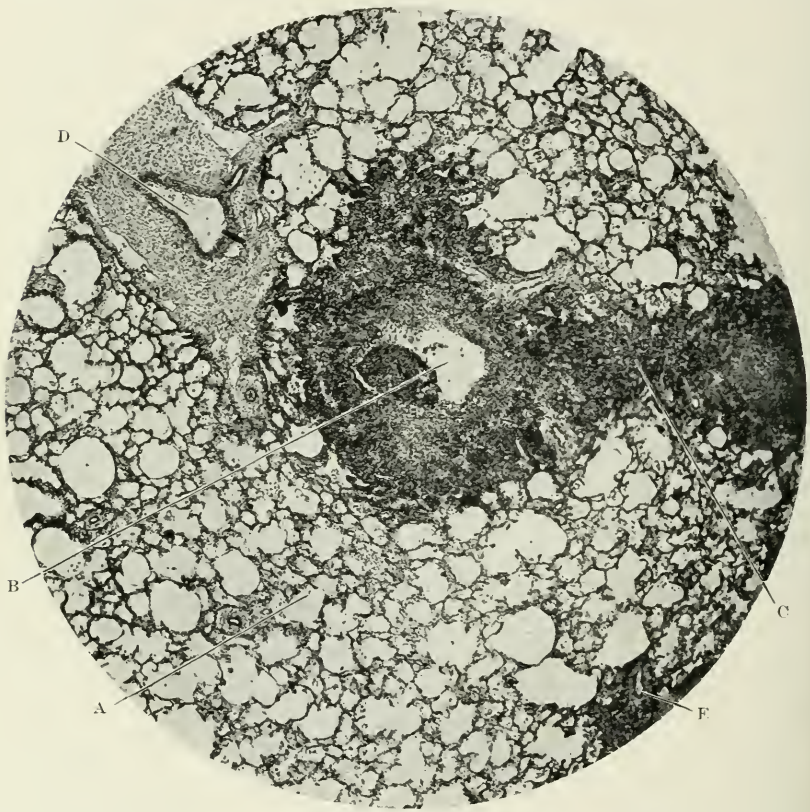
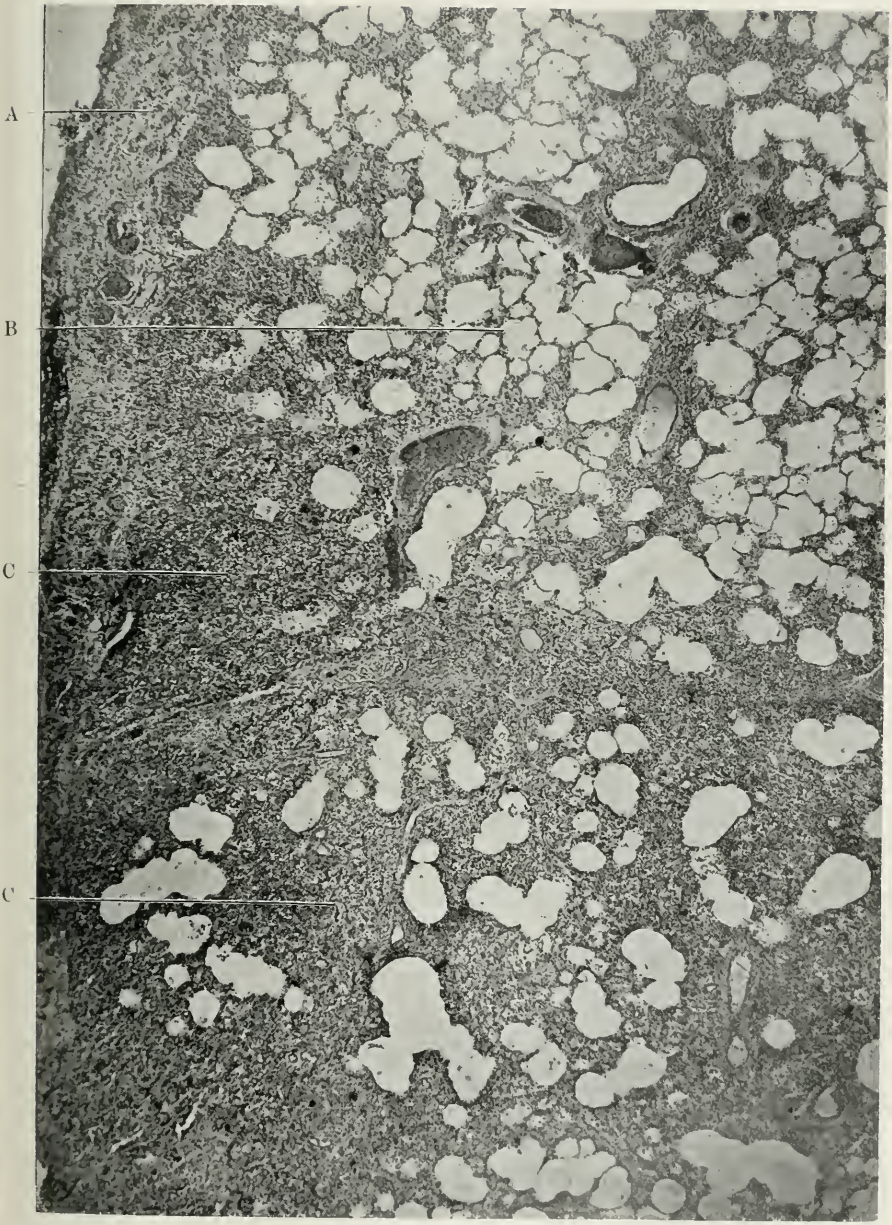


Fig. 74.—Broncho-pneumonia, with thickening of a small bronchus. In the centre of the picture is seen a small bronchus, B, which is cut somewhat obliquely, so that the degree to which its wall, C, is thickened is well shown. It is partially filled with pus, its mucous membrane is nearly destroyed, and its walls greatly thickened from infiltration with leucocytes. This infiltration extends to the lung tissue in the neighbourhood; it forms a peri-bronchitic zone of pneumonia. Elsewhere in the picture the lung tissue, A, is practically normal. D is a small blood-vessel. E is another smaller bronchus. Throughout the lung everywhere accompanying the small bronchi similar changes were seen, in addition to which there were present some large areas of consolidation. The disease was of four and a half weeks' duration; the child, five months old.

areas of pneumonia irregularly scattered through the lung, usually near the surface (Plate XII). From the distribution of the lesions such cases might better be termed lobular than broncho-pneumonia.

Much has been said in the past about pulmonary collapse from ob-

PLATE XII.



ACUTE BRONCHO-PNEUMONIA.

Primary pneumonia in a child two years old, showing the irregular distribution of the hepatization and its incomplete character. A is the pleura somewhat thickened; B, lung tissue which is practically normal; C C are hepatized areas, scattered through which are groups of air vesicles still containing air. (Slightly magnified.)

struction of the small bronchi, as an antecedent condition to this form of pulmonary inflammation. So far as my own observations go, there has been adduced but little evidence that this is the rule, or, indeed, that it often occurs. Even in autopsies made very early in the disease, but little collapse was found, most of the cases supporting the view of Delafield, that when the disease extends from the bronchi to the air cells it involves those surrounding the tube quite as regularly as those to which the tube leads.

The following observations are made from a study of 170 autopsies of which I have records, microscopical examinations having been made in about one third of the number.

Seat of the disease.—In 82 per cent of the autopsies extensive disease was found in both lungs. The parts most affected were the lower lobes posteriorly; next to this the posterior part of both the upper and lower lobes. The left lower lobe was more extensively diseased than the right in over two thirds of the cases. Only a single lobe was involved in but 9 per cent of the cases. It is not common for the disease to be situated in the anterior portion of the lung only, but when this occurs the right apex is the most frequent seat.

Just as the clinical symptoms of broncho-pneumonia follow no regular type, so the pathological process does not pass through a regular order of changes such as are seen in lobar pneumonia. There are a certain number of cases which appear to follow tolerably well-defined stages of congestion, red hepatization, gray hepatization, and resolution; but the disease may be arrested at any of the stages and the case recover, or death may occur at any stage and there may be found at autopsy different portions of the lung representing all the stages mentioned. In considering, therefore, the lesions of broncho-pneumonia, it seems best to describe the condition in which the lungs are found at the various periods when death is likely to occur, rather than to attempt to describe the different stages of the disease, as in lobar pneumonia.

1. *The acute congestive form* (*acute red pneumonia*).—This is the condition in which the lung is usually found if death occurs during the first two or three days of the disease. In the cases severe enough to cause death in the first twenty-four hours, very little can be seen by the naked eye except acute congestion. The vessels of the pleura are distended, and there may be small superficial hæmorrhages. Both lower lobes are usually heavy and dark-coloured. There is to the naked eye no consolidation. All, or nearly all, the lung can be inflated. On section, there is found intense congestion with some œdema. When the process has lasted a little longer the affected areas are more sharply defined. These, usually the posterior portions of both lungs, are of a brownish-red colour, and appear partially hepatized, although with a little force they may in most cases be inflated. After section, pus and mucus flow from the divided bronchi, and the whole lung may be more or less congested or œdematous.

The microscope alone reveals the fact that these are not cases of simple pulmonary congestion or bronchitis of the finer tubes. In one case in which death occurred twelve hours from the first symptoms, I found well-

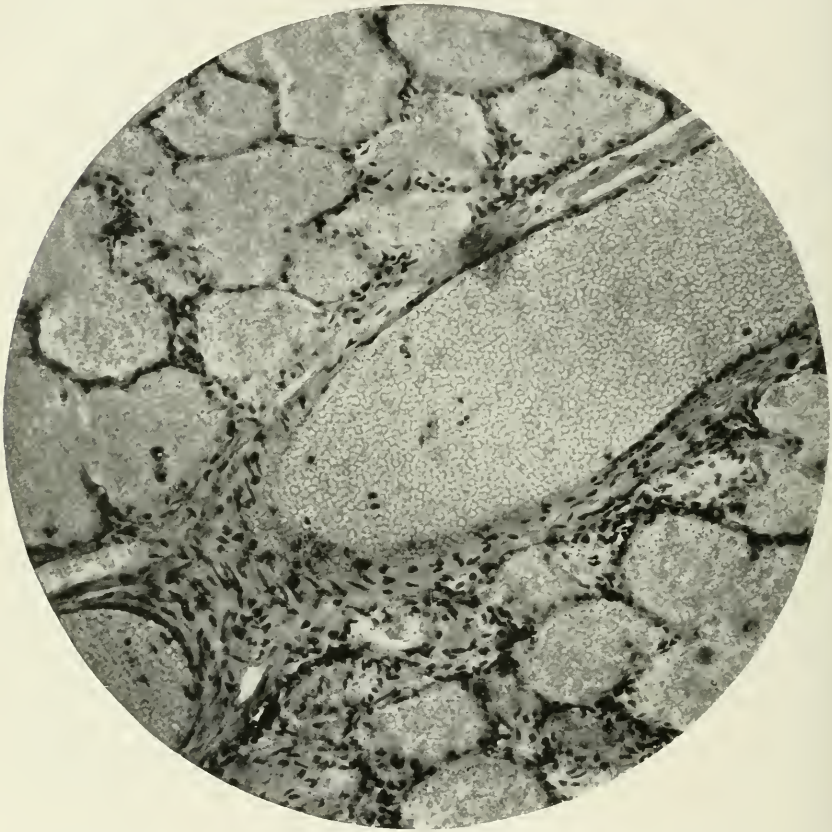


Fig. 75.—Acute broncho-pneumonia with intra-alveolar hæmorrhage (highly magnified). In the picture is shown a small vein, which, as well as the surrounding alveoli, is filled with blood-cells. In other respects the lung shown is normal. This is from the border of a consolidated area. Child fifteen months old; pneumonia of ten days' duration, with a severe exacerbation forty-eight hours before death, temperature 106° F. Extensive hæmorrhagic areas were scattered through the lung most affected.

marked evidences of inflammation of the air vesicles. In these hyper-acute cases, the microscope shows great distention of all the small blood-vessels of the affected area, and small or large extravasations of blood just beneath the pleura, into the alveoli (Fig. 75) and interstitial tissue of the lung. In some cases these hæmorrhages form the most striking feature of the lesion. The air vesicles are partially, some almost completely, filled with red blood-cells, swollen and desquamated epithelial cells, and a few leucocytes (Fig. 72). The red blood-cells predominate. The inflammation may be diffuse, involving nearly a whole lobe, or in small areas in the

neighbourhood of the small bronchi (Fig. 76). The mucous membrane of the large and small bronchi is the seat of catarrhal inflammation, and the walls of the latter are infiltrated with round cells.

When the process has lasted from twenty-four to forty-eight hours all the changes described are more marked, but the red colour of the inflammatory products still persists. Such cases give during life only the signs of congestion and bronchitis.

2. *The mottled red and gray pneumonia.*—This is the usual appearance when the disease has lasted somewhat longer, and is found in most of the cases dying between the fourth and fourteenth days. There are usually at this time quite large areas of consolidation, sometimes affecting nearly an entire lobe, so that at first sight the case may resemble lobar pneumonia. This is sometimes described as the "pseudo-lobar" form. The extent of

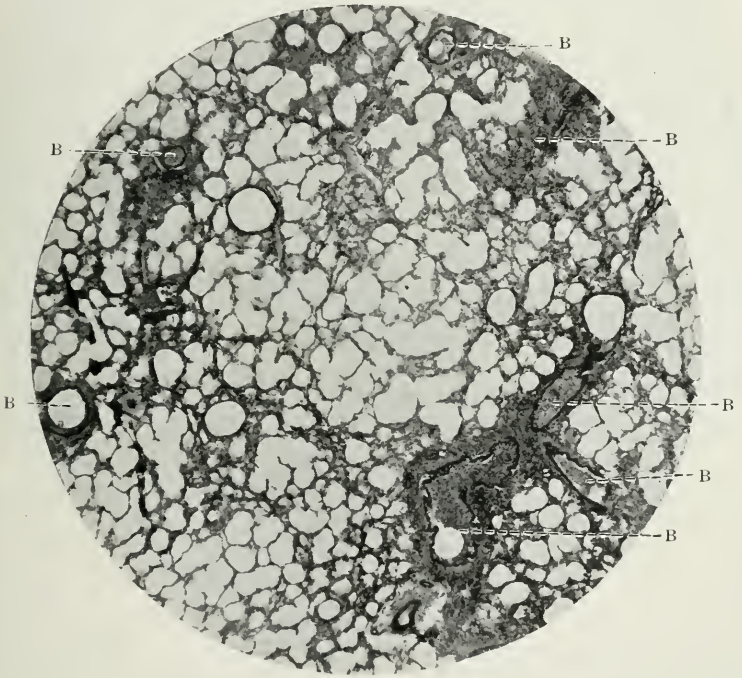


Fig. 76.—Early stage of broncho-pneumonia. There is shown at B B B small bronchi, some of which at the right of the picture have been cut somewhat obliquely, and hence appear irregular in shape. These bronchi everywhere contain pus; the air cells in the neighbourhood are partially filled with leucocytes. The intervening pulmonary tissue is normal. Child five months old.

these areas depends largely upon the duration of the disease. In most cases there is pleurisy over the consolidated portions. This may cause the lung to adhere to the chest wall, the firmness of the adhesions depending upon the duration of the process. The surface of the lung is usually of a mot-

tled red and gray colour; it often has a granular feel, due to the consolidation of some of the superficial lobules of the lung. On section, it is rarely found that an entire lobe is consolidated, the superficial portion

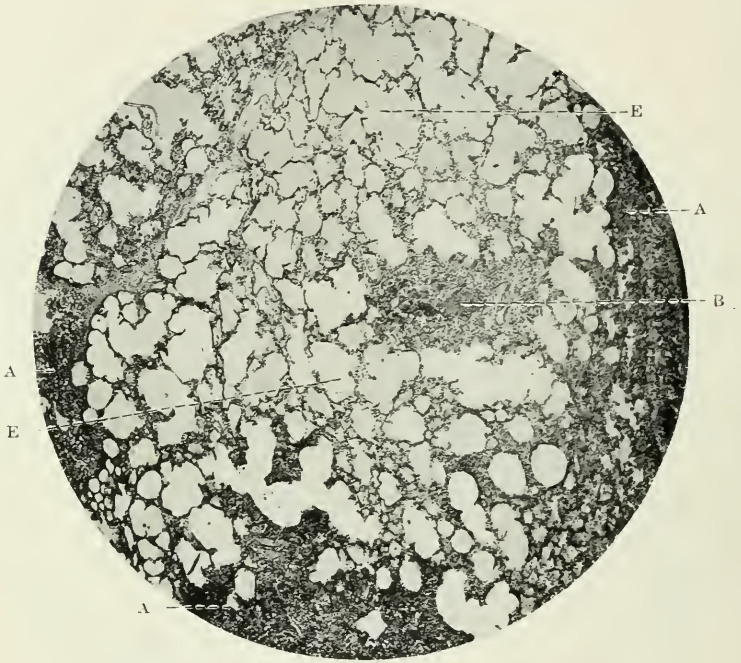


Fig. 77.—Acute broncho-pneumonia. In the centre is shown a small bronchus, B, with a zone of pneumonia about it. The greater part of the section is made up of emphysematous lung tissue, E E, showing dilatation of the alveolar spaces and rupture of some of the alveolar septa. At the border, A A A, are seen the margins of consolidated areas of lung.

being most affected, while the central part is normal or only congested. The colour is mottled, like that of the surface. In some places the hepatization appears complete; in others the hepatized areas are separated by healthy, congested, or emphysematous lung tissue (Fig. 77). The gray areas surround the small bronchi and vary in size from a pin's head upward. The smallest ones look very much like miliary tubercles. The larger ones are seen where the process has existed for a longer time and has gradually invaded the contiguous air cells. If the lung is cut parallel with the bronchi, there may be seen small gray striæ of pneumonia along their course (Fig. 74, C). From the cut bronchi, pus flows quite freely on pressure. The bronchial walls can often be seen even by the naked eye to be thickened. The parts affected are usually the posterior portions of the lower lobes of one side, the remainder of the lobes being congested or œdematous, while in front the lung is emphysematous.

Under the microscope the smaller bronchi (Figs. 74 and 78) are seen

to be much thickened and infiltrated with leucocytes. The gray areas surrounding the bronchi are made up of groups of air vesicles, which are packed with leucocytes (Figs. 79 and 80). Fibrin is sometimes seen in small amount, also red blood-cells and desquamated epithelial cells, but the leucocytes predominate. Surrounding the areas densely infiltrated are groups of air vesicles which are normal or congested, or which show only the earlier stages of the inflammatory process. Under the micro-

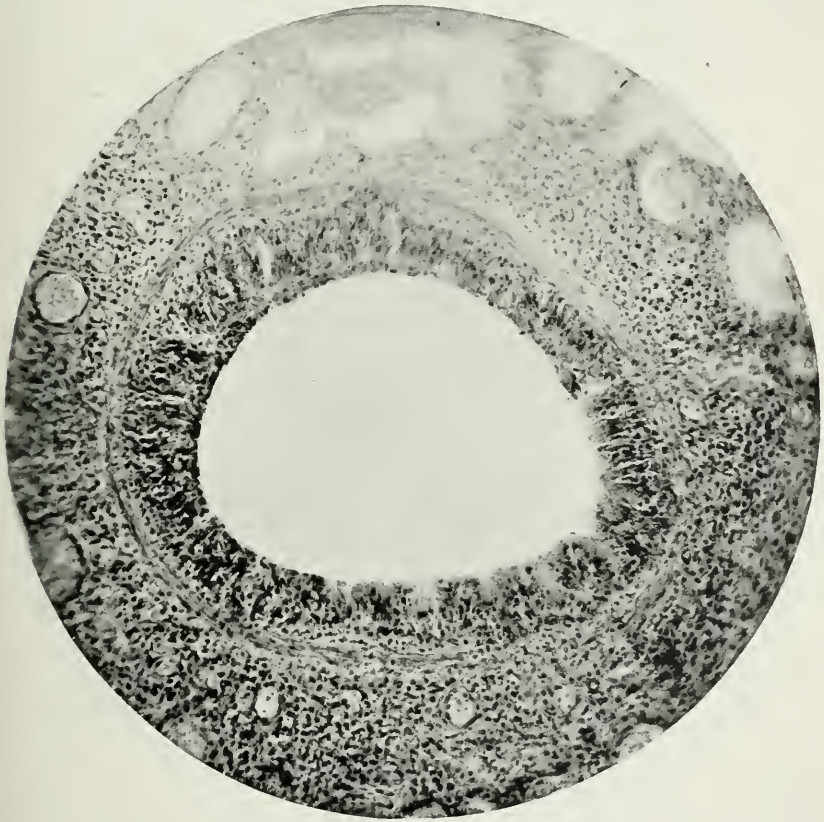


Fig. 78.—Thickening of a small bronchus in subacute broncho-pneumonia following pertussis; child ten months old. The epithelium is well preserved, but the walls of the bronchus are infiltrated with leucocytes and show some enlarged blood-vessels. Magnified about thirty diameters. All the small bronchi in the lung examined showed similar changes. In addition, there were superficial areas of consolidation in both lungs behind.

scope, even better than to the naked eye, is shown the irregularity of the consolidation.

3. *Gray pneumonia* (*persistent broncho-pneumonia*).—This form is seen in protracted cases where there have been continuous symptoms usually for from three to eight weeks; it is not very uncommon. The pleuritic adhesions are more general and firmer. The amount of lung

involved may be very great, often nearly the whole of both lungs posteriorly. The affected lung appears completely consolidated and slightly enlarged. On section, it is of a nearly uniform gray colour, sometimes of a yellowish gray. On pressure, pus exudes from the smaller and larger bronchi. The bronchial walls are markedly thickened, and in some places there may be a slight dilatation of the smaller bronchi. The part of the lung not consolidated may be almost white, owing to vesicular emphysema. In some cases there is also interstitial emphysema. Small cavities containing pus may be found in the lung. The bronchial glands

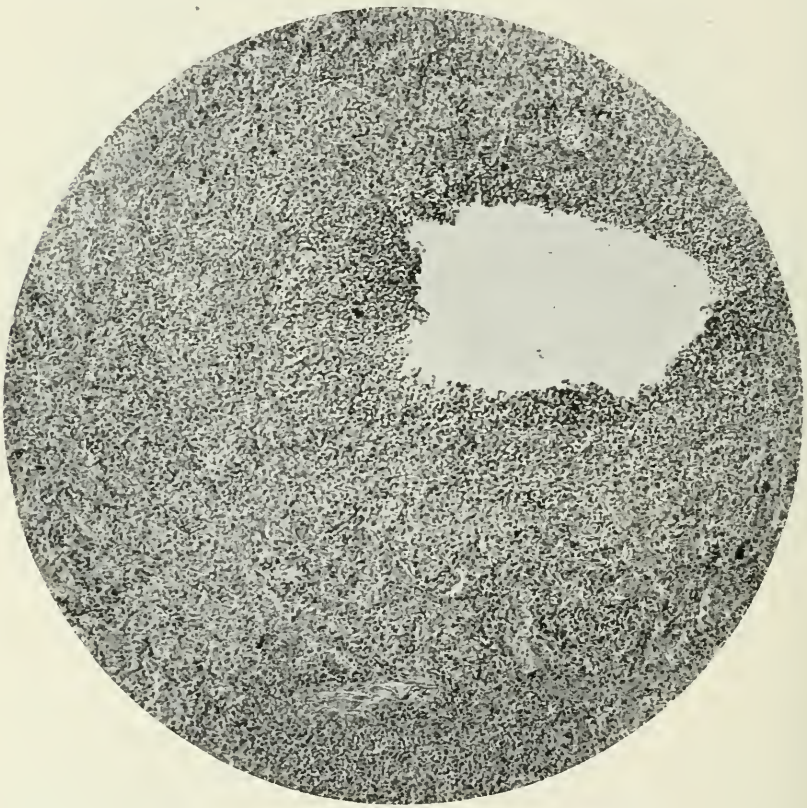


Fig. 79.—Broncho-pneumonia. Dense infiltration of pus cells in and about a small bronchus; under a low power. The cavity shown in the specimen is a cross-section of one of the small bronchi, which is partially filled with pus cells; the epithelium is destroyed. The bronchial wall and the pulmonary tissue in the neighbourhood are so densely infiltrated with leucocytes that almost every trace of normal structure is effaced. Child fifteen months old, disease of four weeks' duration. Extensive areas like this were found in both lungs.

are frequently swollen to the size of a large bean, and are of a reddish-gray colour.

The microscope shows that the air vesicles of the consolidated portions

are distended chiefly with leucocytes, but there are also epithelial and connective-tissue cells. The alveolar septa may be so much thickened as to

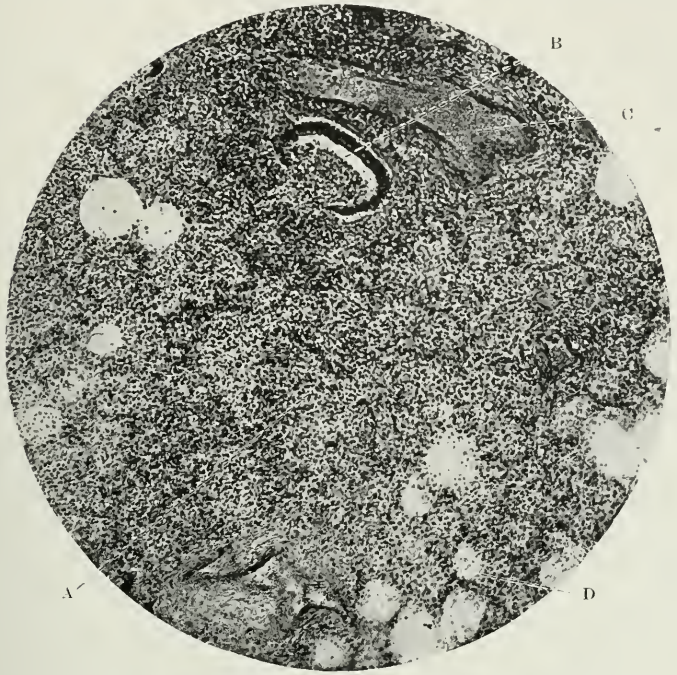


Fig. 80.—Acute broncho-pneumonia, under a low power, showing a portion of the lung, A, densely infiltrated with leucocytes. At B is a small bronchus, the wall of one side partly broken down by the inflammatory process. At the margin of the specimen D are seen alveoli more or less filled with epithelial cells and leucocytes. At C is a small blood-vessel. In other parts of the lung small gangrenous areas were seen. The disease was of nine days' duration, the child seven months old.

encroach upon the alveolar spaces (Fig. 81). Complete resolution is then impossible.

Terminations.—Death may occur at any stage, or the pathological process may be arrested at any stage and the case go on to recovery. Resolution may take place before any consolidation recognisable by physical signs has occurred; in such cases it is usually rapid and complete. If there has been consolidation, resolution may take place after two or three weeks and be complete, or it may be delayed for five or six weeks and still be complete. In many cases, especially those in which it is delayed, resolution is only partial, and there are relapses or recurring attacks. After the first, or after several attacks, there may develop a chronic interstitial pneumonia; or simple pneumonia may be followed by tuberculosis. Such cases as these are to be carefully distinguished from the much more frequent ones in which the broncho-pneumonia has been tuberculous from the outset.

Associated Lesions of the Lungs.—*Pleurisy* is almost invariably found over every large area of consolidation, and in cases of more than four days' duration; while in most of those fatal within the first two or three days the pleura is normal or only congested. It is seen in all grades of severity, from a slight gray film of fibrin that can hardly be stripped off, to a yellowish-green exudation one fourth of an inch thick. A small amount of serum—one or two ounces—in the pleural sac is not uncommon, but a large serous effusion is very rare. Cases in which there is an

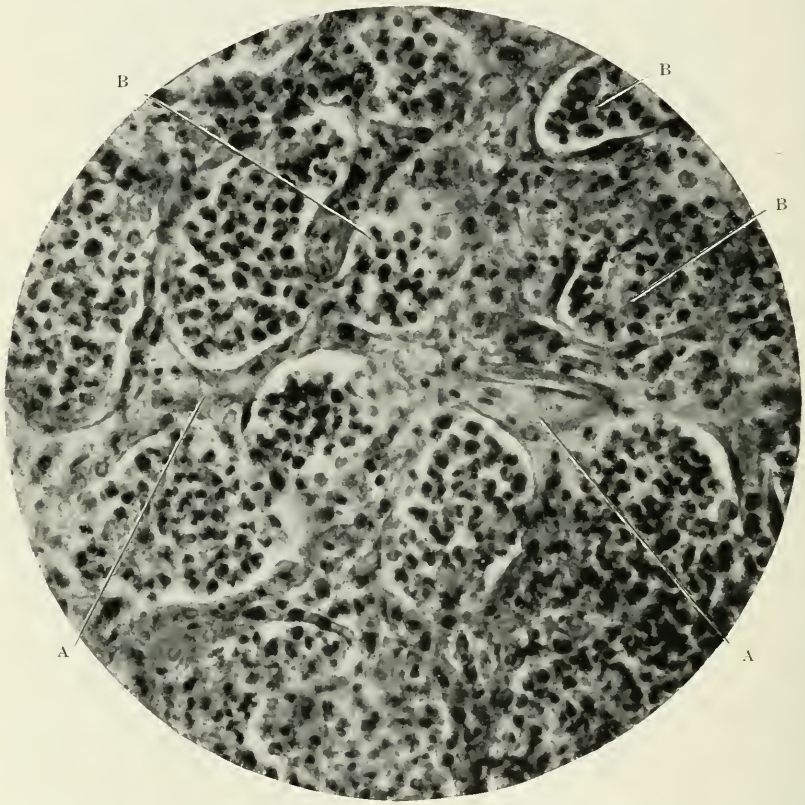


Fig. 81.—Persistent broncho-pneumonia; highly magnified. There are shown at A A marked thickening of the alveolar septa, encroaching upon the alveolar spaces. All the alveoli, B B, are densely packed with leucocytes. A similar condition also through nearly the whole of the affected lung. (For history and temperature, see Fig. 90.)

excessive inflammation of the pleura are considered elsewhere under the head of Pleuro-Pneumonia. Empyema occurs both during the stage of acute inflammation of the lung and while this is subsiding, but it is less frequent than in lobar pneumonia.

Bronchial glands.—In all the recent acute cases these are swollen and red; the usual size is that of a pea or a bean. They show microscopically

the usual changes of acute hyperplasia. In protracted cases, and after repeated attacks, they may be two or three times the size mentioned, and of a gray colour. It is rare that they are large enough to give rise to symptoms unless they become the seat of tuberculous deposits.

Emphysema.—In almost all cases a certain amount of emphysema is present, it being more marked in the protracted cases. It is usually vesicular, involving the greater part of the upper lobes in front and the anterior margin of the lower lobes. Occasionally interstitial emphysema is seen, forming either large striæ upon the surface of the lung, or blebs of considerable size along the anterior margin. This may occur even in cases uncomplicated by pertussis or laryngeal stenosis.

Gangrene.—Gangrenous areas were found in six of my cases. In four of these the pneumonia was primary, in one it followed diphtheria, and in one ileo-colitis. It occurred in scattered areas of a grayish-green colour, varying from one fourth of an inch to two inches in diameter.

Abscesses of the lung are by no means uncommon. They were noted in seven per cent of my autopsies. They are usually minute and multiple, varying in size from one sixth to one half inch in diameter. Sometimes a portion of a lobe is fairly honeycombed with minute abscesses. In one case a large abscess was found occupying the greater part of a lobe, the symptoms resembling those of empyema. Abscesses are usually found in regions where the inflammatory process has been especially intense. They may be found in prolonged cases, in those of unusual severity, as shown by excessively high temperature and rapid extension of the disease, and in very delicate subjects. The microscope shows that these abscesses usually begin as an accumulation of pus in the small bronchi, whose walls become softened and break down on account of the intensity of the inflammation (Figs. 79 and 80). They may be superficial, but are more commonly in the interior of the lung; they contain yellow pus and sometimes broken-down lung tissue. Such abscesses can not be recognised clinically, and they are associated with other conditions which render the case almost certainly fatal.

The lesions in other organs will be considered under Complications.

Symptoms.—The clinical picture presented by broncho-pneumonia is an exceedingly varied one. There is no typical course. The cases differ from each other very markedly, but they may be divided into a few quite distinct groups.

1. *The acute congestive type.*—This may be seen at any age, but is more frequent in young infants. It may be either primary or secondary, being not uncommon in either form. Its symptoms are few and irregular, and the disease is often unrecognised. The entire duration may be only twenty-four hours. High temperature, extreme prostration, cyanosis, and rapid respiration may be the only symptoms. The temperature varies between 104° and 107° F., usually rising steadily until death occurs. The

prostration is extreme from the outset, the patient being overwhelmed by the suddenness and severity of the attack. Cyanosis is frequently present, and is almost always seen shortly before death. The respirations are from 60 to 80 a minute, but in most cases not strikingly laboured. Cough is frequently absent. Cerebral symptoms are often marked. There are dulness and apathy, sometimes quite profound stupor, and not infrequently convulsions just before death. The physical signs are few and inconclusive. There is often nothing abnormal except very rude breathing over both lungs behind; sometimes the breathing on one side is feeble, and on the other much exaggerated. There may be no râles whatever, and no change in the percussion note.

The suddenness and severity of these symptoms are something which it is hard for one who has not observed them to appreciate. I have known an infant to die in twelve hours from the time in which it was apparently in perfect health, and had an opportunity to confirm the diagnosis of pneumonia by a microscopical examination of the lung. The diagnosis can not be positively made during life, and in most of the cases the disease passes under some other name. It is often regarded as malignant scarlet fever or measles with suppressed eruption, or cerebro-spinal meningitis.

If the children are sufficiently strong to withstand the first onset of violent symptoms, they may recover completely in four or five days, the lung clearing up very rapidly. In other cases these grave symptoms may abate in a day or two, to be followed by those of ordinary broncho-pneumonia, which runs its usual course.

The symptoms of some of these cases may be explained by the sudden intense engorgement of the lung, which, owing to the small size of the air vesicles, interferes with its function almost as much as does consolidation. In other cases the symptoms are not so much due to the lungs, as the result of a general pneumococcus infection. A case lately came under my notice in which death occurred after a thirty hours' illness, where the pneumococcus was found by culture in both kidneys, spleen, heart's blood, and both lungs.

2. *Acute disseminated broncho-pneumonia* (*capillary bronchitis*).—Although the symptoms in this class of cases are chiefly due to the bronchitis, I have never failed to find at autopsy evidences of pneumonia also. These are not very common cases. The process begins as an inflammation of the medium-sized and small bronchi, but not of the finest bronchi. The onset is acute, with fever, very rapid and laboured breathing, severe cough, moderate prostration, and in most cases cyanosis.

The temperature is not high, usually only from 100° to 102° F., and it often continues so for three or four days. The pulse is rapid, and at first is full and strong. The respirations are exceedingly rapid, often from 80 to 100 a minute. There is dyspnœa with marked recession of all the soft parts of the chest during inspiration. Cough is always present, usually

severe, and sometimes almost incessant. The prostration is not so great as in the cases previously described, and the development of the symptoms is much less rapid.

There are at first sibilant and afterward subcrepitant râles over the entire chest, with which are usually mingled coarser moist râles. There are no evidences of consolidation. The respiratory murmur is everywhere feeble, but not otherwise altered. Percussion generally gives exaggerated resonance, owing to the emphysema which is present, the note being sometimes almost tympanitic.

The symptoms may gradually increase in severity until death takes place by the third or fourth day, from respiratory and cardiac failure. There is usually marked cyanosis, and toward the end rapidly increasing prostration. Just before death the temperature often rises rapidly to 106° or 107° F. At the autopsy there are found evidences of bronchitis of the tubes of all sizes, and minute zones of pneumonia about the smaller bronchi. The lungs are generally in a state of hyper-inflation, on account of which they do not collapse on opening the chest. There may be in addition extensive congestion or œdema, the development of which has been the immediate cause of death.

In cases which do not prove fatal there is usually by the third or fourth day great improvement in the general symptoms; the finer râles may disappear, and the coarse ones become more and more prominent. By the end of a week there may be complete recovery. Instead of this, there may be a continuance of the constitutional symptoms, and disappearance of the fine râles in front only, while behind there are gradually added to them the signs of consolidation in one of the lower lobes near the spine. From this time the case may progress as one of ordinary broncho-pneumonia.

The prognosis in this class of cases is very much better than in the congestive variety, recovery being probable unless the patients are very young or very delicate infants.

3. *Broncho-pneumonia of the common type.*—When primary, this usually begins suddenly with symptoms not unlike those of lobar pneumonia. This was the mode of onset in two thirds of my cases. In only ten per cent was the pneumonia preceded by bronchitis of the large tubes. In these the symptoms of bronchitis may be slowly (Fig. 91, p. 504) or rapidly (Fig. 82) merged into those of pneumonia. When the onset is sudden it is marked by high fever, frequently by vomiting, rarely by convulsions. In addition there are rapid respiration, cough, prostration, and sometimes cyanosis. The symptoms are more distinctly pulmonary than is generally the case in lobar pneumonia.

The temperature, as a rule, is high; rarely is it continuously so, but it is of a remittent type. The daily fluctuations often amount to four or five degrees. The fever usually continues from one to three weeks, and

gradually subsides. It is rare for it to terminate by crisis. Although, as a rule, we expect a high temperature with acute pneumonia, this is not invariable. Primary cases may run their course, and even terminate fatally, although the temperature has not been above 101° F. I have records of several such cases. A low temperature is more often seen in young and delicate infants than in those who are older and more robust.

The respirations are frequent and laboured; there is real dyspnœa. On inspiration, there are marked recessions of all the soft parts of the chest, and the alæ nasi dilate actively. The usual rapidity of the respirations is from 60 to 80 per minute; very often, however, it rises to 100, and on several occasions I have seen it even 120. Respiration generally seems more embarrassed than the action of the heart, and respiratory failure is a more frequent cause of death than cardiac failure. The pulse is always rapid—from 150 to 200 a minute—and when so it is often irregular. The pulse rate is of much less importance than its character. Early it is full and strong, but soon it becomes soft, compressible, and weak.

The prostration is usually moderate for the first day or two, but steadily increases as the lung becomes more and more involved. Toward the close of the disease there may be present all the symptoms of the typhoid condition.

Cough is much more constant than in lobar pneumonia, and more distressing; sometimes it is almost incessant. It disturbs rest and sleep, and may cause vomiting if the paroxysm occurs soon after eating. There is no expectoration. Mucus is sometimes coughed up into the trachea, or even the pharynx, to be swallowed again, or more frequently aspirated into the lung. If during a severe paroxysm the patient is turned upon his face or inverted, much of this mucus may be dislodged. A strong cough is a good symptom; suppression of the cough is always a bad symptom, indicating a loss of the reflex sensibility of the bronchial mucous membrane and feeble respiratory muscles.

Pain in the chest is not common, and is rarely an annoying symptom.

Cyanosis is present at some time in most of the severe cases. It may occur at the onset, or at any time during the course of the disease. It is usually due to sudden congestion of a portion of the lung not previously involved. Even when slight, it is always a danger-signal of respiratory failure, and when present only in the finger tips or lips indicates that the patient must be carefully watched and energetically treated. In the severe cases the whole body may be of a dull leaden hue.

Nervous symptoms at the onset are not so frequent as in lobar pneumonia, convulsions being rare; but late convulsions, particularly in the pneumonia which complicates pertussis, are exceedingly frequent, and usually fatal. Delirium may be present at any time during the attack. In infants this shows itself by excitement and inability to recognise the

nurse or mother. Occasionally patients present marked cerebral symptoms throughout the disease. In one of my cases nearly every symptom of tuberculous meningitis was present, the autopsy revealing only an extreme degree of cerebral anæmia. As elsewhere stated, the nervous symptoms depend not upon the location of the disease, but upon its extent, the intensity of the infection, and upon the susceptibility of the patient, such symptoms being especially common in rachitic children and in those suffering from pertussis.

Gastro-enteric symptoms are frequent in infancy, and are of much importance. Often there are from four to six stools a day, of a green colour, containing mucus and undigested food. These symptoms depend upon the feeble digestion which is associated with the febrile process, and are often from improper feeding. This may lead to vomiting, which is also due to over-medication or to severe paroxysms of coughing. Vomiting and diarrhœa add much to the danger of the attack, and not infrequently, when the issue is doubtful, turn the scale against the patient. In summer this complication is more frequent and is likely to be more severe. Distention of the stomach or intestines from gas may be the cause of severe symptoms, owing to the added embarrassment of respiration produced by this upward pressure. In infants it may lead to attacks of cyanosis, and even convulsions.

The urine in most cases is scanty, high-coloured, and loaded with urates. A trace of albumin is often present when the temperature is very high; but casts, renal epithelium, and a large amount of albumin are rare.

The following temperature chart (Fig. 82) is a good example of a very frequent course of primary pneumonia of moderate severity terminating

Fig. 82.—Temperature curve in typical broncho-pneumonia of the milder form.

History.—Male, sixteen months old; delicate child; previous bronchitis; onset gradual; signs of consolidation at left base on fifth day, but fine rales over both lower lobes behind; resolution slow, rales persisting for a long time in both lungs.

in recovery. In cases of this type the constitutional symptoms are not grave, and follow very closely the temperature curve.

The next chart (Fig. 83) illustrates a more severe but not uncommon course of the disease in which the fever is prolonged. The usual duration of cases of this type is between three and four weeks. The irregular fluctuations of the temperature, rarely touching the normal line, are exceedingly characteristic of broncho-pneumonia.

The chart shown in Fig. 84 is that of relapsing pneumonia. The first attack was fairly typical, with about the usual duration. Resolution

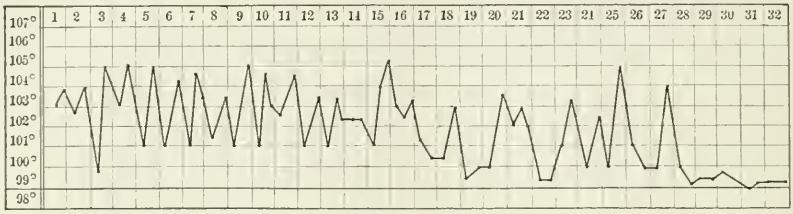


Fig. 83.—Temperature curve of broncho-pneumonia with a prolonged course; recovery.

History.—Female, eighteen months old; in fair condition; sudden onset. Early signs were localized, fine râles over left base; on fifth day signs of consolidation at left base, with râles on both sides behind. General symptoms of moderate severity. Signs of consolidation disappeared about a week after cessation of fever; râles persisted nearly two weeks longer.

had begun, and was apparently progressing favourably, when there was a return of the fever, accompanied by new signs in the chest, the second

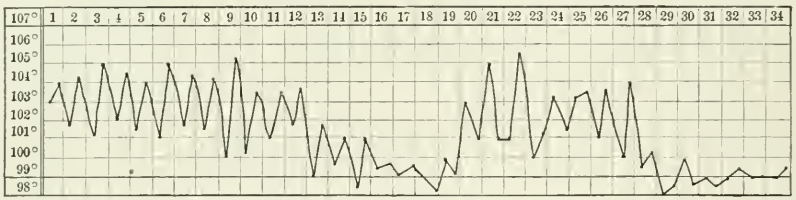


Fig. 84.—Temperature curve of relapsing broncho-pneumonia; recovery.

History.—Male, nineteen months old; delicate. Consolidation on sixth day in left lower lobe behind; two days later small area of consolidation in right lower lobe behind; many râles both sides; eighteenth day, signs of consolidation had disappeared, but many râles persisted. Accession of fever on nineteenth and twentieth days, accompanied by extension of disease as shown by new râles, but no evidences of consolidation during second attack. Slow resolution and convalescence.

attack being shorter and milder than the first. Very often the temperature falls to normal without any signs of resolution, and after an interval varying from two or three days to a week there is recurrence of the fever

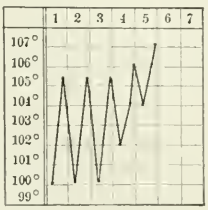


Fig. 85.—Temperature curve of broncho-pneumonia; fatal.

History.—Male, six months old; markedly rachitic; sudden onset. Signs first day were fine moist râles throughout the chest, marked prostration, and cyanosis; on third day, a small area of consolidation in upper lobe of left lung behind; increasing prostration, cyanosis, and death. *Autopsy.*—No pleurisy; consolidation at left apex behind, and posterior two thirds of left lower lobe; consolidation of right apex posteriorly, lower lobe intensely congested.

and other constitutional symptoms, the second attack frequently proving fatal.

A frequent course in fatal cases is shown in Fig. 85. The duration of the disease, instead of being five days as in this case, is often only three or four. The temperature at first fluctuates widely, then rises gradually until death.

Duration of the fever.—The following figures give the duration of the fever in 231 cases. The majority were primary; none were secondary to diphtheria, and only a few complicated measles. Of the 169 cases that were fatal—

There died during the first six days	25·0 per cent.
" " between the seventh and twenty-first days	55·5 " "
" " " " twenty-first and sixtieth days	19·5 " "
	100·0 " "

Of 78 cases which recovered, the duration of the fever was—

Less than seven days	11·5 per cent.
From seven to twenty-one days	66·6 " "
From twenty-one to ninety days	21·9 " "
	100·0 " "

Physical Signs.—In considering the signs of broncho-pneumonia, it is better to connect them with the different conditions in the lung than to group them in stages, as in lobar pneumonia.

(*a*) *Without consolidation.*—It can not too often be repeated that broncho-pneumonia may exist without signs of consolidation at any period during the course of the disease. When the attack is primary, the earliest signs are due to congestion of the lung, associated with bronchitis of the fine tubes, which is usually localized, but which may be general. If the disease has followed bronchitis of the large tubes, its signs are added. Congestion of the lung gives feeble breathing over the affected area, and occasionally slight dulness or diminished resonance. With this are found coarse sonorous, and finer sibilant râles, due to congestion and swelling of the mucous membrane of the larger and smaller bronchi respectively. These signs are soon replaced by very fine moist râles, which are usually localized in one of the lower lobes behind (Fig. 86). These localized fine râles are the first distinctive sign of broncho-pneumonia. Soon a change in the respiratory murmur is heard in the affected area, becoming feebler in intensity and higher in pitch. Elsewhere in the chest there may be coarse râles, due to bronchitis of the large tubes. In such cases the areas of pneumonia are so small and so scattered as to give in themselves no additional signs, and the case may go on to recovery without presenting anything more distinctive than the signs mentioned.

(*b*) *With areas of partial consolidation.*—In the lung at this time there are small areas of consolidation, generally superficial and separated

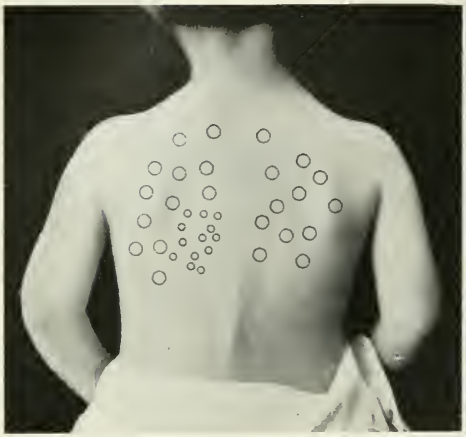


FIG. 86.—First stage. Coarse râles over both lungs; localized fine (subcrepitant) râles at the left base. No change in breathing sounds.

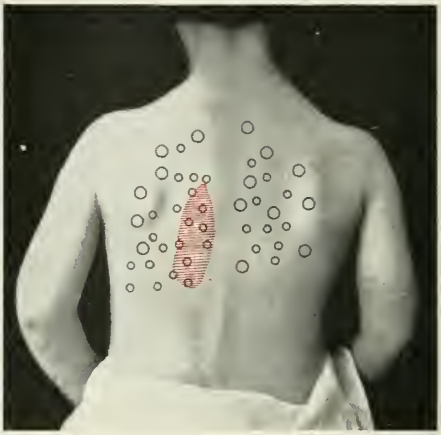


FIG. 87.—Second stage. Coarse and fine râles over both lungs behind; at left base an area of partial consolidation, with broncho-vesicular breathing, exaggerated voice, and very sharp râles.

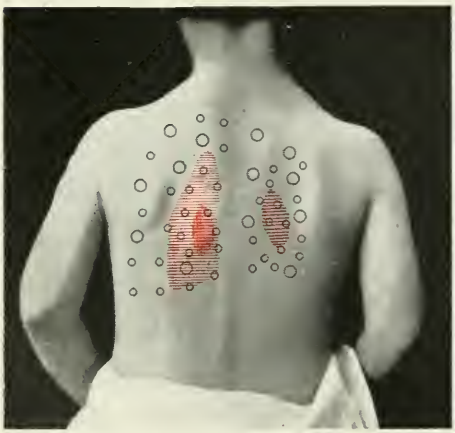


FIG. 88.—Third stage. A larger area of partial consolidation, and in the centre a small area of complete consolidation, with bronchial breathing and voice and slight dulness. Signs over the right lung similar to what were previously present over the left.

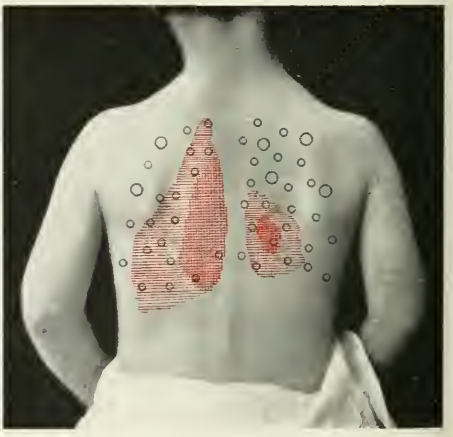


FIG. 89.—Fourth stage. Extensive disease of both sides; large area of complete consolidation on the left, with dulness, bronchial breathing and voice, and no râles; surrounding this, broncho-vesicular breathing, with many râles. Signs in the right lung similar to those previously present over the left.

NOTE.—The disease may stop at any one of these stages and resolution take place.

by healthy or congested lobules. Percussion in these cases usually gives negative results, but sometimes there is very slight dulness. The vocal fremitus is not usually altered. The fine moist râles may be heard over quite a large area, but at some point, usually near the spine, over one of the lower lobes, they are sharper, louder, higher pitched, and seem close under the ear (Fig. 87). Respiration is feebler here than elsewhere, and broncho-vesicular in quality, approaching bronchial breathing more and more as the consolidation increases. The resonance of the voice and cry is exaggerated.

(*c*) *With areas of consolidation more or less complete.* On percussion there is dulness, but surprisingly little in comparison with the other signs of consolidation present. It is due to the fact that the consolidated portion, though extensive, is superficial, and does not involve the lung to any great depth, and also that there are in the consolidated area many alveoli which still contain air (Plate XII). On palpation there is usually a slight increase in the vocal fremitus. On auscultation, there are still present the evidences of bronchitis, usually only behind, but sometimes over the entire chest. Coarse and fine râles are intermingled. Over the consolidated parts are heard bronchial breathing and bronchial voice. At the centre of these areas the bronchial breathing is pure and râles are usually absent, but at the margin râles are present and the breathing approaches the broncho-vesicular type (Fig. 88). The signs of consolidation thus are rarely sharply circumscribed as they are in lobar pneumonia, but shade off gradually. The consolidated area is at first small, usually in one of the lower lobes near the spine, but may gradually extend until nearly the whole of one or even both lungs behind are more or less completely solidified (Fig. 89). The signs are found as far forward as the axillary line, but usually stop here. Friction sounds may be heard over the consolidated areas, but very rarely except where signs of complete consolidation are present. It is often impossible to obtain any idea of the condition of an infant's lung during quiet, superficial respiration. Sometimes over a part which is completely consolidated there is heard only very feeble breathing, or the lung may be almost silent. If, however, the child be made to cry or to take a deep inspiration, both the bronchial breathing and râles are distinctly brought out. The intensity of the consolidation increases as the case advances, and the signs become more and more like those of lobar pneumonia. During resolution there is first a disappearance of the signs of consolidation, which may be quite rapid, but friction sounds and râles of all kinds often persist for three or four weeks longer.

The following statistics are of some interest, as showing the frequency with which signs of consolidation were found, and the day when they were discovered. Their value is increased by the fact that the children were under observation in an institution at the time they were taken sick, and that in all the fatal cases—thirty-six in number—in which signs of con-

solidation were absent, the diagnosis of pneumonia was confirmed by autopsy:

Consolidation noted on or before the fourth day	47	cases.
" " from the fifth to the seventh day	36	"
" " " the eighth to the twelfth day	12	"
" " after the twelfth day	9	"
No signs of consolidation	62	"
	166	"

In general, it must be borne in mind that in many cases signs of consolidation are never present, as the areas of pneumonia are small and widely scattered; that where there is consolidation it is usually incomplete, because there are small areas of healthy lung tissue between the hepatized portions; that the signs of consolidation usually shade off gradually; and that both sides are almost invariably involved, although one side usually to a greater degree than the other.

(4) *The protracted form—Persistent broncho-pneumonia.*—This is seen in primary cases, especially among delicate children, and it is not uncommon in pneumonia complicating pertussis. The onset and course of the disease for the first two or three weeks do not differ from an ordinary attack of moderate severity, but at the end of this period there is seen no tendency in the process to subside. The fever continues, but it is not high, and by physical examination it is found that the areas of consolidation are gradually increasing day by day, until sometimes the greater part of both lungs behind are involved. The air vesicles become so distended with cells that the signs of consolidation are more complete than in ordinary broncho-pneumonia. There is marked dulness, sometimes almost flatness; bronchial breathing is exaggerated in intensity, until it resembles cavernous breathing, and it may be impossible to distinguish between them. However, the fact that it is heard over so large an area, that it shades off gradually, and that it is accompanied by friction sounds, usually make a distinction possible.

The temperature in these protracted cases for the first two or three weeks is from 100° to 105° F.; but after this time it is generally lower —from 100° to 102° or 103° F. The course is not at all regular, but marked by frequent exacerbations and remissions. The general symptoms are those of progressive asthenia. There are continued wasting, anæmia, and steadily increasing prostration. The appetite is lost, often there is an aversion to food, and vomiting is easily excited if food or stimulants are forced. The stools show that even what food is taken is very imperfectly digested and assimilated. The skin becomes dry, and loses its elasticity; bed-sores may form; fine punctate hæmorrhages are seen over the abdomen, sometimes over the chest and extremities. The latter is always a very bad symptom, and I have never seen recovery where it was present.

The chart in Fig. 90 is typical of the course of one of these protracted

cases terminating fatally. The temperature shows four distinct exacerbations.

Death takes place from slow asthenia, usually after five or six weeks, but the attack may be prolonged for eight or ten weeks. The general

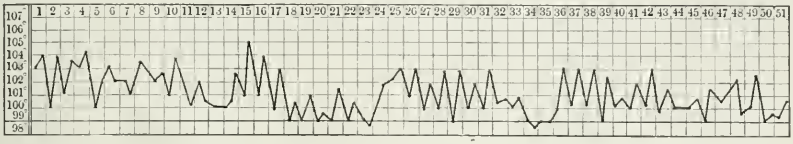


Fig. 90.—Temperature curve of persistent broncho-pneumonia, terminating fatally.

History.—Male, two and a half years old; healthy; sudden onset; for two weeks the only signs were very fine moist râles throughout both lungs, front and back. The râles in front in great part gradually cleared up; those behind persisted, but it was not until the thirty-fourth day that positive signs of consolidation were discovered in the left lower lobe behind; these signs gradually extended, and, before death, were present over nearly the whole left lung behind and over the right lower lobe. There were also friction sounds over both lungs. *Autopsy.*—Old and recent pleurisy with general adhesions; left lower lobe completely solid, patches of consolidation in left upper lobe. Right lower lobe about one half consolidated, with patches elsewhere. Bronchial glands large, but not cheesy. No evidence of tuberculosis upon either gross or microscopical examination (see Fig. 81).

symptoms, the temperature, and the wasting strikingly resemble cases of tuberculosis, and such is the diagnosis often made.

Although the majority of the cases in which the fever lasts over four weeks run the fatal course just described, such apparently hopeless cases occasionally recover. The temperature gradually falls lower and lower, until it remains at the normal point. For some time after this, often two or three weeks, little change can be seen, either in the general symptoms or in the physical signs. Gradually the appetite returns, the child is brighter and begins to take an interest in its surroundings, the cough abates, and little by little the signs in the lungs clear up, and the case may go on to complete recovery. Convalescence, however, is always slow, and may be interrupted by relapses, it being many months before health is fully restored. Although the signs of consolidation disappear in a few weeks, râles are apt to persist for a much longer time. It is probable in such cases, even though all signs of disease disappear from the chest, that the lung does not become quite normal, and relapses and second attacks are always possible. The general health may be so undermined that the child never regains his former vigour; yet in a surprising number of these cases recovery seems to be complete.

5. *Secondary pneumonia.*—(*a*) Complicating pertussis.—It is not often that pneumonia develops during the first two weeks of this disease. The most frequent time is from the third to the fifth week, when the patient has become exhausted from the previous severity of the pertussis. In two thirds of my cases the development of the pneumonia was gradual, following bronchitis of the larger tubes. The temperature chart shown in Fig. 91 illustrates well this course.

When the onset is sudden, the symptoms do not differ essentially from those of primary pneumonia. The temperature of pertussis-pneumonia is usually low, in a very large number of cases not rising above 103·5° F., and ranging most of the time from 101° to 103° F. These cases are very apt to be prolonged, the fever often lasting for three or four and some-

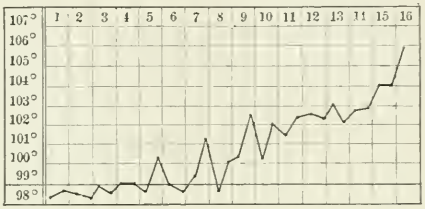


Fig. 91.—Temperature curve of fatal broncho-pneumonia, complicating pertussis.

History.—Male, six months old; delicate; pertussis for three weeks. Early signs of bronchitis of large tubes only; on the eleventh day signs of consolidation in right upper lobe. Increasing prostration, cyanosis, and death. *Autopsy.*—Large areas of consolidation in right middle and upper lobe, small scattered spots throughout left lung.

times even for six weeks. The physical signs of consolidation may persist for a long time after the temperature has become normal, and yet the case may recover entirely. I have seen one case in which complete recovery occurred after the signs of consolidation had persisted for six months, and another in which they had persisted for over eight months. Very often the signs continue during the entire attack of pertussis. Cerebral symptoms are common, especially toward the close of the disease. Of fifty-four fatal cases twenty-five had convulsions, and in twenty-two this was the mode of death. Only one case which developed convulsions recovered.

(*b*) Complicating measles.—In a small number of cases the pneumonia begins simultaneously with the invasion of measles, but generally not until the eruption appears. Instead of gradually falling to normal with the fading of the eruption, the temperature continues high. Any of the clinical types of primary pneumonia may occur in measles, the acute congestive variety which is fatal in two or three days, being especially common. In its course and duration the pneumonia of measles resembles the severe form of primary pneumonia. The broncho-pneumonia of scarlet fever differs in no way from that of measles.

(*c*) Complicating diphtheria.—In many cases this does not give a distinct clinical picture of its own, its symptoms being mingled with those of diphtheritic bronchitis, with which it is frequently associated. In others the forms resemble those seen in measles. The majority of cases occur as a complication of diphtheria of the larynx, although it is not infrequent in the septic cases in which only the upper air passages are involved. Pneumonia developing after laryngitis is usually seen within two days from

the beginning of laryngeal symptoms, and runs a very rapid course. In rare cases it may develop as late as the middle or end of the second week. When it complicates diphtheritic bronchitis, pneumonia is recognised by the high temperature, rapid breathing, and increased prostration, much more certainly than by the physical signs, which are always obscured by the laryngeal sounds. Percussion may aid in the diagnosis of consolidation where the signs on auscultation are doubtful. In the early cases, death usually occurs before the disease has advanced far enough to give the physical signs of consolidation, but in the late pneumonia, which develops more slowly, these may be present.

(*d*) Complicating influenza.—Without doubt many cases regarded as primary are really secondary to influenza, particularly when that disease is prevailing, for very often the pneumonia of influenza differs in no essential points from the primary form. There are, however, two types which are quite characteristic. In the first, high temperature and prostration exist for several days before there are any physical signs of pulmonary disease, and often before there are any symptoms pointing definitely to the lungs. Pneumonia may then develop and run its usual course. The second variety are the cases of short duration often lasting but three or four days, and sometimes only two, but with excessively high temperature and very severe general symptoms.

(*e*) Complicating ileo-colitis.—This is usually a somewhat subacute form of pneumonia, which is scarcely recognisable except by the physical signs. It is seen in the protracted cases of ileo-colitis, usually of the ulcerative variety, and occurs late in its course. The temperature is not high. Cough, pain, and dyspnœa are slight or entirely wanting. Accelerated respiration is frequently the only symptom suggestive of pulmonary disease. By physical examination there are found the usual signs, generally involving both lungs posteriorly. Very often pneumonia is not suspected during life, the constitutional symptoms being sufficiently explained by the intestinal lesions, although the autopsy discloses the fact that death was due to pneumonia.

Complications.—Those relating to the lungs have been described with the lesions. Pleurisy will be separately considered. Emphysema can rarely, and abscess and gangrene never, be recognised by the physical signs.

Purulent meningitis may complicate acute broncho-pneumonia. It was met with twice in one hundred and seventy autopsies. It is in all respects similar to that occurring with lobar pneumonia. Meningeal hæmorrhage was seen only once, and was the cause of death in a patient eleven months old, who a few days before was seized with convulsions, followed by a gradually increasing stupor, which continued until death. The hæmorrhage covered the entire convexity of the brain. Endocarditis is extremely rare; it was not observed in any of my cases. Acute

pericarditis was seen but twice, in both cases complicating pneumonia of the left side. Complications referable to the digestive tract are quite common. Herpetic stomatitis is frequent, and occasionally the ulcerative variety is seen. Thrush often occurs in the protracted cases among very young infants. Gastro-enteritis is not very common, considering the frequency of vomiting and diarrhœa, these depending usually upon functional derangement. In only three of my cases was there nephritis. In all it was of the acute exudative variety, and in only one case was it severe enough to affect the prognosis.

Old lesions of tuberculosis—cheesy nodules in the lungs and sometimes in the pleura—are not infrequently met with in patients dying of acute pneumonia of a non-tuberculous character.

Diagnosis.—An acute onset with continuous high fever, rapid respiration, and cough, should always lead one to suspect pneumonia. When to these symptoms are added prostration and cyanosis, the diagnosis of pneumonia is almost certain. Cases of the acute congestive type are the ones most frequently unrecognised, and in many of these cases a positive diagnosis is impossible during life. Many atypical cases of pneumonia are seen, particularly in young infants. An unusual temperature course is perhaps the symptom most likely to lead to a mistake. While this, as a rule, is high and remittent, it is sometimes not so, and may be but little above normal. Rapid respiration is almost always present, but cough may be very slight, especially in infants. In very young infants, the diagnosis often rests upon the prostration, cyanosis, and rapid respiration, the other acute inflammatory symptoms being absent. Only the physical signs of the disease can positively settle the question of diagnosis.

When pneumonia follows bronchitis of the large tubes, whether the bronchitis is primary or complicates one of the infectious diseases, the extension of the disease to the lungs is usually marked by three symptoms —a steadily rising temperature, more frequent respiration, and increasing prostration. It may be twelve or twenty-four hours before the change is indicated by the physical signs.

The diagnosis of broncho-pneumonia from congenital atelectasis has to be considered only during the first three or four months, it being rare for atelectasis to give symptoms after this time. In early infancy the danger of confusing the two is increased by the fact that atelectasis and broncho-pneumonia may be associated. If the infant has been strong and well for the first two months, congenital atelectasis can be excluded. It is likely to be found in delicate infants, where there is a history of difficulty in resuscitation at birth and feeble cry during the early days of life. The temperature is low, often subnormal, the cyanosis is out of proportion to the other symptoms, and the physical signs are doubtful or absent.

At the outset, pneumonia can not be positively diagnosticated from severe bronchitis. Such a bronchitis often begins with severe pulmonary

symptoms and a temperature of 103° or 104° F.; but this high temperature is of short duration, usually falling after twenty-four or forty-eight hours to 100° or 101° F. The prostration is much less, and all the symptoms, possibly excepting the cough, less severe. The only physical signs are coarse râles, which are heard throughout the chest.

The same rules apply to bronchitis of the smaller tubes. The râles are heard both in front and behind, and usually over both sides. If with such râles the temperature continues to rise for three days in succession above 103° F., it may be assumed that pneumonia is present, provided there is no other disease which might explain the temperature. If, instead of being generalized, the signs of bronchitis are limited to a single lung, or to one lung posteriorly, the existence of broncho-pneumonia may be regarded as certain. Localized bronchitis, then, is always to be interpreted as broncho-pneumonia, provided tuberculosis can be excluded. In doubtful cases the chances largely favour broncho-pneumonia rather than bronchitis. Attention is again called to the fact already mentioned, that there are a large number of cases of pneumonia without signs of consolidation.

The differential diagnosis of broncho- from lobar pneumonia will be considered in connection with the latter disease. On account of the remittent temperature, broncho-pneumonia may be confounded with malarial fever; if with the latter there is some bronchitis, or if accompanying the onset of a severe malarial paroxysm there is pulmonary congestion—two not infrequent combinations—the difficulties are increased. A positive diagnosis is often impossible except by careful observations of the temperature for one or two days. The points of differentiation are, that the temperature of pneumonia, though often remittent, is very rarely intermittent, and that it is not affected by quinine. In addition, the characteristic features of malaria—enlargement of the spleen, the plasmodium in the blood, and a history of exposure—must, of course, be taken into account.

Both the acute and the persistent forms of simple broncho-pneumonia may be confounded with the tuberculous form; the points of distinction are considered in the chapter on Tuberculosis.

Prognosis.—Broncho-pneumonia is always a serious disease, and in an infant dangerous to life. The prognosis depends upon the age, surroundings, and previous condition of the patient, upon the nature of the infection, whether the disease is primary or secondary, and, if the latter, upon the character of the primary disease. In private practice the mortality from broncho-pneumonia is from 10 to 30 per cent, depending upon the conditions mentioned. One whose knowledge of broncho-pneumonia is derived from observations in private practice can, however, form but little idea of the frequency and severity of this disease in hospitals and asylums for infants and young children, particularly when it occurs with epidemics of measles, diphtheria, and pertussis. The statistics in the fol-

lowing table are taken from the records of two institutions with which I am connected, and fairly represent the results seen in such places in children under three years:

Forms of Pneumonia.	Cases.	Deaths.	Percentage mortality.
Primary broncho-pneumonia	194	96	49·4
Following bronchitis of the large tubes	29	19	65·5
Secondary to measles	89	56	62·9
" " pertussis	66	54	81·8
" " scarlet fever	7	7	100·0
" " diphtheria	47	47	100·0
" " ileo-colitis	19	18	94·7
" " epidemic influenza	6	1	16·6
" " varicella	2	2	100·0
" " erysipelas	2	2	100·0
Totals	461	302	65·5

The mortality varies directly with the age of the patient, being the highest during the first year, and diminishing steadily thereafter, as shown by the following table giving the result in three hundred and forty-five cases:

Age.	Cases.	Percentage mortality.
During the first year	202	66
" " second year	102	55
" " third year	33	33
" " fourth year	6	16
" " fifth year	3	..

In this table are included no cases secondary to measles, scarlet fever, or diphtheria.

Probably the best of all guides to the nature and virulence of the infection is the temperature. An excessively high temperature indicates a virulent type of infection. Some idea of this may be gained from these figures, giving the highest temperature and the mortality in two hundred and thirty-one cases, not including cases with measles or diphtheria:

Highest Temperature.	Cases.	Deaths.	Percentage mortality.
106 F. or over	55	47	85·5
105 or 105·5	94	56	60·0
104 or 104·5	53	26	49·0
102 to 103·5	22	13	60·0
99·5 to 101·5	7	5	71·0

The high mortality of the cases with unusually low temperature is due to the fact that they nearly always were seen in infants with very feeble

vitality. Cases with a steadily high temperature—between 102·5° and 104° F.—usually do better than those with wide fluctuations, such as 100° to 105·5° F. The probable explanation of this is, that the former are due to the pneumococcus, while the latter are apt to be cases of mixed infection, or due to the streptococcus. As a rule, the danger of the disease increases steadily with every degree of temperature above 104·5° F.

An important factor in the prognosis is the previous condition of the patient. One of the most unfavourable is rickets, both on account of the feeble muscular power of these children and their thoracic deformities. Any condition which diminishes the general vitality increases the danger from broncho-pneumonia. As a rule, second attacks are more serious than the primary ones, especially if the interval between them is short.

In making the prognosis in any given case, the symptoms to be considered are the height and course of the temperature, the presence or absence of nervous symptoms, the condition of the organs of digestion, the presence of cyanosis and the extent of the disease as shown by the physical signs.

Nervous symptoms early in the disease do not affect the prognosis. Three cases in which convulsions occurred at the onset all recovered, but of thirty-seven cases in which convulsions occurred at a late period during the course of the disease, all but one proved fatal.

So long as the food is well taken and retained and the stools show that it is being assimilated, no case is hopeless, no matter how severe the other symptoms may be; but the existence of vomiting, diarrhœa, or severe indigestion makes the issue doubtful, even though the other symptoms are very favourable. These conditions are especially important in protracted cases, where death is usually due to slow asthenia.

Treatment.—The most important part of prophylaxis is to give careful and early attention to every attack of bronchitis in an infant, for every such attack should be regarded as a possible precursor of pneumonia. It is striking that one sees broncho-pneumonia so seldom in private practice among the better classes, even though bronchitis is very frequent; while among hospital and dispensary patients, where bronchitis is very often neglected, broncho-pneumonia is constantly seen. The question of isolating cases of pneumonia is one which is lately becoming more and more important. While it may not often be the case that primary pneumonia is due to contagion, there seems to be little doubt that this is at times true of the pneumonia secondary to measles and diphtheria. Twice in one institution have I seen regular epidemics of broncho-pneumonia occur with outbreaks of measles—in some of the wards nearly every case of measles developing pneumonia. In another institution, during one entire season (1888–'89), almost every case of diphtheria transferred to a certain isolation pavilion developed pneumonia, and died from that complication. Cases of measles and diphtheria which are complicated by pneumonia

should, if possible, be carefully isolated from others, and wards in which they are treated should be thoroughly disinfected before they are used for simple cases.

The hygienic treatment of pneumonia is important, and usually it receives too little attention. The child should be kept in a large, well-ventilated room, preferably one with an open fire; if possible, being changed from one room to another two or three times a day, to allow thorough airing. Nothing is more important for an infant sick with acute pulmonary disease than plenty of oxygen. Older children should be kept in bed. Infants for a considerable part of the time may be held in the nurse's arms. A frequent change of position in all cases is essential; no child should be allowed to lie for hours directly on the back. The general rules for feeding all sick children (page 190) should be followed here. As a rule, neither stimulants nor medicine should be administered in the food.

The same local treatment may be employed as in cases of bronchitis (page 467). The oiled-silk jacket should be worn throughout the attack, and counter-irritation maintained by the use of the mustard paste. Hot poultices of flaxseed may be employed occasionally, but never continuously.

Emetics.—What was said of expectorant mixtures and emetics in the treatment of bronchitis applies here with even greater force. In infants both had better be omitted altogether.

Stimulants.—Alcoholic stimulants are needed in all secondary cases, and in a large proportion of those which are primary. No doubt they have been greatly abused, and, when pushed in the early stage, often do much harm; but in most of the severe cases they are indispensable. They are usually needed from the outset, where the pneumonia is secondary to measles, diphtheria, scarlet fever, or other infectious diseases. They are called for when the pulse is weak, compressible, rapid, and irregular. Whisky or brandy is usually to be preferred, although the taste of the patient often has to be consulted, and when these are refused, some wines, like sherry or tokay, may be readily taken. (For methods of administration see page 49.) The dose is to be regulated by the condition of the patient. From one half to two ounces daily may be given to an infant of one year. It is rarely advisable to go above this limit except for a few hours at a time at critical periods; then two or three times as much may be used. Contrary to the statement of many writers, these stimulants are usually well borne, even by young children. Stimulants are most needed when the temperature is low, or falls suddenly, as at the crisis of the disease. When the temperature is high, smaller amounts are generally required.

In many cases strychnine is even more valuable than alcohol. Usually they should be combined, as the indications are the same. Where the dose is to be repeated every three hours, $\frac{1}{300}$ of a grain is as much as it is

wise to give to an infant a year old. This may be kept up for days, and for a shorter time larger doses may be given, the effect always being carefully watched. For older children digitalis may be used, but I have rarely seen much benefit from it in infants. In attacks of heart failure associated with pulmonary congestion, nitroglycerin should be given—gr. $\frac{1}{500}$ every hour for four or five doses, or even longer.

Respiratory stimulants are needed in most cases, even more than are cardiac stimulants, but we have none which can be wholly depended upon. For a short time, atropine gr. $\frac{1}{800}$, caffein gr. $\frac{1}{20}$, or strychnine gr. $\frac{1}{300}$, may sustain a child with sudden failure of respiration, but in the slow respiratory failure that results from exhaustion their effect is but temporary. The doses mentioned are for an infant of one year. The drugs may be used successively or together; for immediate effect they should be given hypodermically. Oxygen may be classed with the respiratory stimulants. It should be given continuously, but always freely mixed with atmospheric air. A good method is to place the child in a half-open tent, beneath which the gas is introduced. Gentle friction of the chest wall, without disturbing the patient, is sometimes useful in stimulating the respiratory muscles, especially in protracted cases.

Antipyretics.—It must be remembered that the normal range of temperature in broncho-pneumonia is from 101° to 104·5° F. This temperature is not in itself exhausting, and the chances of recovery are not, I think, improved by systematic efforts at reducing it so long as it remains within these limits. Too much can not be said in condemnation of the practice of giving such drugs as phenacetine, antipyrine, and antifebrine in full doses for the reduction of temperature. In small doses they are often useful to allay nervous irritability, restlessness, and promote sleep. Quinine can not be considered an antipyretic in pneumonia except in cases complicated by malaria. Otherwise it does little if any good, and often great harm, by disturbing the stomach.

Antipyretic measures are indicated in cases of hyperpyrexia, which we may define as 105° F. or over, or when extreme nervous symptoms exist, even though the thermometer may not register the degree mentioned. Under these circumstances, the most certain, the most within our control, and hence the safest antipyretic, is cold. It may be used by the graduated bath, the cold pack (pages 47, 48), sponging, or an ice-bag applied to the chest.

The most convenient and efficient methods of using cold are the bath and the cold pack—the bath for infants, and the pack for older children. The peripheral circulation should be closely watched, and maintained by friction of the body during the bath, and the application of heat to the extremities immediately after it. In most cases the bath should be preceded by stimulants. The effects are often very striking; when there have been a flushed face, hot dry skin, extreme restlessness, and muscular twitchings,

all these symptoms may subside rapidly and a quiet sleep follow. The bath should be repeated as soon as these symptoms return, whether the thermometer has risen to its former height or not. When with hyperpyrexia we have general cyanosis, cold surface, feeble pulse, shallow respiration, and stupor, cold is contraindicated and a hot mustard bath should be used.

Inhalations.—These are of more value in relieving cough and in promoting bronchial secretion than any other means we possess. At the same time, they seem often to have a beneficial influence upon the local process. They are useful in proportion to the amount of bronchitis which is present. The same substances are to be used, and in the same way as mentioned in the article on Bronchitis.

The *nervous symptoms*, restlessness, loss of sleep, etc., are often best controlled by cold or tepid sponging; in other cases by small doses of phenacetine—i. e., one grain every two hours to a child of six months. Opium is to be avoided unless there is severe pain, which is very rare; or, when the incessant cough is not relieved by inhalations. Dover's powder is the preparation to be preferred, and an occasional dose of a quarter of a grain usually all that is necessary.

Sudden *attacks of general collapse* with cyanosis are frequent in severe cases of broncho-pneumonia. They may come on at any period in the disease. When occurring in the early stage, if promptly and energetically treated, recovery may take place, but when they come on in the late stages they are usually fatal. They may be due to acute congestion or œdema of the lung not previously involved. The most efficient treatment is to put the child into a hot mustard bath (page 54), to use strychnine and nitroglycerin hypodermically, and to give oxygen continuously. For a few hours alcohol should be given *ad libitum*. Nitrite of amyl is sometimes more efficient than nitroglycerin, because of its almost instantaneous effect. I must confess to have seen very little benefit from the use of camphor, although many excellent observers esteem it very highly.

Treatment of protracted cases.—Where the fever continues for five or six weeks, with no disposition on the part of the disease to subside, about all that can be done is to continue the sustaining treatment adopted in the earlier part of the disease—careful feeding, judicious stimulation, and proper hygienic means. Many of these cases will recover if the patient's strength holds out; but, unfortunately, in the majority the continuance of the pneumonic process is in itself evidence of the weakened vitality of the patient, and, though he may live a long time, the attack proves fatal in the end.

Where the fever has disappeared, and there is only a persistence of the physical signs and the general cachexia, the cases are more hopeful. Here, a change of air is more important than all other means of treatment. If in the winter or spring the child can be removed to a warm, dry cli-

mate where it can be kept in the open air, or if, in the summer, it can be taken to the mountains, immediate improvement is often seen, followed by rapid recovery. This experience we see repeated every year with hospital patients when they are transferred from the city to the country in May or June. With the change of air a general tonic plan of treatment should be followed, cod-liver oil, arsenic, iron, and quinine being used, according to the indications in each particular case.

In specific drugs to promote resolution I have no faith. Where the cough continues, creosote may be used both internally and by inhalation, as after bronchitis. One should never declare one of these cases of protracted pneumonia to be hopeless, nor should he be too ready to assume that tuberculosis is present because the child is wasted and anæmic, and the physical signs have persisted. In private practice the cases of simple protracted pneumonia outnumber the tuberculous ones, three to one.

Summary.—In the treatment of broncho-pneumonia it should be borne in mind that, while very little can be done for the disease, very much can be done for the patient. The hygienic measures generally grouped under the term "careful nursing" are of great importance, and many of the mild cases need no other treatment. In severe cases, the patient may be in great danger in the early stage from two causes: first, from the intensity of the general infection, which is best combatted by the use of alcohol and strychnia; and, secondly, from the mechanical embarrassment of the heart and respiration, in consequence of the sudden interference with the function of the lungs, partly from inflammation, but chiefly from congestion; this is best relieved by counter-irritation to the chest and heat to the extremities. During the later stage the principal danger is from exhaustion; this forbids the use of all depressing measures, and necessitates the most careful attention to the nutrition of the patient throughout the disease. All unnecessary medication is to be avoided, particularly the use of expectorant mixtures, on account of the disturbance of the stomach. Opium is to be used very sparingly, and in most cases it should be withheld altogether. The cough is best relieved by inhalations of creosote, and the nervous symptoms by phenacetine or baths. For local use, the oiled-silk jacket is better than poultices. Counter-irritation by mustard should be continued throughout the attack, when there is much bronchitis. Where antipyretics are required, cold is safer and more efficient than the use of drugs. Of the cardiac stimulants, alcohol and strychnia are most to be depended upon. Care should be taken in all cases to maintain a good peripheral circulation. In sudden general collapse, the most valuable measures are hot mustard baths, strychnia hypodermically, alcohol freely by the mouth, and the inhalation of oxygen. In protracted cases, and in those with delayed resolution, change of air is more important than all other means combined.

CHAPTER V.

DISEASES OF THE LUNGS.—(Continued.)

LOBAR PNEUMONIA.

Synonyms: Fibrinous pneumonia, croupous pneumonia, pneumonic fever.

With our present knowledge, this may be best defined as an infectious disease, caused by the micrococcus lanceolatus (pneumococcus) and accompanied by a local lesion in the lungs. While in most cases the general symptoms correspond with the extent and severity of the local lesion, they may be out of all proportion to each other.

Etiology.—*Age.*—Lobar pneumonia may occur at any age. I have recently seen a case in an infant of three months which followed the typical course. It may be seen even in the newly born, but it is not until after the second year that it begins to be frequent. After the third year nearly all the cases of primary pneumonia are of this variety.*

Of 160 personal cases, and 340 collected from various sources, the ages were as follows:

Age.	Cases.	Per cent.
During the first year	76	15
From the second to the sixth year	309	62
" " seventh to the eleventh year	104	21
" " twelfth to the fourteenth year	11	2
Totals	500	100

The greatest susceptibility appears to be from the second to the sixth year, and during this period it is most frequent from the third to the fifth year.

Sex.—Of my own cases, 60 per cent were males, and the same proportion was noted in 544 collected cases. This predominance of males has been everywhere observed, but is as yet unexplained.

Season.—In my series of cases, the seasons were divided as follows:

	Cases.	Per cent.
In the three winter months	48	35
" " spring "	62	46
" " summer "	6	4
" " autumn "	20	15
Totals	136	100

* For the relative frequency of broncho- and lobar pneumonia during infancy, see the table on p. 479.

Lobar pneumonia, in children therefore, as in adults, occurs most frequently during the spring months. April showed the largest number of any single month.

Previous condition.—In my hospital cases, 82 per cent of the children were previously in good condition, and only 18 per cent were delicate, rachitic, or syphilitic. This observation has been borne out by my experience in private practice—viz., that as a rule lobar pneumonia affects children who were previously healthy.

Previous disease.—Previous attacks of pneumonia are observed in but a small proportion of cases. It was noted only five times in 160 cases. In the vast majority of cases lobar pneumonia is a primary disease, although it occasionally occurs as a complication of pertussis, measles, typhoid or scarlet fever, and even diphtheria—chiefly, however, in children over three years old.

Epidemics of lobar pneumonia I have never witnessed, although on several occasions I have seen two children in a family attacked either simultaneously or in rapid succession. Exhaustion, fatigue, and exposure are to be ranked as associated exciting causes.

In addition to other causes, there is required for the production of the disease the presence and growth of the pneumococcus.

Lesions.—*The seat of the disease.*—In 950 cases in children under fourteen years, this was as follows:

Seat of Disease.	Personal cases.	Collected cases.	Totals.
Right lung, upper lobe only	39	137	176
" " middle " "	8	4	12
" " lower " "	26	142	168
" " more than one lobe	13	64	77
Totals, right lung	86	347	433
Left lung, upper lobe only	25	68	93
" " lower " "	49	214	263
" " more than one lobe	9	29	38
Totals, left lung	83	311	394
Both lungs, upper lobes	..	13	13
" " lower "	3	38	41
" " elsewhere	9	60	69
Totals, both lungs	12	111	123

The right lung was thus affected in 45·5 per cent; the left lung in 41·5 per cent; both lungs in 13 per cent. In the order of frequency, the disease involves, first, the left base; second, the right apex; third, the right base; fourth, the left apex. The disease affects, as a rule, a single lobe, and often only a circumscribed portion of a lobe, stopping sharply at the interlobar fissure.

Lobar pneumonia among children is so rarely fatal that the opportunities for a study of the peculiarities of the lesion have been somewhat limited. I have myself made eleven autopsies, and have among my hospital records reports of nine others, making twenty cases in all. The anatomical changes resemble those seen in the adult lung. There is an exudation into the alveoli and smaller bronchi of fibrin, serum, leucocytes, and red blood-cells (Fig. 73). There is usually in addition an inflammation of the mucous membrane of the larger bronchi and of the pleura. The frequency and severity of the pleurisy is a peculiarity of the lesion in children.

In the first stage, that of *congestion*, the portion of lung involved is dark-coloured, heavy, and œdematous, and shows under the microscope a serous and cellular exudation into the air vesicles, with swelling of the epithelial cells lining the alveoli.

In the second stage, that of *red hepatization*, there is usually some exudation upon the pulmonary pleura, generally a thin layer of fibrin, giving it a dull, granular look. The lung itself is of a uniform dark-red colour. It is solid, and cuts like liver. It looks as if it had been inflated to its utmost extent and then injected with a material which had solidified. The consolidated area is sharply defined. Under the microscope the air vesicles are seen to be distended with an exudation which is chiefly fibrin, but with some leucocytes, red blood-cells, and desquamated epithelial cells. The cells are chiefly leucocytes, and are usually more abundant than in the pneumonia of adults.

In the third stage, or *gray hepatization*, the lung is more moist, and the inflammatory products are partly decolourized. This change takes place irregularly throughout the lung, giving it a mottled appearance.

The fourth stage, that of *resolution*, follows gray hepatization, and consists in the degeneration and liquefaction of the products of inflammation, which are ultimately carried away by the lymphatics, or pushed out into the bronchi and removed by coughing.

The duration of the stage of congestion is from a few hours to several days; that of the stage of red hepatization from two days to two or three weeks. This is the condition in which the lung is most often seen at autopsy. The stage of gray hepatization is commonly shorter. Resolution usually begins when the temperature falls to normal, but occasionally it may be delayed for several days. It is generally complete in about a week.

Variations in the lesions.—(1.) Instead of clearing up at the usual time, the lung may remain consolidated for several weeks, and then resolve. (2.) The stage of gray hepatization may be followed by a great exudation of pus cells, which may everywhere infiltrate the affected lung; or these may be circumscribed so as to form a single large abscess or many small ones. (3.) There may be small areas of gangrene. All these conditions

are very rare in children. Purulent infiltration and delayed resolution were not noted in any of my cases, and gangrene but once. (4.) There may be excessive pleurisy, or pleuro-pneumonia. This was found in one half of my autopsies. These cases will be separately considered elsewhere.

Lesions in other organs.—With pneumonia of the left side, if complicated by pleurisy, there may also be pericarditis. This was seen in two of my cases. The pericardial inflammation closely resembled that of the pleura. There was a very abundant exudation of fibrin and pus, coating both surfaces of the pericardium. Acute meningitis has been rarely observed. It was met with twice in my cases. The form of inflammation was an acute purulent meningitis, with a very abundant exudation of greenish-yellow lymph, chiefly at the convexity. In one of my cases peritonitis was also seen as a complication of pleuro-pneumonia. As the pneumococcus is found in all these inflammations, they may be regarded as examples of a more generalized infection than usually occurs. In most of these the other processes are secondary to that in the lungs, but sometimes they begin simultaneously with, or may even precede, the pulmonary lesion.

The heart is generally found in diastole, with the cavities, especially those of the right side, distended with soft clots. There may be found ante-mortem thrombi, which may extend into the pulmonary artery or the aorta.

Symptoms.—(1.) *The typical course.*—A child three or four years of age, after a few hours of slight indisposition, is suddenly taken with vomiting, followed by a rapid rise in temperature. He is dull and heavy, complains of headache and general weakness, refuses food, and is easily persuaded to remain in bed. He has the appearance of being quite ill, even after a few hours. Occasionally sharp pain in the side is complained of. The skin is dry; there are marked thirst, restlessness, and the other symptoms which accompany fever. The temperature is found to be 104° F., or even higher; the respirations 40 to 50 a minute; the pulse full, strong, and 120 to 130. On the second day the patient is no better. The temperature remains high; the tongue is coated; the anorexia continues; the pain is more severe; cough is present and may be quite frequent.

After the second or third day the patient is usually more comfortable, and sleeps better, but may be disturbed by the cough. At times there is restlessness, and at night there may even be slight delirium. The respiration continues rapid and the temperature high. These general symptoms show very little change until the sixth or seventh day, when, after a long sleep, which has been more natural than before, the patient wakes, decidedly improved as to all his symptoms. There is less fever, and the temperature continues to fall rapidly until it touches the normal line, or it may even go below this. As the fever subsides the pulse drops to 90 or 100, and the respirations to 25 or 30 a minute. The appetite soon returns,

and convalescence is usually rapid. In a week the patient is out of bed, and in a month from the beginning of the illness he is out of doors; but it may be another month before he can be considered to have entirely recovered. This is the course seen in fully two thirds of all the cases of lobar pneumonia at this age.

(2.) *Pneumonia of short duration.*—Instead of running the usual course of from five to eight days, cases are seen in which the duration is only three or four days, although the physical signs indicate that the process in the lung passes through the usual stages. These are the cases of short pneumonia, and they differ from the ordinary type chiefly in their duration. They are always mild.

(3.) *Abortive pneumonia.*—This form of the disease is rarely seen in hospitals, but it is not infrequent in private practice where the physician is summoned at the earliest signs of illness. The onset is precisely like that of ordinary pneumonia, and may even be as severe as the average case. The physical examination of the chest gives all the signs of the first stage of the disease, but on the second or third day the physician is greatly surprised to find that the temperature has fallen to normal, and that all the physical signs have disappeared. The process in such cases does not seem to go beyond the first stage of congestion; there is no evidence of hepatization of the lung. The course is often such as to lead the physician to the opinion that he has made a mistake in his diagnosis. There seems, however, to be no doubt that these are cases of genuine pneumonia. D'Espine found the pneumococcus in the sputum of such a case. This type of pneumonia corresponds with abortive types of other infectious diseases so frequently met with in children. The temperature curve in such a case is shown in Fig. 95, page 521. The diagnosis of these cases is always attended with some uncertainty. There can be no doubt that very many of the unexplained high temperatures of brief duration which are seen in children are from this cause. Exactly why the disease terminates in this way is not known. It may be because the resistance of the patient is greater than usual, or the virulence of the pneumococcus is less.

(4.) *The prolonged course.*—Although usually lasting about a week, it is not rare for pneumonia to continue ten, twelve, or even fifteen days. This prolonged course is often due to the fact that the disease spreads from one part of the lung to another, involving in succession two and sometimes three lobes; but it may occur when the process is limited to a single lobe. A prolonged temperature should always suggest the possibility of complications, usually pleurisy. Prolonged cases are generally severe.

(5.) *Cerebral pneumonia.*—This term was first applied by Rilliet and Barthez to cases of pneumonia in which the cerebral symptoms predominated. They will be considered under special symptoms.

Onset.—Prodromal symptoms of more than a few hours' duration are quite rare. The onset of lobar pneumonia is almost invariably sudden, with well-marked symptoms—vomiting, diarrhœa, chill, or convulsions. Vomiting is altogether the most frequently seen. It was the mode of onset in about one half my cases. In summer particularly, there may be vomiting and diarrhœa. A distinct chill is rare in a child under five years of age, and is not very common even in older children. Convulsions are not very infrequent, being seen in about five per cent of the cases. Their occurrence depends upon the suddenness of the invasion and the susceptibility of the patient.

Cough.—This is present in most of the cases throughout the disease, but often is not marked for the first day or two. It is seldom a distressing symptom. A disposition to suppress the cough on account of pain is very frequently noticed.

Expectoration.—This is rarely seen in childhood, and practically never under five years of age. Children of ten or twelve may have the same expectoration as adults—white and viscid, or brownish-red early in the disease, yellow and abundant toward its close.

Pain.—Headache and general muscular pains in the back and extremities are frequent during the invasion. The characteristic pain, however, is pleuritic. It is not necessarily felt in the region of the affected lung, and often not in the chest at all. It is frequently referred to the loin, the epigastrium, or to any region to which the intercostal nerves are distributed. In a recent case, in a boy of seven years, for the first twelve hours there was intense localized pain in the right iliac fossa, associated with such extreme tenderness as to lead to the suspicion that the case was one of appendicitis. The pain may last throughout the disease, and occasionally it is a most distressing symptom; but usually it is only moderate, and rather more severe early than late in the disease.

Prostration.—This is one of the characteristic features of pneumonia. The patient is generally willing to go to bed on the first day of the attack, and shows little desire to leave it while the disease continues. "Walking cases" are not common in children.

Respiration.—This is always accelerated, and generally out of proportion to the pulse. The normal ratio of the respiration to the pulse is one to four; in pneumonia, frequently one to two. The respiration is not laboured and not quite panting, although this term is sometimes used to describe it. It is jerky. There is a short inspiration, then a momentary pause, followed by a quick expiration, which is accompanied by a short moan. This expiratory moan is very characteristic. The rapidity of respiration is usually in proportion to the amount of lung involved, but it is also modified by the temperature, as the respirations often drop from 60 to 30 in the course of a few hours at the crisis.

Pulse.—In the early part of the disease this is frequent, full, and

strong, from 110 to 140 a minute. Later it may be weak, small, compressible, and sometimes irregular. It is relatively more rapid in the child than in the adult. The frequency of the pulse is of less importance than its character.

Temperature.—The typical temperature curve of lobar pneumonia (Fig. 92) is characterized by an abrupt rise usually to 104° or 105° F., and by daily fluctuations generally within the limits of two or three degrees

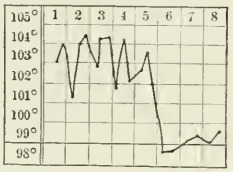


Fig. 92.—Typical temperature curve of lobar pneumonia.

History.—Male, three years old; in fair condition; sudden onset; signs of consolidation—bronchial respiration and voice, and dulness—over left lower lobe behind, not distinct until the morning of the fifth day. On the seventh day the lung was resolving.

until the crisis, at which time the temperature falls to normal, usually in the course of twenty-four hours. After this time it does not go above the normal line. Such a curve is seen in the majority of cases over three years of age.

In cases under three years of age it is not uncommon for the temperature to be of a more or less remittent type (Fig. 93).

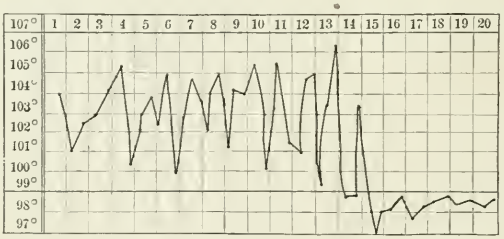


Fig. 93.—Lobar pneumonia with remittent temperature.

History.—Female, eighteen months old; in fair condition; sudden onset; repeated examinations of chest made, but no abnormal signs until the ninth day, when there were very rude respiration and slight dulness at the right apex, in front; on the twelfth day all the signs of consolidation at the same point, no rales; four days after the crisis the lungs were clear.

These wide fluctuations often lead to great difficulty in diagnosis, particularly if the physical signs appear late, as they not infrequently do. It is possible that some of them are to be explained by mixed infection.

The following chart (Fig. 94) illustrates three features which are often seen in pneumonia: (1) A temperature which early in the disease is steadily high and as the day of crisis approaches becomes remittent; (2) a secondary rise after being normal for twenty-four hours, which was due

in this instance to an extension of the disease to a new part of the lung; (3) a fall to a point considerably below normal at the time of the crisis. In this case the temperature fell in the course of eighteen hours from

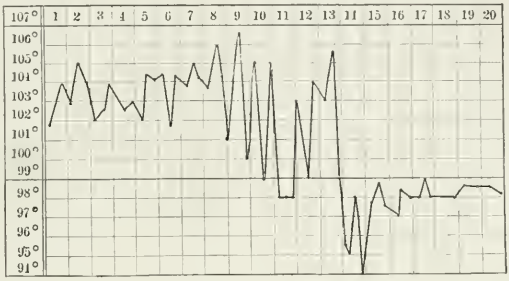


Fig. 94.—Lobar pneumonia with subnormal temperature after the crisis.

History.—Female, nineteen months old; fairly healthy; sudden onset; symptoms typical but physical signs delayed; consolidation in left mammary region on the eighth day; on the ninth in right lung middle lobe; on the eleventh day a pseudo-critical drop, followed after twenty-four hours of apyrexia by a further rise, which was accompanied by signs of extension of the disease in the right lung. Resolution rapid after crisis.

105° to 95° F., and later still lower; it was two days before it finally remained at the normal point. A fall to 96·5° or 97° F. at the time of crisis is not uncommon.

In the foregoing cases the fever terminated by crisis. In Fig. 95 is shown one ending by lysis. This is a mode of termination much more frequent in young children than in those who are older. Thus, in ninety-

Fig. 95.—Abortive pneumonia in left lung, followed by typical pneumonia in right lung, terminating by lysis.

History.—Male, seventeen months old; healthy; sudden onset; on the second day disseminated fine râles in both lungs behind, and over left lower very feeble respiration, high-pitched—i. e., some bronchitis, with congestion (?) of left base. On the third, fourth, and fifth days, general symptoms gone and signs nearly disappeared. On the sixth day all symptoms of pneumonia, and on the seventh distinct consolidation of right base, rest of chest clear. Subsequent course typical; resolution rapid and complete.

three of my own cases, nearly all of which were under three years of age, the fever ended by crisis in forty-nine, and by lysis in forty-four; while in five hundred and twenty-two collected cases, the majority of which were in older children, three hundred and ninety-six ended by crisis, and one hundred and twenty-six by lysis.

The following table shows the day of crisis in five hundred and sixty-seven cases of lobar pneumonia in children who recovered :

The Day of Crisis.

Second day	3 cases.	Eleventh day	18 cases.
Third "	22 "	Twelfth "	7 "
Fourth "	43 "	Thirteenth day	8 "
Fifth "	88 "	Fourteenth "	7 "
Sixth "	83 "	Fifteenth "	1 case.
Seventh "	132 "	Eighteenth "	3 cases.
Eighth "	73 "	Twenty-first day	1 case.
Ninth "	55 "	Twenty-sixth "	1 "
Tenth "	22 "		567

From this table it will be seen that the most frequent critical day is the seventh, and that in 66 per cent of the cases it was from the fifth to the eighth day. The causes of a post-critical rise in the temperature are chiefly two—extension of the disease to a new area, or the development of pleurisy, which is apt to be purulent. Less frequently it is due to meningitis, pericarditis, gastro-enteritis, or malaria. In fatal cases the temperature is generally high until the end. In general, it may be said that the temperature is considerably higher in children than in adults; in the majority of cases it reaches 105° F., the usual range being from 102° to 105° F. In fifteen of one hundred and thirty-seven cases, or 11 per cent, it reached 106° F. or over.

Gastro-enteric symptoms.—These are more common in infants than in older children. At the onset there is frequently vomiting, sometimes also diarrhœa. A continuance of the vomiting is rare, and is generally due to improper feeding or medication. It may be a very serious complication. Diarrhœa is also rare, except at the onset and in summer cases. It is sometimes seen at the time of crisis. Throughout the disease there are anorexia, coated tongue, and the usual symptoms of high fever.

Nervous symptoms.—Cerebral symptoms are frequent and very often misleading. In seven of my cases the pneumonia was ushered in by convulsions. These differ in no respect from convulsions from other causes, and may be repeated two or three times in the course of the first twenty-four hours. They are sometimes followed by drowsiness or stupor, sometimes by active delirium. Cerebral symptoms may predominate for several days. There may be opisthotonus, dilated or contracted pupils, irregular pulse, retracted abdomen, and, in fact, almost every symptom of meningitis. Occasionally the decubitus *en chien de fusil*, or gun-hammer position, is assumed. These are often described as cases of *cerebral pneumonia*, and in many of them pneumonia is not suspected until the fourth or fifth day of the disease, sometimes not until the crisis occurs, when the rapid disappearance of all these nervous symptoms indicates their origin. Early

convulsions are not generally followed by an especially severe type of the disease, only one of seven cases beginning in this way proving fatal. On the other hand, late convulsions are usually fatal. In two of the three cases in which I have noted them, the convulsions ushered in an attack of meningitis.

Delirium is much more frequent than convulsions, and is seen in nearly one fourth of the cases. Generally it is slight, and noticed only at night or when the temperature is very high. It is usually mild, but may be low and muttering, like that of typhoid, or wild and active, like that of cerebro-spinal meningitis. It is most pronounced at the height of the disease. Other nervous symptoms belonging to the typhoid state, such as incontinence of urine or fæces, muscular twitchings, and tremor of the tongue on protrusion, are occasionally seen, but only in the worst forms of the disease.

There is no relation between the seat of the disease in the lungs and the occurrence of cerebral symptoms. They are more frequent in children under five years than in those who are older, and depend upon the suddenness of the invasion, the intensity of the infection, and the susceptibility of the child. Late in the disease they may indicate exhaustion, toxæmia, or complicating meningitis. They are frequently associated with very high temperature and extensive disease.* The usual nervous symptoms—restlessness, headache, sleeplessness, etc.—are nearly always proportionate to the height of the temperature.

Urine.—Throughout the febrile period of the disease the urine is scanty, high-coloured, with a high specific gravity, and usually loaded with urates. In a small number of cases a trace of albumin may be found, and occasionally a few hyaline casts. Evidences of serious renal disease I have seldom found in lobar pneumonia, and in the experience of all observers it is extremely rare in early life.

Skin.—The face, in pneumonia, is usually flushed, sometimes on both sides and sometimes only on one; in other cases it is pale, but not indicative of pain. Cyanosis is rare except toward the close of the disease and is usually a sign of respiratory failure. Herpes of the lips or face is quite frequent.

Physical Signs.—The earliest signs in pneumonia are due to the acute congestion of the affected lung or lobe, in consequence of which less air enters this portion and more air the rest of the lungs. Percussion gives diminished resonance or slight dulness over the affected area, and exaggerated resonance over the remainder of this lung and over the opposite lung. Auscultation over the affected lobe gives feeble respiratory murmur, rather high in pitch; sometimes there may be absence of all breath-sounds

* For a fuller discussion of the cerebral symptoms of pneumonia, see a paper by the author, in the New York Medical Record, April 7, 1888.

so complete as to suggest fluid. The normal respiratory murmur over the healthy portions of the lungs is intensified. In children this exaggerated breathing is not infrequently mistaken for bronchial breathing, and the physician may be led into the error of locating the pneumonia upon the wrong side. Exaggerated breathing does not differ from normal breathing except in intensity, and is heard only on inspiration. Bronchial breathing is higher in pitch, and is heard with nearly equal intensity both on expiration and inspiration. If the chest is frequently auscultated, crepitant râles (Figs. 96 and 97) may usually be heard at some period at the end of full inspiration, but often they are present but for a few hours, and they may be missed altogether.

In the second stage, that of consolidation (Fig. 98), no air enters the affected part of the lung. Upon palpation there is found here exaggerated vocal fremitus, and on percussion there is marked dulness, but very rarely flatness. Over the rest of this lung there is exaggerated, sometimes even tympanitic, resonance; this is especially frequent at the apex of the lung in front, when there is consolidation at the base behind. Under these conditions cracked-pot resonance may sometimes be obtained. Over the healthy lung there is exaggerated resonance. On auscultation over the consolidated portion there are bronchial breathing and bronchial voice, the area over which they are heard being sharply defined. Râles are usually absent, but there may be pleuritic friction sounds.

In the stage of resolution there is a gradual disappearance of the signs of consolidation. The pure bronchial is replaced by broncho-vesicular breathing, the vesicular element gradually predominating. Moist râles of all varieties are heard. Usually the most persistent signs are slight dulness or diminished resonance, with a respiratory murmur which is feebler than normal and a little higher in pitch; sometimes there are also dry friction sounds. These signs may persist for two or three weeks.

Exceptional physical signs.—While in the majority of cases the signs of consolidation are distinct on or before the fourth day, in not a few they may be delayed much longer. Of eighty-two cases in which the day was noted on which consolidation was found, it was not until the fifth day or later in one fourth the number. In six of them, although carefully and repeatedly examined, no consolidation was found until the seventh day or later and in one case not until the twelfth day. It has been customary to look upon these cases of delayed or concealed physical signs as cases of central pneumonia. That pneumonia may exist in the centre of a lung for a number of days is, to my mind, extremely improbable. At autopsy, superficial pneumonia I have very frequently seen, but central pneumonia never. There are two regions in which pneumonia may exist and yet not be accessible by our means of physical examination, viz., at the apex of the lung in the part covered by the shoulder, and along the posterior border of the lung where it lies against the vertebræ. In either

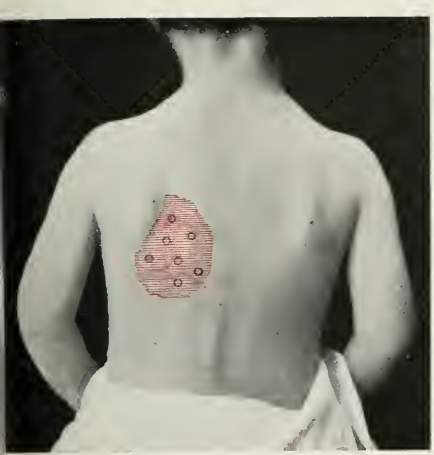


Fig. 96.—First stage. Congestion of left lower lobe, with crepitant râles. Feeble breathing of a rude character, with slight dulness.

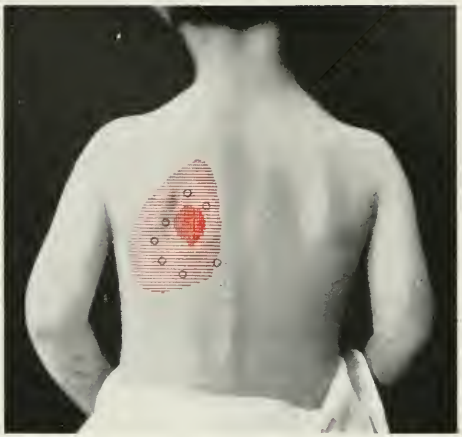
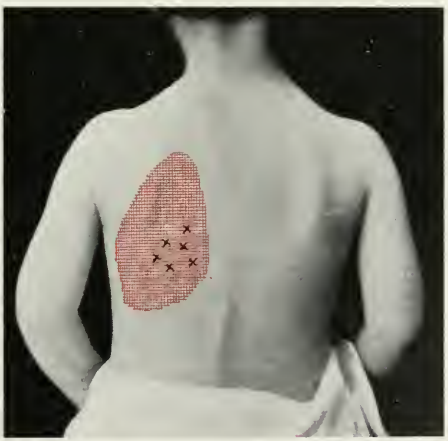


Fig. 97.—In the centre of the area, a small spot of pure bronchial breathing and voice; surrounding this an occasional crepitant râle, with broncho-vesicular breathing and slight dulness.



ig. 98.—Second stage. Complete consolidation of left lower lobe. Pure bronchial breathing and bronchial voice; marked dulness; increased vocal fremitus, and at the lower part a few friction sounds.

Note.—During resolution the signs take the inverse order: those of Fig. 98 give place to ose of Fig. 97, and these in turn to those of Fig. 96. In addition, many coarse râles may heard.

of these situations pneumonia may be present without our being able to find it. It is quite common in cases with late physical signs that the first distinctive evidences of disease are found high in the axilla, or beneath the clavicle in front, and these regions should be closely watched in doubtful cases. Sometimes the delay is best explained by assuming that constitutional symptoms due to the pneumococcus infection, may be present for several days before the development of the local lesion in the lung.

Complications.—The occurrence of dry pleurisy over the consolidated portion of the lung is so constant that it can hardly be considered a complication. A slight serous exudation of two or three ounces is not uncommon, but more than this is very rare in young children. In the most severe cases of pleurisy there is an excessive exudation of fibrin and pus. This occurred in eight per cent of my cases. This variety is known clinically as pleuro-pneumonia, and will be considered separately. Pericarditis is rare; it was seen in only two of my cases; in both it was associated with pleuro-pneumonia of the left side, the exudation resembling that found on the pleura. It rarely gives rise to any new symptoms. Meningitis was seen twice, once with pleuro-pneumonia. It is nearly always ushered in by repeated attacks of vomiting or convulsions. Its course is short and progressive. Peritonitis was seen once, also associated with pleuro-pneumonia. Occasionally there is gastro-enteritis.

Course and Termination.—In the great majority of cases lobar pneumonia terminates either in perfect recovery or in death. When ending in recovery, resolution commonly begins immediately upon the cessation of the fever, and is complete in about a week. Delayed resolution is very rare; chronic pneumonia and tuberculosis are also extremely infrequent as sequelæ, but empyema is quite common. Its symptoms sometimes develop immediately after the pneumonia, the temperature continuing high; or there may be an interval of a few days before the development of the pleural symptoms. Some pleuritic adhesions probably remain in every case in which there has been much dry pleurisy, and when severe and extensive, these may be the cause of subsequent symptoms, like any other dry pleurisy.

Death from uncomplicated pneumonia may be due to exhaustion, or to heart failure, with or without failure of the respiration. The signs of heart failure sometimes develop quite rapidly in cases which are apparently doing well. The symptoms are: coldness of the hands and feet, then of the legs and arms; a rapid, compressible, and sometimes irregular pulse; muscular weakness and pallor, but usually no cyanosis. The symptoms of respiratory failure are: very rapid superficial respirations, sometimes 100 a minute; blueness of the lips and finger nails; often a leaden hue of the whole body; there are loud tracheal râles, and recession of all the soft parts of the chest on inspiration.

Death may result early in the disease, where the pneumonia has spread rapidly, involving both lungs. The earliest deaths I have seen were on the fourth day, and were due to a failure of the heart and respiration. In most of the uncomplicated fatal cases, death results from heart failure at about the time of the crisis. In the complicated cases death usually occurs in the second week. I once knew fatal meningitis to develop at the end of the fourth week.

Diagnosis.—The most characteristic differences between broncho- and lobar pneumonia are shown in the following table:

BRONCHO-PNEUMONIA.	LOBAR PNEUMONIA.
1. More than half the cases secondary.	1. Almost always primary.
2. Under three, chiefly under two years.	2. Most common between three and eight years.
3. Occurs more frequently in delicate and debilitated children.	3. More often in those previously healthy.
4. Bacteria—in primary cases, usually the pneumococcus; in secondary cases, chiefly the streptococcus, but usually mixed infection.	4. The pneumococcus.
5. Products of inflammation chiefly cellular; process often diffuse.	5. Chiefly fibrin; process circumscribed.
6. Onset often gradual, sometimes insidious, especially when secondary.	6. Onset sudden, with well-marked symptoms.
7. No typical course; fever often lasts three or four weeks; rarely terminates by crisis.	7. Typical course; crisis usually from fifth to eighth day.
8. Involves both lungs as a rule, most frequently lower lobes posteriorly.	8. Usually one lobe or a part of a lobe; left base most frequently, right apex next.
9. Signs of bronchitis mingled with those of consolidation; râles in other parts of the same lung, or in the opposite lung, throughout the disease.	9. Râles only early, and during resolution; frequently no signs in opposite lung.
10. Consolidation later—fourth to seventh day: there may be none; apt to be incomplete; shades off gradually.	10. Consolidation earlier; second or third day. Consolidation complete; area usually sharply defined.
11. Resolution slow, one week to two months; often incomplete; strong tendency to become chronic.	11. Resolution rapid, usually complete within a week.
12. Relapses and second attacks frequent.	12. Both are rare.
13. Sequelæ: Empyema, chronic interstitial pneumonia, sometimes tuberculosis.	13. No sequelæ except empyema.
14. Prognosis always serious from the age and the circumstances under which disease occurs.	14. Prognosis good; rarely fatal except from complications—empyema, meningitis, pericarditis.
15. Hospital mortality 50 per cent of primary cases, 65 per cent of all cases.	15. Mortality 4 per cent of all cases.

In the majority of cases the symptoms are plain and the physical signs so typical that it is difficult to overlook pneumonia if any degree of care is used in the examination of the patient. The characteristic features are the sudden onset, with vomiting, convulsions, or chill; prostration; rapid respiration, with the expiratory moan; a temperature of 102° to 105° F.; cough and thoracic pain; and the physical signs of a rapidly developing, circumscribed consolidation in one lobe or a portion of a lobe. The difficulties in diagnosis are due to the great variation that is seen in the general symptoms, and to the late appearance of the physical signs. The error usually made is to mistake pneumonia for some other disease, rather than to mistake some other disease for pneumonia. On account of its frequency in children, pneumonia should always be excluded before accepting any other explanation of a continuously high temperature. It is surprising to find how often obscure and indefinite symptoms accompanied by high fever, are due to pneumonia. The rule should be followed, in all cases of acute illness, of making a thorough examination of the chest daily until the diagnosis is clear. If to high temperature rapid respiration is added, one should always suspect the lungs, no matter what the other symptoms may be. It not infrequently happens that the general symptoms are quite characteristic and yet the physical signs appear late. In such cases pneumonia should always be looked for high in the axilla or just beneath the clavicle, since it is particularly in the cases of apex pneumonia that this obscurity is likely to exist. If frequent and thorough examinations of the chest are made, very few cases will be overlooked.

In their onset, scarlet fever, tonsillitis, and gastro-enteritis may all resemble pneumonia. Scarlet fever is recognised by the sore throat and the characteristic eruption on the second day; tonsillitis, by the local symptoms. Pneumonia is distinguished from gastro-enteritis, by the fact that the temperature and prostration are out of all proportion to the intestinal symptoms, and continue even after these symptoms have subsided. It is most likely to be mistaken for gastro-enteritis in summer, and in infancy, when it often begins with vomiting and diarrhœa. Malaria is distinguished from lobar pneumonia by the points mentioned in the diagnosis of bronchopneumonia (page 507). From all other general diseases, pneumonia is to be differentiated by the physical signs.

Pneumonia with marked cerebral symptoms sometimes resembles cerebro-spinal meningitis. In both we may have the abrupt onset, convulsions, delirium or stupor, opisthotonus, and prostration. In pneumonia the temperature is usually higher than in meningitis; the pulse is never slow and intermittent; the respiration is rapid, instead of slow and irregular; and the stupor is usually less profound; and there are no localized paralyses. In meningitis there is a steady increase in the severity of the nervous symptoms for the first three or four days; in pneumonia they

are as a rule most marked during the first twenty-four or forty-eight hours, and then gradually diminish, always subsiding completely at the crisis. While most of the individual symptoms belonging to meningitis may be present, they are usually less severe and less persistent in pneumonia.

The question sometimes arises, in a case of pneumonia, whether the cerebral symptoms are functional, or whether meningitis also exists. If the nervous symptoms are present from the beginning, there is probably no meningitis. If they develop suddenly during the course or toward the close of the disease, meningitis should be suspected.

Lobar pneumonia is to be differentiated from a pleuritic effusion. The most common mistake which I have seen made is to confound empyema with unresolved pneumonia. The latter is very infrequent, so that the probabilities are always strongly in favour of the diagnosis of empyema. In pneumonia rarely, if ever, is the whole lung affected. There are increased vocal fremitus, dulness, bronchial voice and breathing, and occasionally râles or friction sounds. In empyema the whole lung is often affected, there are displacement of the heart, flatness on percussion, diminished or absent vocal fremitus, and although bronchial voice and breathing are present, they are usually distant and feeble. There are no râles or friction sounds. In doubtful cases an exploratory puncture should always be made. Serous effusions are rare, but are differentiated by the same signs as empyema.

Prognosis.—There is probably no disease in which the patient appears so ill, and where there is really so little danger to life, as in lobar pneumonia in a child over three years old. Of 1,295 collected cases, chiefly from hospital practice, there were but 39 deaths, a mortality of three per cent. In 187 cases of my own there were 21 deaths, a mortality of eleven per cent. Only one of the fatal cases was over two years old. The difference between the mortality among my cases and the general mortality given, is due to the fact that a large proportion of the first group were observed in children under two years, while of the collected cases the vast majority were in older children. Combining the above figures, we have a total of 1,482 cases with 60 deaths, a mortality of four per cent. In nearly all my cases death was due either to complications or to very extensive disease, as when both lungs were involved, or nearly the whole of one lung. In only one case was an uncomplicated pneumonia of a single lobe fatal.

The prognosis depends upon the age of the patient, the presence or absence of complications, and the extent of the disease. These factors are to be taken into consideration rather than any special symptoms. Early convulsions do not materially affect the prognosis. Of seven such cases only one was fatal. Late convulsions are always very unfavourable, indicating either exhaustion, toxæmia, or the development of meningitis.

The development of vomiting or diarrhœa late in the disease is also unfavourable, especially in infants.

A temperature range between 102° and 105° F. is the rule, and within these limits the fever does not affect the prognosis. Even very high temperature does not increase the danger from the disease as much as would be expected. Of fifteen cases in which the temperature touched 106° F. or over, all but three recovered; while of six cases in which it was 106·5° or over, only one died. The highest recorded temperature in my cases—107·5° F.—was in a patient who recovered. A transient rise, even though the temperature may go very high, is not often serious. Much more serious is a fever which remains steadily above 105° F., as in most cases this accompanies either very extensive disease or pleuro-pneumonia. The continuance of the fever after the tenth day is a bad symptom, for, although the crisis may be postponed until the twelfth day and occur normally, such a prolonged temperature is apt to be an indication of a new focus of disease or the development of complications.

It is an unfavourable sign for resolution not to begin as soon as the temperature becomes normal. There should then be apprehended a relapse, the development of empyema, or of some other complication.

Treatment.—In the treatment of lobar pneumonia in children, several cardinal facts are to be kept in mind. It is a self-limited disease, having a strong tendency to recovery in the great majority of cases regardless of the treatment adopted. The fatal cases are almost always in children under three years of age; the rare deaths in older ones are usually due to complications. I believe that there is no means of treatment by which we can abort pneumonia or shorten its course. It follows, therefore, that the indications are, so far as possible, to make the patient comfortable during his illness, to prevent complications, and to treat the individual symptoms as they arise.

In perhaps the majority of cases, hygienic treatment is all that is required. The patient should be kept in bed, no matter how mild the attack; he should be lightly covered, kept as quiet as possible, and allowed plenty of fresh air in the room. Food should be given at regular intervals, never oftener than every two hours, and usually only every four hours. It should not be forced when the patient is suffering only from thirst. These measures, careful nursing, an occasional dose of phenacetine when the patient is very restless, fretful, or sleepless, and cold sponging when the temperature makes him uncomfortable, are usually all that is necessary, except to keep a sharp lookout for complications.

Special symptoms may require treatment. The nervous symptoms are, in most cases, better controlled by phenacetine than by opiates. Often a single dose in twenty-four hours is enough. Sometimes sponging with

tepid water is better than drugs. Severe nervous symptoms, such as delirium, stupor, great restlessness with impending convulsions, when associated with high temperature, call for ice to the head, cold sponging, or the cold pack or bath. Pain, if moderate, may be relieved by counter-irritation by a mustard paste or by a hot poultice; if severe, morphine must be used in addition. The cough is rarely severe enough to require treatment. When it is so severe as to prevent sleep, small doses of Dover's powder or codeia should be given. Antipyretic measures are not necessarily called for if the temperature is high. This not infrequently continues for a few hours while the patient may be quiet and appear perfectly comfortable. Under such conditions the temperature should be closely watched, but not necessarily interfered with unless other symptoms develop. The nervous symptoms are a better guide than the thermometer to the use of antipyretics. When they exist, even with a moderate elevation of temperature, interference is indicated. Cold I believe to be the safest and most certain antipyretic we possess. It may be given as a cold sponge bath or the cold pack (pages 47, 48). There is no objection to the bath except the prejudice of the laity. While cold is applied to the trunk the extremities should be closely watched, and heat applied if necessary. The duration of the pack or bath, and the frequency of their use, will depend upon the individual case. Stimulants are not required in the majority of cases. They are called for when the pulse is weak, compressible, and rapid, when the face is pale and the extremities are cold. The same stimulants are to be employed, and in the same way, as in broncho-pneumonia (page 510). Cardiac stimulants are usually required in larger quantity at the time of and just after the crisis. Respiratory stimulants are indicated as in broncho-pneumonia.

PLEURO-PNEUMONIA.

Under this term are included cases of pneumonia with an excessive amount of pleurisy, the two processes uniting to produce a single clinical type of disease.

In nearly all cases of lobar pneumonia there is a certain amount of inflammation of the pulmonary pleura, and also in those cases of broncho-pneumonia which are accompanied by any marked degree of consolidation. In both of these the pleurisy is usually co-extensive with the consolidation. But in certain cases, in both forms of pneumonia, the amount of pleurisy is excessive, and this so modifies the symptoms and course of the disease as to require for them a separate consideration. In some it appears that the inflammatory process begins almost simultaneously in the lung and in the pleura; while in others the pleurisy follows the pneumonia. These cases are, I believe, almost invariably due to the pneumococcus, although in some there is a mixed infection.

In 398 hospital cases of pneumonia there were 27, or 6·8 per cent,

which could be classed as pleuro-pneumonia, the diagnosis being confirmed either by autopsy or operation. Of 190 fatal cases, 12·5 per cent were pleuro-pneumonia. Most of these hospital patients were under three years of age, and the disease is, I think, more frequent at this period than in older children.

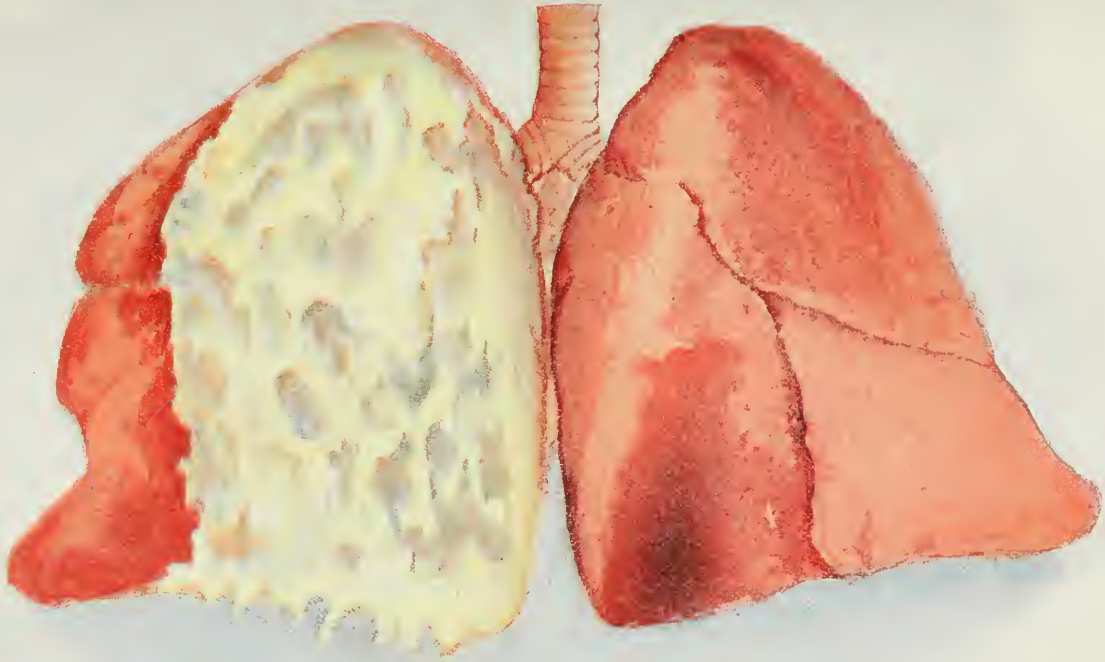
Lesions.—Of these 27 cases, 17 were classed as broncho-pneumonia and 10 as lobar pneumonia. The left lung was more frequently affected than the right in the proportion of three to two. In most of the cases the pleura covering the entire lung was involved, even though the pneumonia affected but a single lobe, or only a part of a lobe. In nearly half the cases both lungs were involved, but one to a very much less extent than the other. In a small number of cases the pleurisy was limited to the posterior surface of the lung, stopping at the axillary line.

In pleuro-pneumonia both the visceral and the parietal pleura are coated with a layer of yellowish-green fibrin, in thick, shaggy masses, by which the lung is adherent to the chest wall, the diaphragm, and the pericardium (Plate XIII). The exudation varies between one eighth and one half an inch in thickness. It can often be stripped from the lung or scraped from the chest wall by the handful. In its meshes small pockets may form, which contain only a few drops, or sometimes a drachm of pus, or less frequently serum. This is the condition in which the lung is usually found where death has occurred at the height of the disease. If the process has lasted longer, larger collections of pus may be present. The lung itself shows the usual changes of pneumonia, and if there has been any considerable accumulation of fluid, there are in addition the evidences of compression.

With pleuro-pneumonia of the left side, the pericardium is occasionally involved. This was seen in two of my cases, the lesions closely resembling those of the pleura. In two cases there was also meningitis, and in one peritonitis, the exudation in all cases having the same characteristics.

An inflammation of the intensity described is very often fatal in the acute stage, if the patient is a child under two years old. Occasionally at this age, and very frequently in older children, we see the later stages of the process. The most frequent course is for more and more pus to be poured out from the inflamed pleura until the chest is filled, the case becoming thus one of empyema. Sometimes the fluid is serous instead of purulent, but this is very rare in infancy. Under other circumstances the exudation is partly absorbed, but the greater part becomes organized so as to form a thick jacket of fibrous tissue which binds the lobe or lung to the chest wall, and interferes seriously with its subsequent full expansion. Chronic interstitial pneumonia may follow.

Symptoms.—There is little which distinguishes a case of pleuro-pneumonia except the severity of all the constitutional symptoms; the tem-



ACUTE PLEURO-PNEUMONIA.

The lungs have been separated in front and spread out to show the whole external surface as seen from behind. The left lung, with the exception of a narrow strip along its anterior border, is completely covered with a thick, ragged exudation of fibrin. The left lower lobe was hepatized; the right lower lobe deeply congested.

From a child one year old, who died in the New York Infant Asylum.

perature is often higher, the prostration greater, and the patient in every way impresses one as being more seriously ill than with ordinary pneumonia. Sometimes the thoracic pain is more severe and more constant than is usual in pneumonia. The diagnosis, however, is to be made by the physical signs.

In the early stage the pleuritic friction sounds are unusually prominent; after two or three days the signs of consolidation come out clearly in most cases, but still accompanied by loud friction sounds. After the fibrinous exudation is very abundant, the signs are often obscure and confusing, and there may be at no time well-defined signs of consolidation. There is usually a mingling of the signs of consolidation with those of effusion. There is marked dulness, and sometimes flatness. The vocal fremitus is apt to be diminished, and it may be absent. Bronchial voice and breathing are heard, but they are not distinct as in consolidation; they are, however, feeble and distant, as over fluid. There are usually coarse, moist, crackling pleuritic sounds, but these may be absent. The signs may be found over one entire lung, or they may be limited to the posterior region, and even to a single lobe. They resemble those present over fluid, with one exception—viz., the heart is not displaced. If an exploratory puncture is made, nothing is found; occasionally the exploring needle happens to strike one of the small pockets of pus in the meshes of the fibrin, and a few drops of clear pus are withdrawn. If an incision is made under the supposition that the case is one of empyema, no more pus may be found, the surgeon coming upon the pulmonary adhesions as soon as the chest is opened. There is scarcely any condition in the chest giving signs more puzzling than those just enumerated. They are, however, easily explained by the pathological conditions.

Prognosis.—The prognosis in pleuro-pneumonia is much worse than in simple pneumonia. In infants the outlook is very bad, the majority of cases dying during the acute stage, usually in the second week. Very young children may be overwhelmed with the extent and the intensity of the inflammation, and die in four or five days. In children over two years old the most frequent result is for the case to go on to empyema, which with proper treatment usually terminates in recovery. Where there is organization of the fibrin with the production of extensive adhesions, the ultimate result is often not so favourable as when empyema develops. Convalescence is usually slow, and the patients are liable to exacerbations of pleurisy; they may suffer for years from the partial crippling of one lung.

Diagnosis.—This is to be made only by the physical signs. A differential diagnosis from fluid in the chest can in some cases be made only by an exploratory puncture.

Treatment.—Cases of pleuro-pneumonia require no special treatment. In general they are to be managed like the ordinary cases of pneumonia

of the severe type. In some, the excessive pain may call for more active counter-irritation and a freer use of opium than in other forms of pneumonia, and the greater prostration may require that stimulants be given earlier and in larger quantities.

HYPOSTATIC PNEUMONIA.

This can not often be recognised clinically, but it is very frequently seen upon the post-mortem table. It is present in some degree in almost every case where an infant has died of chronic disease. It is particularly frequent in those who have died of marasmus. It is sometimes described as "strip pneumonia," on account of its position. It invariably occupies a strip along the posterior border of both lungs, and usually of both the upper and lower lobes. This is from one to two inches wide, of a uniform dark-red colour, and is sharply outlined. The pleura is not involved, and the remainder of the lung may be normal, congested, or slightly emphysematous. On section, it is seen that the pneumonic area is quite superficial, rarely involving the lung to a greater depth than half an inch. Under the microscope there is found a distention of the small blood-vessels in the affected area, and the air vesicles are filled with many red blood-globules, epithelial cells, and a few leucocytes. Between the areas of consolidation are groups of air vesicles which are normal, congested, or collapsed. It is a lobular rather than a broncho-pneumonia. The lesions in this form of pneumonia are probably the result of venous stasis, owing to the child's recumbent position.

At autopsy the condition may be confounded with atelectasis; this, however, is almost invariably more marked in the interior of the lung, while pneumonia is always more marked upon the surface. The two conditions are sometimes associated. Little significance is to be attached to the finding of hypostatic pneumonia at autopsy, and it alone should never be regarded as a sufficient cause of death, although it is perhaps the only lesion present. During life it may give rise to fine moist râles, which are heard along the spine, usually upon both sides; but there is neither dulness nor bronchial breathing.

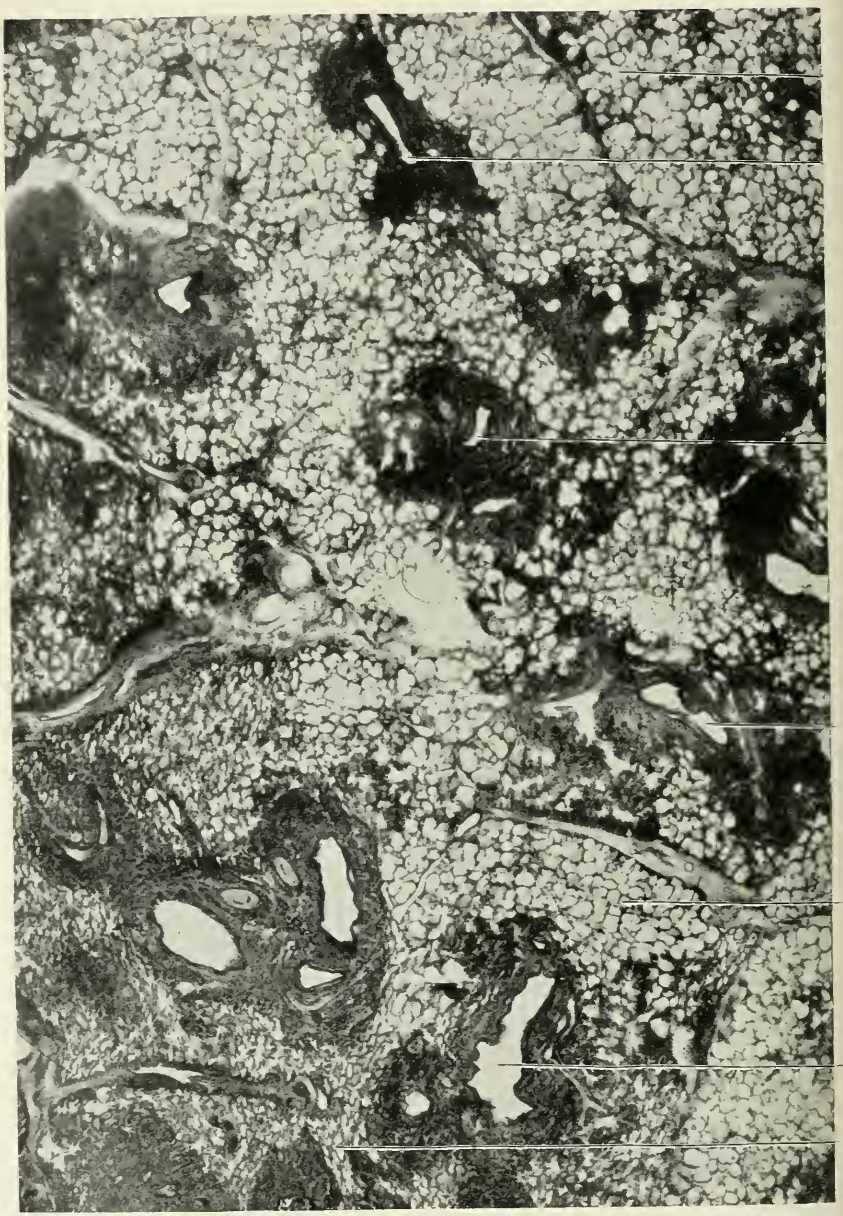
The treatment is that of the primary disease.

CHRONIC BRONCHO-PNEUMONIA—CHRONIC INTERSTITIAL PNEUMONIA—BRONCHIECTASIS.

This is an inflammation of the connective-tissue framework of the lung, involving the stroma, the alveolar septa, the walls of the bronchi, and the pleura. It is usually accompanied by cylindrical dilatation of the bronchi—bronchiectasis.

Etiology.—In children, as in adults, this process is most frequently associated with pulmonary tuberculosis; but in early life it is not an in-

PLATE XIV.



Chronic Broncho-Pneumonia.

In the greater part of the specimen the disease is limited to the vicinity of the small bronchi, A A A, each of which is surrounded by a zone of new connective tissue, the result of the inflammatory process, the intervening lung tissue, B B, being normal. In the lower left-hand portion, the disease is more diffuse: the air vesicles, C, between the areas of new connective tissue are greatly compressed, and in some places entirely obliterated. (After Delafield.)

frequent condition apart from tuberculosis. The non-tuberculous cases, as a rule, are preceded by an attack of acute broncho-pneumonia, sometimes by several such attacks, separated by longer or shorter intervals.

Lesions.—The part of the lung affected may be an entire lobe, but usually it is a portion of one lobe, or there are areas in more than one lobe. There are dense connective-tissue adhesions binding the diseased part to the chest wall, to the diaphragm and to the pericardium, often so firmly that the lung is torn on removal. The affected lung is smaller than in health; it is hard, tough, and fibrous. Surrounding the fibrous portions are emphysematous areas. On section, the process is seen to be somewhat irregularly distributed through the lung, the lesion being usually most marked in the vicinity of the smaller bronchi, and sometimes seen only there, the intervening lung being nearly normal (Plate XIV). In some portions, where the process is most advanced, almost all trace of lung tissue has disappeared, the part resembling a solid fibrous tumour, through which run the bronchial tubes, usually much dilated. In places this dilatation may be sufficient to form cavities of considerable size. The bronchial glands are often enlarged to the size of a hazelnut, and they may be tuberculous.

Upon examination with the microscope, the pleura is found greatly thickened, with bands of new fibrous tissue passing from it into the lung. The walls of the small bronchi are generally thickened, but in some places they have undergone cylindrical dilatation, and are filled with pus. The walls of the alveoli are greatly thickened from the proliferation of the connective-tissue elements, and the alveoli are filled with organized inflammatory products, so that they are nearly or quite obliterated. The stroma is greatly increased in amount throughout the affected lung.

Symptoms.—In most of the cases there is a history of an attack of acute broncho-pneumonia, from which the child made a slow convalescence, remaining pale, anæmic, and sometimes wasted for several months. Improvement then takes place in the general symptoms, the appetite and strength return, and in many cases the lost weight is nearly or quite regained. However, neither the pulmonary symptoms nor the physical signs entirely disappear. There remains a dry, hard cough, which at times is severe. Pains in the chest are occasionally complained of, and perhaps shortness of breath on exertion is noticed. Examination shows a persistence of the dulness on percussion, with a rude or broncho-vesicular respiratory murmur of very feeble intensity. Little change may take place in these signs for months; then an acute attack of bronchitis or broncho-pneumonia may occur. If the latter, the same lung is affected, and a fresh consolidation is added to the previous disease. This attack may not be very severe, but it drags on for several weeks, with slight fever and little or no change in the physical signs. Partial resolution may then take

place, but the lung is left much more seriously crippled than before. In many cases there is a history of several such attacks, each one leaving the lung a little worse than it found it.

The characteristic physical signs of chronic broncho-pneumonia are not usually present until the process has continued for many months, sometimes for several years. They may involve part of a lobe, or they may be present over an entire lobe, or even an entire lung. On inspection, there is seen in a well-marked case, a retraction of the chest, which is especially noticeable when the disease is situated at the apex of the lung. The vocal fremitus is usually increased, but it may not be abnormal. There is marked dulness, often flatness, over the affected area, with exaggerated resonance over the rest of the lung. The area of flatnessis not sharply circumscribed, but shades off gradually. The most striking thing on auscultation is the very feeble respiratory murmur; in many cases the lung is almost silent. In other cases the respiration is distinctly bronchial in character, and, if marked bronchiectasis exists, it may be cavernous. Râles and friction sounds are usually absent except during an acute exacerbation of the symptoms, when they may be heard as in any attack of broncho-pneumonia. There is no displacement of the heart.

The course of these cases is always uncertain. When once present the lesions are permanent, and there is always a tendency to increase rapidly or slowly, according to the child's vigour of constitution, its surroundings, and, most of all, the frequency with which exacerbations occur. If the disease is extensive the general health is so undermined that the patient succumbs either to some intercurrent disease or to an acute attack of pneumonia; if limited in area, the process may be arrested and the patient recover, always, however, to be more or less embarrassed because of the crippling of a part of one lung. Not a small number of these children ultimately die of tuberculosis, and in such cases it is always a difficult matter to decide whether tuberculosis was present from the beginning, or whether there was subsequent infection. The classical symptoms which are presented by adults with bronchiectasis are rarely seen in young children.

Prognosis.—From what has already been said, it will be evident that the prognosis in these cases is always doubtful as to the ultimate result. It depends on the extent of the disease, the patient's age and constitution, and on our ability to prevent by treatment, climatic and otherwise, the occurrence of acute exacerbations. Under the most favourable conditions, a few patients may recover completely so far as symptoms are concerned; but the majority at best remain delicate during childhood, or even throughout life.

Diagnosis.—The most important thing is to distinguish between the simple and the tuberculous cases, and this, it must be confessed, is in the

majority impossible. Repeatedly have I seen a process proved at autopsy to be simple, which all who had observed the case had unhesitatingly pronounced to be tuberculous, and quite as often the opposite has been true. If the family history is good, if the patient lives in the country, if his symptoms began with a well-defined acute attack of pneumonia, if the seat of disease is the base of one lung, and if the examination of the sputum is negative, the process is probably simple. If the family history is doubtful or is positively tuberculous, if the patient lives in the city, and especially if he is an inmate of an institution or if his home is among the tenements, if the initial symptoms were indefinite, if the seat of disease is the axilla, the mammary region, or the apex in front, the process is probably tuberculous. The discovery of tubercle bacilli in the sputum is, of course, conclusive. Even the course of the disease may not settle the diagnosis, unless there develop in the bones or in other viscera, lesions undoubtedly tuberculous.

Treatment.—Nothing has any essential influence upon the disease except change of climate. This should be the same as for tuberculous cases. The treatment of the patient has for its object the maintenance of the general nutrition at its highest point, by careful feeding, judicious exercise, and by most of the measures enumerated in the chapter on Malnutrition. Cod-liver oil should be given throughout every winter season. The cough may be treated as in cases of chronic bronchitis.

GANGRENE OF THE LUNG.

Pulmonary gangrene is quite rare in children, although it is probably more common than in adults. It is most frequently associated with pneumonia. It is usually circumscribed, and in the majority of cases it is latent.

Etiology.—Children of all ages may be affected; all of my own cases have been under three years old, the youngest being an infant of four months. It occurs for the most part in children who are ill-conditioned, feeble, or cachectic, and often follows one of the infectious diseases, particularly measles. In such cases it may be associated with gangrene of the mouth or of the vulva. It is seen in general pyæmia, and has followed caries of the petrous bone. Of the local causes, altogether the most frequent is broncho-pneumonia. Of nine cases which have come under my personal observation, six complicated acute broncho-pneumonia and one lobar pneumonia. It has been present in three per cent of my autopsies upon cases of pneumonia. It may accompany pulmonary tuberculosis, bronchiectasis, and pulmonary apoplexy, or it may be due to a foreign body in one of the bronchi. The immediate cause of the necrotic process is interference with the circulation in a part of the lung, which is usually due to thrombosis or embolism of some of the branches of the pulmonary artery. To this there is added the entrance of putrefactive

bacteria. In some cases the process may begin as a septic thrombosis, this infection originating in some process in a distant part of the body.

Lesions.—According to general experience, the lower lobes are more frequently affected than the upper, and this is borne out by my own cases. The surface of the lung, rather than the central portions, are most often involved.

Two forms of gangrene may be seen: the diffuse form, which affects a whole lobe, or even a whole lung; and the circumscribed form, which occurs in a number of small scattered areas, usually from half an inch to two inches in diameter. The latter is the variety usually seen in children. In the diffuse form the lung is of a dirty green or brown colour, moist, and emits a gangrenous odour. In the circumscribed form, when occurring in pneumonia, the parts affected are of a gray or green colour, usually wedge-shaped, with the base at the surface of the lung. In the early stage they are not softened, and have no gangrenous odour; later, both these conditions may be present, and masses of necrotic lung tissue may be found in a cavity with ragged walls, partly filled with fetid pus. Careful dissection will reveal, in many cases, the presence of thrombi in the vessels leading to the gangrenous parts. The later stages of the process are very rarely seen. However, in some cases the gangrenous masses may be coughed up and the cavity closed by cicatrization. This is more likely to happen where there is but one area, as when the process is due to the presence of a foreign body. Sometimes rupture into the pleura takes place, and empyema or pneumothorax follows.

Two unique cases of necrosis of the lung have come to my notice; they were in all respects similar. The surface of the lung was of a uniform dark reddish-brown, and seemed to be slightly softened. On section, a large part of the lower lobe was of a dark-red colour and of a semifluid consistency, the pulmonary tissue being so completely disintegrated that it could be washed away with a stream of water. There was no gangrenous odour. No thrombosis was found in these cases, and no explanation of their origin was discovered even by microscopical examination. There was some broncho-pneumonia present. Both cases occurred in infants suffering from marasmus. These are perhaps to be classed as examples of diffuse gangrene, although they differed very markedly from the form usually seen.

Symptoms.—There are but two distinctive symptoms of pulmonary gangrene: the gangrenous odour of the breath, and the expectoration of masses of necrotic lung tissue. In the cases associated with acute pneumonia, which include the majority of those seen, death nearly always takes place before there is any separation of the sloughs, and even before very active decomposition in the necrotic areas has occurred. Both the peculiar symptoms are therefore wanting, and the diagnosis is made only at the autopsy. This has been true of all the cases which have come

under my own observation. But these patients, with one exception, were infants. In older children, particularly in cases secondary to the entrance of a foreign body, the characteristic symptoms are more frequently seen, and there may be a third symptom—hæmorrhage. This is present in about one fourth of the cases (Rilliet and Barthez), and may be fatal. The general symptoms associated with gangrene are those of profound depression, and often all the signs of the typhoid condition are present.

From what has already been said, it will be evident that the diagnosis is very difficult in children, and that most cases of gangrene of the lung are overlooked. When the characteristic odour of the breath is present, conditions in the mouth from which it might arise must first be excluded. The physical signs differ in no respect from those of ordinary cases of pneumonia. The termination is almost always in death. This is due not only to the condition itself, but to the circumstances in which it is seen.

Treatment.—The general treatment is supporting and stimulating, as in all very severe cases of pneumonia. For the local process but little can be done, except the inhalation of antiseptics, of which creosote and turpentine are undoubtedly the best.

ACQUIRED ATELECTASIS—PULMONARY COLLAPSE.

These terms are applied to a state of the lung resembling the fœtal condition, but which occurs in a lung which has once been expanded. Two varieties are met with: collapse from compression and collapse from obstruction.

Collapse from Compression.—The principal cause of this form is pleuritic effusion. It may also be produced by pneumothorax, enlargement of the heart, pericardial effusion, deformities of the chest from rickets or Pott's disease, and tumours of the mediastinum or thoracic wall. In these conditions, on account of the external pressure, the air vesicles are not filled, although the bronchi are pervious. The elasticity of the vesicles tends to expel the air which they contain. This form of collapse may be complete or partial, according to the cause. After it has existed for a considerable time, changes may take place in the lung which render expansion difficult or impossible. Unless, however, there are thick pleuritic adhesions, expansion often takes place readily after many weeks and even months, as in most cases it is the condition of the pleura, rather than of the lung itself, which interferes with it. In recent cases only moderate force is required at autopsy to produce expansion; in old cases it is more difficult and may be impossible. The symptoms and signs are those of the original disease.

Treatment is available chiefly in that form which follows pleuritic effusion, and will be considered in the chapter on Empyema.

Collapse from Obstruction.—This is due to two factors: blocking of either the large or small bronchial tubes, and feeble inspiratory force. The importance of collapse from obstruction as a factor in the acute diseases of the lung in infancy has, I think, been very much exaggerated. It is well known that whenever a large or small bronchus is completely obstructed by a foreign body so that the entrance of air is prevented, the portion of the lung to which the bronchus is distributed gradually becomes collapsed. If it is one of the primary bronchi which is occluded, a whole lung may be collapsed; if one of the lobar divisions, an entire lobe; if one of the smaller divisions, a small area, usually somewhat wedge-shaped. The collapse does not take place immediately, but the contents of the air vesicles are gradually absorbed by the blood, requiring perhaps twenty-four hours, or even longer. According to Lichtheim, the oxygen is first absorbed, then the carbon dioxide, and finally the nitrogen. The collapsed portion of the lung is smaller than the inflated portions, and consequently is slightly depressed below the surface. It is of a dark-red colour, very vascular, and to the naked eye resembles a pneumonic area, which it may subsequently become.

It has been the fashion since the writings of Gairdner to explain the development of broncho-pneumonia from bronchitis of the smaller tubes, through the intervention of pulmonary collapse. It has been assumed that the obstruction of the small bronchi from swelling of their walls and the accumulation of secretion, produced the same result as the plugging of a bronchus by a foreign body. Without going into a full discussion of the subject, I will only say that from personal observations upon nearly one thousand autopsies upon infants, in which are included a very large number of the acute pulmonary diseases of all varieties, I have found very little support for this theory. In acute bronchitis of the smaller tubes the lumen is narrowed, but not often to such a degree as entirely to prevent the entrance of air. This condition of stenosis results, as a rule, in the production of emphysema, not atelectasis. Such, at least, has been the condition in the cases in which I have had an opportunity to make autopsies in the earliest stage of broncho-pneumonia, when it has developed from a generalized bronchitis of the fine tubes. It is certainly true that there are very often groups of collapsed air vesicles found surrounding those which are the seat of pneumonia, but these are neither an essential nor a very important part of the lesion. Anything approaching collapse of a large part of the lung, or even of a lobe, I have never seen, either in pertussis or in acute bronchitis, nor do I believe that it occurs in the way mentioned.

There is occasionally seen, usually in very delicate infants or in those who are markedly rachitic, a form of collapse which comes on very gradually. It is accompanied by bronchitis affecting the tubes in the

dependent part of the lung. Its seat is the lower lobes posteriorly, sometimes also the posterior border of the upper lobes. In general appearance it may resemble the congenital form of atelectasis. Under the microscope there is almost invariably found accompanying the collapse, lobular pneumonia and bronchitis of the tubes in the affected regions.

The symptoms are much the same as in persistent congenital atelectasis. In marked cases the respiration is rapid, and there may be inspiratory dyspnœa with deep recession of the chest walls, especially if there is rickets. There is also cyanosis of variable intensity, which may be constant or intermittent. There are usually present a short cough, feeble cry, and poor circulation with cold extremities. The temperature is not elevated, but frequently is subnormal. The physical signs are very uncertain. There may be slight dulness and very feeble respiratory murmur over the affected areas, occasionally accompanied by moist râles. The course and termination are the same as those seen in some of the cases of congenital atelectasis. The essential point of difference is, that in the acquired cases the patients are often strong at birth, crying and breathing well, giving no signs of anything wrong in the lungs until the general nutrition has suffered from some other cause. The symptoms come on gradually.

The following is a fairly typical case: A female infant thirteen months old had been under observation in the Nursery and Child's Hospital for several months before death. During this period she suffered a great part of the time from mild bronchitis. The child was extremely rachitic, and the chest showed deep lateral furrows. The respiration was always accelerated, and on inspiration the lateral recession of the chest was at times extreme. There was occasionally seen slight cyanosis, and during the last few weeks it was constant. Death occurred quite suddenly. At autopsy there was found very marked vesicular emphysema of both lungs in front. Nearly the whole of both lower lobes were in a condition of collapse, and of a uniform grayish-purple colour. The posterior portion of the upper lobes was similarly affected, but to a less degree. With moderate force all of the collapsed areas could be completely inflated. Bronchitis was present, but the pleura was normal.

The treatment of these cases is the same as that outlined in the chapter upon Congenital Atelectasis (page 75).

EMPHYSEMA.

Pulmonary emphysema consists primarily in overdistention of the air vesicles. It may result in their rupture and the escape of air into the interlobular connective tissue of the lung. In infancy and childhood emphysema is usually associated with acute processes.

Etiology.—Cases of emphysema are divided into two groups which are due to quite different causes. In one group it is compensatory, and consists

in overdistention of the air vesicles in certain parts of the lungs because the full expansion of other parts is prevented either because they are consolidated, as in pneumonia or tuberculosis, bound down by adhesions from old pleurisy, or subjected to external pressure, as from chest deformities due to Pott's disease or rickets. In these conditions it is probable that the emphysema is produced during inspiration. It may also be produced by the artificial inflation of the lungs of the newly born.

In the second group of cases emphysema is produced by obstructive expiratory dyspnœa or cough. It is seen in all forms of laryngeal stenosis, in acute bronchitis and broncho-pneumonia, in asthma, pertussis, and occasionally it is produced by any condition which requires deep inspiration and holding the breath. A case has been reported to me which occurred in a little boy, who, while playing that he was a steam engine, would hold his breath for a long time and then issue short, forcible expiratory puffs. In bronchitis the obstruction may be caused by swelling of the mucous membrane or by an accumulation of secretion. In this group of cases air enters the lung, but as it can not readily escape, the air vesicles are distended, sometimes to such a degree that their resiliency is almost entirely lost.

Lesions.—The most common form in early life is acute vesicular emphysema, which occurs when the force distending the air cells is only moderate. In this form there is dilatation of the vesicles with very slight structural changes, there being usually rupture of a few alveolar septa only (Fig. 77). Although the dilatation may be quite marked, the emphysema is not permanent. The parts most affected are the upper lobes, particularly the anterior borders. In appearance the emphysematous lung is pale, sometimes almost white. The areas are prominent, and do not collapse upon opening the chest. With a lens, or even with the naked eye, the individual air vesicles can often be distinguished as minute pearly bodies, at times resembling miliary tubercles. When the disease is secondary to acute bronchitis or laryngeal stenosis it may affect nearly the whole of both lungs.

With a greater distending force rupture of many of the air vesicles results, and this may give rise to interstitial or interlobular emphysema. At times blebs are formed, varying in size from a pin's head to a cherry. These are usually seen at the anterior border or at the root of the lung on its inner surface. Again, the air finds its way between the lobules, dissecting them apart in all directions throughout the lung. Sometimes a large part of the surface of both lungs is seamed with irregular deep crevasses containing air, the largest being an inch or more in length and nearly one fourth of an inch wide. The most severe cases occur in pertussis. On two or three occasions I have seen this form of emphysema, once to an extreme degree, where children had died from diseases unconnected with the respiratory tract, and where no history could be obtained

which threw any light upon the etiology of the emphysema. Rupture of the blebs which form at the root of the lung may lead to emphysema of the mediastinum, or even of the subcutaneous connective tissue of the body. This is occasionally seen in whooping-cough and in laryngeal stenosis. The primary or substantive form of emphysema seen in adult life rarely if ever occurs in childhood.

Symptoms.—Emphysema occurring in acute pulmonary diseases gives rise to no peculiar symptoms and to no physical signs except exaggerated resonance upon percussion. If the patients recover from the original disease, the emphysema undoubtedly disappears completely in the course of a few weeks or months. Acute interlobular emphysema can not be diagnosticated during life. The lesion is of such a nature that complete recovery is impossible, although improvement often takes place.

The treatment of emphysema is that of the original disease.

CHAPTER VI.

PLEURISY.

All the common forms of inflammation of the pleura are seen in childhood. In the great majority of cases they are secondary to disease of the lung itself. Serous effusions are much less frequent than in adults, and under three years they are extremely rare. Purulent effusion (empyema) is, however, much more often seen than in adult life, and it is the most important variety of pleurisy with which the physician has to deal.

Whether inflammation of the pleura ever occurs as a strictly primary disease is still a mooted point. Cases are occasionally observed clinically in which both the serous and purulent forms of the disease appear to be primary, but these are extremely rare. Acute pleurisy may, however, follow inflammation of the lung so rapidly that it is not easy to determine that the lung was first affected. In infants, extension from the lung is almost the sole cause. It occurs both with lobar and broncho-pneumonia, existing to some degree in nearly every case in which there is consolidation of the lung. Next in frequency to simple pneumonia as a cause of pleurisy are the tuberculous processes of the lung. Tuberculous pleurisy without tuberculosis of the lungs or the bronchial glands is of doubtful occurrence. Acute pleurisy is not an infrequent complication of the infectious diseases, particularly scarlet and typhoid fevers, measles, and influenza. In most of these cases also it is secondary to disease of the lung. Pleurisy in older children occasionally follows cold and exposure,

although it is doubtful whether in any case this is the only cause. In them also it may occur as a complication of rheumatism.

The most important cause of acute pleurisy being extension from pneumonia, it follows that it is most frequent in the cold season, that it occurs more often in males than in females, and between the ages of one and five years. It may, however, be seen at all ages, and may even occur in intra-uterine life. The youngest case in which I have found extensive pleuritic adhesions as an evidence of previous inflammation was in an infant of three months, who died at the Randall's Island Hospital. In this case firm connective tissue adhesions were found over the whole of both lungs.

DRY PLEURISY.

In infants and young children this usually accompanies pneumonia or tuberculous processes in the lung. In older children it may be primary.

Lesions.—On account of the frequency with which this occurs in pneumonia we have an opportunity of observing it in all stages. In the mildest varieties it affects only the pulmonary pleura, and occurs over the pneumonic areas. The pleura is injected, has lost its lustre, and appears dull or roughened. This is due to an exudation of fibrin upon its surface. If the process continues, more fibrin is poured out, and there are in addition swelling and a proliferation of the connective-tissue cells, and an exudation of leucocytes from the blood-vessels. The pleura is then coated with a layer of fibrin of variable thickness, in which are entangled pus cells and new connective-tissue cells. The layer of fibrin varies from the thickness of tissue paper to that of an ordinary book cover. In recent cases it may easily be stripped off, while in older ones it becomes organized and is firmly adherent. The colour of the exudate varies with the number of pus cells. It is gray, grayish-yellow, or yellowish-green, according as these cells are few or numerous. As a rule, dry pleurisy is localized, but the two opposing surfaces are affected. Part of the exudate is usually absorbed, but it is doubtful if complete recovery occurs, there being left behind some adhesions between the visceral and parietal layers.

In some cases of dry pleurisy there is an excessive exudation of pus cells. These cases are most common in young children, and usually occur with pneumonia, constituting what is known as "pleuro-pneumonia." The process is essentially the same as in the cases just mentioned, yet the gross appearance differs very much from ordinary dry pleurisy. The lesions have already been described under the head of Pleuro-Pneumonia (page 532).

In the dry form of tuberculous pleurisy there may be only an exudation of fibrin, or the pleura may be covered with gray tubercles and yellow tuberculous nodules. These are not only seen upon the pleura, but develop in the exudation. In this form, which is usually chronic, great thickening of the pleura may take place. Both the serous and purulent effusions

occurring in conjunction with tuberculosis are likely to be sacculated because of the previous existence of adhesions.

After nearly every case of dry pleurisy there probably remains some slight thickening of the pleura. In certain cases there follows a chronic inflammation of the pleura with the production of new connective tissue, which results in thickening and adhesions, which may be so extensive as to entirely obliterate the pleural cavity. Either one or both sides may be affected. This form is extremely rare in childhood.

Symptoms.—As an independent clinical disease, acute dry pleurisy has no existence in infancy or early childhood. The cases which are occasionally so diagnosticated have in my experience invariably proven to be broncho-pneumonia. In children from ten to fourteen years old, dry pleurisy may occur under the same conditions as in adults.

The symptoms are sharp, localized pain increased by full inspiration, sometimes tenderness upon pressure, and a short, teasing cough. The pain is not always felt upon the affected side, and it may be referred to the abdomen. Upon physical examination, dry pleurisy is recognised by the presence of a pleuritic friction sound. This is usually of a moist, crackling character, generally localized, and heard both on inspiration and expiration. It is quite superficial, and not changed by coughing. This form of pleurisy, as a rule, runs a course of a few days or a week, without constitutional symptoms. When dry pleurisy occurs as a complication of pneumonia it is recognised by the signs just mentioned; but it usually causes no new symptoms except pain.

Treatment.—The treatment consists in counter-irritation by mustard, iodine, or blisters, according to the severity of the inflammation, and in the use of opium. Severe pain can sometimes be relieved by firmly encircling the chest with a broad band of adhesive plaster.

PLEURISY WITH SEROUS EFFUSION.

This form of pleurisy is infrequent in children, and under three years it is very rare. It may occur as a complication of pneumonia, nephritis, acute rheumatism, scarlet fever, or any of the other acute infectious diseases. It may be tuberculous. In rare cases it appears to be primary. Bacteria are occasionally present in the exudation, even in cases which do not become purulent, but their number is usually small. The pneumococcus, the streptococcus, and the tubercle bacillus are the forms most often seen.

Lesions.—The early changes are much the same as in dry pleurisy, but in addition serum is poured out from the blood-vessels, in some cases almost from the beginning of the inflammation. This may be small in amount, or it may fill the pleural cavity. The lesions are similar to those seen in adults, except that there is apt to be more fibrin in children. The process usually terminates in absorption of the serum, but, as in dry pleurisy,

more or less extensive adhesions are left behind from the fibrinous exudation.

Symptoms.—The small serous effusions of one or two ounces, occurring with the dry pleurisy that complicates pneumonia, rarely cause either symptoms or physical signs by which they can be recognised. In the present connection only those cases will be discussed in which the amount of effusion is considerable. This form of pleurisy sometimes follows a well-defined attack of pneumonia. Other cases come on with acute febrile symptoms somewhat resembling those of pneumonia, but with all the symptoms less severe, except the pain. After an illness of only two or three days the chest may be found full of fluid. In a third class the disease comes on insidiously, with little or no fever, and often with no distinct pulmonary symptoms except shortness of breath. There are general weakness, sometimes loss of flesh, anæmia, and moderate prostration; but usually the patients are not sick enough to go to bed. The symptoms of pleurisy with effusion vary greatly. When it occurs as a complication of some acute infectious disease, it is often latent, and the diagnosis is to be made only by the physical examination of the chest.

The usual course of the disease is for the fluid to disappear gradually by absorption, the case going on to spontaneous recovery. Serious symptoms resulting from pressure upon the heart and lungs are not common, but may occur when the fluid accumulates rapidly; hence they are most likely to be seen early in the attack. There may be great dyspnœa, sometimes orthopnœa, cyanosis, weak pulse, and even attacks of syncope. Death may occur with these symptoms. In certain cases there is seen no tendency to spontaneous absorption, and the exudation may remain stationary for months. There may then be fever, usually slight but sometimes quite regular, with a decline in the general health, pallor and anæmia, which may strongly suggest the existence of pus, although this is not present. Others are regarded as cases of tuberculosis.

Physical Signs.—The signs in the chest are essentially the same whether the fluid is serous or purulent. On inspection, there is diminished movement of the affected side, sometimes bulging of the intercostal spaces, and if the effusion is large, an increase in the measurement of the affected side of the chest. The apex beat of the heart will usually be considerably displaced if the effusion is upon the left side. It may be found at the epigastrium, at the right border of the sternum, or even in the right mammary line. In disease of the right side the displacement is less, and occurs only with a large effusion. It may then be found in or near the left axillary line. On palpation, the vocal fremitus is usually diminished or absent, but it may be but little changed. Percussion gives marked dulness or flatness. In a large effusion this is over the entire lung. There is also a sensation of increased resistance appreciable by the percussing finger. With a smaller effusion there is usually flatness over the lower

part of the chest and dulness or tympanitic resonance above; sometimes dulness is found behind and tympanitic resonance at the apex in front. The line of flatness may change with the position of the patient. The signs on auscultation are variable, and probably lead to more frequent mistakes in diagnosis than in any other pulmonary affection. Bronchial breathing and bronchial voice over the fluid are the rule in children; they are generally more distinct the greater the effusion. Absence of both voice and breathing is sometimes met with, but it is exceptional. The bronchial breathing over fluid usually differs from that over consolidation, in that it is feebler and distant; in some cases, however, it is indistinguishable from that heard over consolidation. Friction sounds may be heard above the level of the fluid, or when the fluid is subsiding, and there may be bronchial râles.

Diagnosis.—The most reliable signs for diagnosis are displacement of the heart, flatness on percussion, absence of râles and friction sounds, and (usually distant) bronchial breathing. In an infant, flatness should always lead one to suspect fluid. If there is flatness over one entire lung, the existence of fluid is almost certain. Between serous and purulent effusions a positive diagnosis is possible only by the use of the exploring needle. This should be employed in every case, as for treatment it is important to know at once whether or not we have a purulent effusion to deal with. The amount of fluid in serous pleurisy is generally less than in the purulent variety.

Pleurisy is further to be differentiated from pneumonia, and from tuberculosis. From pneumonia, the acute cases are distinguished by the lower temperature, the less severe prostration, and the fact that all the general symptoms are milder, but especially by the physical signs. The differential diagnosis by the physical signs between effusion and the various forms of consolidation is considered under the head of Empyema (page 552).

Prognosis.—These cases, as a rule, terminate in recovery, death being very infrequent. In cases coming on without definite cause there should always exist a suspicion of tuberculosis, and hence every patient should be closely watched for the development of the other signs of that disease.

Treatment.—In the great majority of cases, only symptomatic treatment is required during the acute period. The patient should be kept in bed, and pain relieved by opium, counter-irritation, or hot poultices. After the fever has ceased the patient may be allowed to sit up, but all exertion should be carefully avoided if the effusion is large. Sudden death has often occurred when this rule has been violated. The patient should in suitable weather be kept in the open air as much as possible. In the course of a few weeks the effusion usually subsides under simple tonic treatment. Absorption may sometimes be hastened by counter-irritation and diuretics; but convalescence is apt to be slow, and it may be several months before the health is entirely restored.

The removal of the fluid by operation is indicated in the acute stage when it is accumulating so rapidly as to endanger life from the pressure upon the heart and lungs; also when there is no tendency to absorption after from two to three weeks of constitutional treatment. In such cases nothing is to be gained by waiting, and harm may be done to the lung by the delay. The usual method is by aspiration. In the acute stage enough should be removed to relieve the patient's symptoms, aspiration being repeated if necessary in twelve or twenty-four hours. In the sub-acute stage the removal of a portion of the fluid may be all that is required, spontaneous absorption of the remainder often taking place then quite promptly. A few cases of serous pleurisy have been incised and drained as cases of empyema. Scharlau (New York) operated in such a case in an infant two years old. The effusion came on acutely and was excessive, the chest having refilled very quickly after aspiration. The chest was incised and drained and the patient recovered in five days. In chronic cases, in which there are slight fever and a gradual failure of general health, the operation of incision is by some preferred to aspiration.

EMPYEMA.

Fully nine tenths of the cases of empyema in children under five years either occur with or follow pneumonia, being usually the sequel of the form described as pleuro-pneumonia. In some of these cases, however, the pleurisy masks the pneumonia, so that the former appears to be the primary disease. Tuberculosis is a rare cause in early childhood, but becomes more frequent after the seventh year. Empyema may complicate scarlet fever, measles, or any of the other acute infectious diseases. It is met with in pyæmia from all causes. It may occur in the newly born as the result of infection through the umbilical wound or the skin. It is seen with suppurative inflammations of the joints and in osteo-myelitis. It may complicate suppurative processes in the abdomen, such as appendicitis or purulent peritonitis. Among the local causes may be mentioned traumatism, necrosis of a rib, and the rupture into the pleural cavity of abscesses originating in the mediastinum, in the thoracic wall, or below the diaphragm.

Bacteriology.—Much light upon the etiology of empyema has been thrown by the bacteriological investigations of the past few years, especially by the work of Fraenkel, Weichselbaum, Levy, and Netter in Europe, and Prudden and Koplik in this country. Bacteriologically, we may divide the cases into several groups:

1. Those containing the pneumococcus (micrococcus lanceolatus), usually in pure culture. This is the largest group, and includes nearly all the cases secondary to pneumonia. The pleura is usually involved by direct infection from the lung.

2. Those containing other pyogenic germs, particularly the strepto-

coccus pyogenes and the staphylococcus. Of these the streptococcus is the most important. It may be found alone, but is usually associated with the pneumococcus. This combination is likely to be found in cases secondary to the pneumonia which occurs with the infectious diseases. The streptococcus and staphylococcus occur in the pleurisy of pyæmia, and usually also when the disease is due to the rupture of abscesses into the pleural cavity.

3. The cases due to tuberculosis. In this group the presence of the tubercle bacillus is very often difficult to demonstrate, and it may be absent. From this fact the statement is made by Levy that, if no bacteria can be found in a purulent exudate, tuberculosis should always be suspected. It is not, however, safe to conclude that under these circumstances tuberculosis is always present.

Of nineteen successive cases of empyema occurring in my own practice, the pneumococcus was found alone in fourteen; the streptococcus alone in three; the pneumococcus and streptococcus in one; and the staphylococcus alone in one.

Lesions.—This is an inflammation with the production of serum, fibrin, and pus. In most of the cases—and the younger the child the more frequent its occurrence—empyema succeeds the form of pleurisy in which there is first an exudation of fibrin with an excess of pus cells (*vide supra*). As the process continues, more and more pus is poured out, with serum. At first the fluid collects in small pockets formed by the slight adhesions. As it accumulates these are broken down, and the pleural cavity may be filled with pus. If the original inflammation involved but a portion of the pleura the empyema may be sacculated. This is often seen even in infants. Sacculated empyema is usually posterior, but may be in any part of the chest. In very rare cases there may be several sacs containing pus, separated by septa. This I have never seen in empyema following pneumonia. The cases just described are those in which, in infants and young children, the pneumococcus is regularly found. The amount of fibrin is large, covers both surfaces of the pleura, and many large masses float in the fluid. The pus is usually thick, creamy, and odourless. In another group of cases the evidences of inflammation of the pleura are much less marked, and in some they may be slight. There is but little fibrin in the exudate, and adhesions are rare. In this form the streptococcus or the staphylococcus are the organisms usually found. In these cases the inflammation may be purulent from the outset, and the pus is thinner than the preceding variety. It is rare that empyema in a young child results from a serous effusion which has been gradually converted into a purulent one. I can recall but a single instance.

Even when the fluid is moderate in quantity it is not all at the bottom of the chest, but is generally distributed over a considerable part of its surface, and its depth at the middle and upper part of the chest may be

only half an inch, or even less. When the accumulation is larger, the lung does not float on the surface of the fluid, but the fluid surrounds the lung, which is compressed on all sides (Fig. 99). The heart is displaced; the diaphragm and the abdominal viscera are somewhat depressed, and there may be bulging of the chest on the affected side. The amount of fluid in ordinary cases is from half a pint to two pints, although in neglected cases it may accumulate until it amounts to four or five pints. The effect upon the lung will depend upon the amount of fluid and the duration of the compression. When the quantity is small, or when the pressure is removed early, the lung in most cases readily expands, air being forced into it from the opposite lung, especially during the act of coughing. If the pressure is great and has been long continued, the adhesions over the lung may become so dense and firm that expansion is difficult, and can at best be only partial. In such cases recession of the chest wall occurs. In very old cases, expansion is still further interfered with by the changes taking place in the lung itself, usually a low grade of interstitial pneumonia.

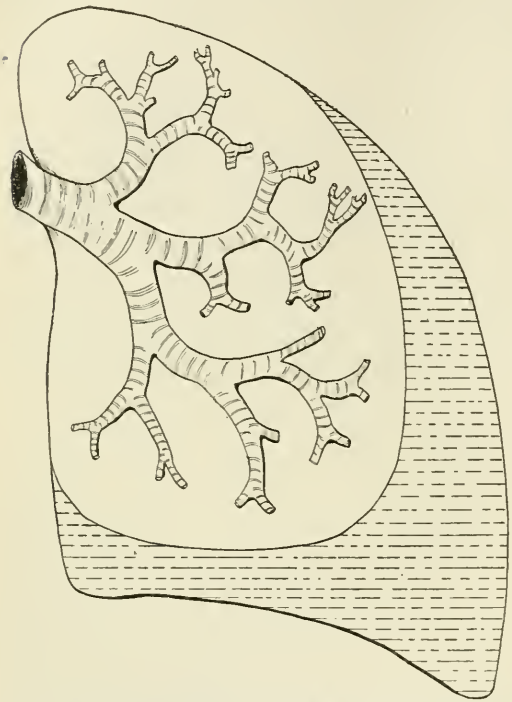


Fig. 99.—Section of a lung to illustrate the distribution of the fluid in the chest in a moderately large effusion (diagrammatic).

In cases of empyema receiving proper surgical treatment reasonably early, full expansion of the lung occurs, and, with the exception of adhesions, recovery may be complete. Although wide in extent, the adhesions are not usually strong enough to interfere seriously with the function of the lung. In cases receiving no treatment, absorption of the pus is possible, but is not to be expected. It generally seeks an external outlet; the lung may be perforated and the pus evacuated through the bronchi, or external rupture may occur, generally in the neighbourhood of the nipple. In still other cases the pus may burrow along the spine, or through the diaphragm may reach the peritonæum.

Empyema is more often of the left than of the right side, the propor-

tion being about three to two. It is double in about three per cent of the cases. The most serious complication in young children is pericarditis, which usually occurs with empyema of the left side. In older children the most frequent complication is pulmonary tuberculosis.

Symptoms.—When it occurs as a sequel of pneumonia, the symptoms of empyema may follow those of the original disease without any intermission; or after the temperature has been normal or nearly so for several days it may rise again, sometimes quite suddenly, but more often gradually. With this accession of fever there are other symptoms pointing to an increase in the thoracic disease. After scarlet fever or other infectious diseases, the onset of empyema is often signalized by cough, rapid breathing, and the other usual symptoms of pulmonary disease. In the cases where empyema appears to be primary, the onset is sudden, with high temperature and general and local symptoms resembling those of pneumonia. After such a beginning, the chest may be found full of pus by the third or fourth day. In rare cases empyema may come on with gradual, and even insidious, symptoms, there being only slight fever, dyspnœa, and cachexia. This is usually seen in older children.

Whatever may have been the mode of onset, when the pus has been in the chest for some time the symptoms are fairly uniform. There are cachexia, pallor, anæmia, and prostration which is generally sufficient to keep the child in bed. The respirations are always accelerated, being usually from forty to seventy a minute. Cough is present; there is dyspnœa, sometimes marked, but more often it is scarcely noticeable. Fever is exceedingly variable; it is rarely high, not often above 102° or 103° F.; in many cases it is not over 100° F., and it may be absent altogether. A typical hectic temperature with sweating, is in my experience very rare. The pulse is rapid but of fair strength. There is loss of flesh, sometimes even emaciation and anorexia; occasionally there is diarrhœa. In chronic cases the general symptoms may closely resemble those of tuberculosis. There may be clubbing of the fingers, albuminuria, and even swelling of the feet.

Diagnosis.—The physical signs do not differ essentially from those present in serous effusions (page 546). Usually the history and the constitutional symptoms enable us to make a diagnosis between serous and purulent effusions with tolerable certainty. If the patient is under three years of age, the fluid is almost certain to be purulent; and from the third to the seventh year, pus is much more often found than serum. In every case in which fluid is suspected the exploring needle should be used, because of the great importance of an early diagnosis. The skin should be washed, the needle sterilized, and the arm raised so as to separate the ribs. Pus may not be found because the needle is too small, too short, or because it is introduced too far into the chest; for when the layer of pus is thin the needle may be pushed through this into the lung. If the physical

signs point to fluid, and if it is not found at the first trial, repeated punctures should be made until the presence or absence of fluid is definitely settled. In some cases eight or ten punctures may be necessary to decide the matter.

Empyema is most frequently confounded with unresolved pneumonia. The mistake of regarding empyema as unresolved pneumonia is much more common than the reverse. The history may be the same in both cases, and the general symptoms may closely resemble each other. The differential points are, that in unresolved pneumonia there is dulness, usually over a single lobe, râles or friction sounds are heard, and there is no displacement of the heart. Empyema gives flatness over the whole lung, or over the lower half of the chest in front and behind, with no râles or friction sounds, and the heart is displaced; and when empyema is sacculated, it is generally, but not always, at the base behind. In both conditions we may get bronchial breathing and voice. The difficulty in differentiating consolidation due to acute pneumonia or tuberculosis from empyema, generally arises from placing too much reliance upon the auscultatory signs. Here also the flatness, displacement of the heart, and the feeble, distant character of the bronchial breathing usually suffice to make clear the diagnosis. In pleuro-pneumonia, with an excessive exudation of fibrin, the signs may be identical with those of empyema, except that the heart is not displaced. I have once seen pulmonary tuberculosis with caseation of an entire lobe which gave signs that were identical with those of a sacculated empyema. It is by the exploring needle, and by that alone, that empyema is positively differentiated from other pulmonary diseases. Other diseases than those of the lung may be confounded with empyema, particularly typhoid fever and malaria; but from these empyema is distinguished by the physical examination of the chest.

Prognosis.—The outcome of a case of empyema depends upon four factors: the cause, the age of the patient, the duration of the symptoms, and the treatment. The best results are obtained in the cases that follow pneumonia. Tuberculosis before the seventh year is an exceedingly infrequent cause, and gangrene of the lung and general pyæmia are both rare causes in early life. The three etiological factors last mentioned are those which make the prognosis of the disease in adults so serious. I can recall but two deaths in children over two years old which were due to empyema. In one case operation was refused, and in the other death was due to multiple abscesses of the lung. The mortality in hospital cases in infants under one year is high—fully 50 per cent—not only because of the tender age, but because of the wretched general condition of the patients. Empyema in older children, seen reasonably early—i. e., within six or eight weeks—and receiving proper treatment, almost invariably terminates in recovery, unless the disease is double or complications exist. The longer operation is delayed the worse the prognosis, because the more

difficult the expansion of the lung, the more tedious the disease, and the greater the likelihood of a sinus remaining. With proper early treatment these patients not only recover, but they recover perfectly, and in most cases rapidly. Retraction of the chest and its resulting lateral curvature of the spine are extremely rare, and seen only in neglected cases. In the great majority of the cases I have seen, in which a reasonably early operation was done, it was impossible, after the lapse of one or two years, to detect any difference whatever in the physical signs of the two sides of the chest. There is no serious disease the treatment of which is usually more satisfactory than that of acute empyema in a young child.

Spontaneous recovery in empyema may take place by absorption; but this is so rare that it is never to be expected, although there is conclusive evidence that it is possible. The pus may be evacuated spontaneously through a bronchus, rupture having taken place through the visceral pleura. When this occurs, a large amount of pus may be coughed up in a few hours, usually followed by immediate, but not always lasting, improvement. This is the most favourable of the natural terminations. External opening may take place, usually about the nipple. There is an area of redness, then a fluctuating tumour, and finally the pointing of an abscess. The discharge may continue for months, or even for years. External opening rarely occurs until the disease has lasted several months. Of 19 cases of empyema in children collected by Schmidt, in which a spontaneous discharge of pus occurred either externally or through a bronchus, there were 17 deaths and 2 recoveries. Empyema may burrow behind the diaphragm into the abdominal cavity, appearing as a psoas abscess; it may burrow posteriorly into the lumbar region; it may rupture into the œsophagus, or through the diaphragm into the peritoneal cavity. All these conditions, however, are very rare. The chances of spontaneous cure in empyema are small. Of 32 cases, reported by Rilliet and Barthez, which received no surgical treatment, 21 proved fatal. The statistics of empyema before the general adoption of surgical treatment are simply appalling. Patients were either worn out by the protracted suppuration, or died from amyloid degeneration, pneumonia, or tuberculosis.

Treatment.—The medical treatment relates to the patient only; the disease is always to be treated surgically. Like any other acute abscess, empyema requires free incision and drainage with proper antiseptic precautions.

Aspiration as a means of cure has been almost entirely given up in New York. Unquestionably it sometimes suffices to cure empyema, most frequently when it is localized. How often this occurs is shown by the following statistics: Of 139 cases which I collected that were treated by aspiration, 25 were cured, 8 of these by a single aspiration; 13 died, and the remaining 101 were afterward subjected to other treatment. The objections to aspiration are: That it is not possible to remove all the pus; that it

affords no opportunity for the removal of the fibrinous masses usually present in large quantities in the exudate; and, finally, that it is only a possible means of cure. The terror caused by repeated aspirations is almost as great as that of incision without anæsthesia. In this way valuable time is lost, the disease is unduly prolonged, and the chances of success by subsequent incision are greatly diminished. Aspiration, therefore, is to be advised only for temporary relief when the amount of fluid is large and the symptoms are urgent. Enough pus may thus be removed to relieve the immediate symptoms, incision being deferred for a day or two. Even under these conditions its advantages over a primary incision are open to question.

Puncture with a trocar and canula was formerly much practised, but it has almost entirely passed into disuse.

Simple incision and drainage.—Incision is usually advisable as soon as the diagnosis is made. There is no advantage in delay, provided the patient's general condition be such as to stand the slight shock of the operation. The dangers attendant upon general anæsthesia are so great that it is better not to employ it at all. I have known four deaths to occur on the table during the operation, and in several other cases have seen very dangerous symptoms from general anæsthesia. Chloroform is more to be feared than ether. We should, then, rely upon the local anæsthesia obtained by a spray of chloride of ethyl or ether, or, better still, by cocaine. The most favourable point for incision is the posterior axillary line in the seventh intercostal space upon the right side, the eighth upon the left. In a case of a localized empyema, the lowest point at which pus can be obtained by puncture should be chosen. The incision is made in the middle of the intercostal space. No matter what has been found by puncture on previous occasions, the exploring needle should always be used at the time of operation and at the site of the incision before the latter is made. The cutaneous incision should be an inch and a half long, and the opening in the pleura made large enough to allow the little finger of the operator to pass into the pleural cavity. The hæmorrhage is very rarely sufficient to require a ligature. Masses of fibrin presenting at the opening should be removed with forceps. The wound may be held open by forceps or a tracheal dilator, and as much of the fibrin as possible removed at the time; or, if the patient's condition is bad, the tube may be immediately inserted and the dressings applied. The drainage tube should be of heavy rubber, fenestrated, three eighths or half an inch in diameter and four or five inches long. It is passed into the deepest pocket of the empyema. To secure it from slipping into the cavity, its outer end should be transfixed by a large safety-pin before its introduction. It is often advisable to insert two tubes side by side. This diminishes the danger of stopping the discharge by the plugging of the tube with fibrin. Iodoform gauze is placed over the wound beneath the safety-pin, and a

compress of the same over the opening of the tube, the dressing being completed by a large mass of absorbent cotton and a snug roller bandage. The pus now slowly escapes into the dressing as the lung expands. The pad of gauze placed over the end of the tube acts as a valve, preventing air from entering the chest, although permitting pus to escape as the lung is expanded by inspiration or by the act of coughing. When there is no reason for haste during the operation, a larger part of the pus may be removed before the application of the dressing. This should be allowed to escape slowly, the opening being closed from time to time by a compress. From ten to twenty minutes should be consumed in evacuating the pus.

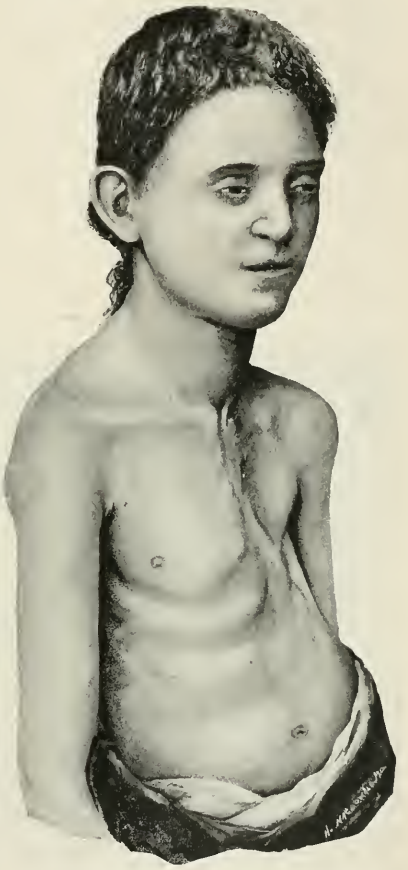


Fig. 100.—Deformity after an old empyema of the left side for which Estlander's operation was performed. Portions of five ribs were removed. (From a photograph seven years after operation.)

For the first two days the dressings should be changed twice daily, then once a day for ten days or two weeks, and later at longer intervals. The tube is gradually shortened at each dressing, until, at the end of a week or ten days, it is reduced to the length of two inches. After the fourth or fifth day a smaller tube may be substituted. Usually by the end of the third week, and often by the end of the second, the tube may be dispensed with altogether, the tract being kept open by the introduction of a narrow strip of iodoform gauze. The time of redressing and the removal of the tube is determined by the amount of discharge and by the temperature. While this does not usually rise after the second day, unless the drainage is imperfect, it may do so when the lung does not expand properly, or when there is still active disease in the lung itself, as is not very uncommon in the cases coming on most acutely. The drainage tube is very liable to be blocked by masses of fibrin, even when one of large size is used. This is the first thing to be suspected if the temperature rises. At each dressing it is well to remove the tube to see if it is clear. The mistake is often made of allowing the

drainage tube to remain too long a time, so that a sinus is kept open which would otherwise heal. Another is that of allowing a very large tube to remain for a long time; this may cause erosion of the periosteum and even necrosis of a rib. Washing out the pleural cavity is indicated only in cases in which the pus is in a putrid condition. A single washing for the purpose of removing fibrin is the routine practice of some surgeons. For this a warm sterilized salt solution should be used. Personally I have not found this necessary. Repeated irrigations should on no account be employed. The usual duration of the discharge in cases treated by simple incision is from three to six weeks, the average being about five weeks. The earlier the operation the shorter the course, because of the facility with which the lung expands.

Resection of a rib.—Many of the best surgeons favour this as a routine procedure, with the belief that with the larger opening which is thus made, more perfect drainage is secured, that masses of fibrin can be removed with greater facility, and that it is altogether a more certain and efficient means of treatment than is a simple incision. While admitting some of the advantages claimed, my own experience has been that in the great majority of recent cases in young children simple incision with drainage is all that is required. Rib resection is necessary if there is overlapping of the ribs, or if the intercostal spaces are so narrow as not to allow the introduction of a good-sized drainage tube. These are usually the cases in which the disease has lasted much longer than the average time. One inch of rib is all that it is necessary to remove. The periosteum is preserved, and there is rarely any permanent deformity.

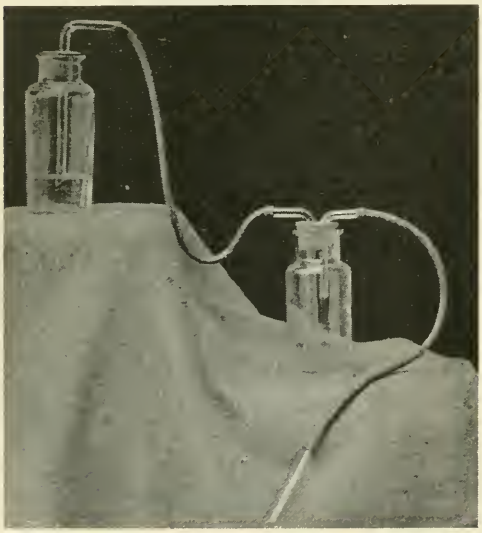


Fig. 101.—James' apparatus for expanding the lung after empyema.

In chronic cases, or those which have been long neglected, some further operative treatment is often necessary. Some of these are cases which have opened spontaneously and discharged for many months before coming under observation. The lung is so bound down by firm adhesions that further expansion is impossible, and even after the chest has receded to its utmost, so that the ribs are in contact, there still remains a cavity

which can not close. For such cases the only hope is in an operation by which portions of several ribs are removed, thus allowing a greater collapse of the chest. This is known as thoracoplasty, or Estlander's operation. The operation is of itself a serious one, and only to be advised as a last resort in inveterate cases. By it, however, life may be saved in some that are otherwise hopeless. Such an operation is, of course, always followed by very great deformity (Fig. 100).

Methods of inducing expansion of the lung.—In most of the cases, particularly the recent ones, complete expansion of the lung takes place without any difficulty, the chief agent being the cough. In some cases this may be insufficient. The apparatus shown in the accompanying cut (Fig. 101), devised by James (New York), serves at the same time as a toy for the child's amusement and as a most efficient means of inducing forced expiration. One bottle is placed a few inches higher than the other, and the child blows a coloured fluid from the lower into the higher bottle, allowing it to siphon back. By raising the second bottle, a greater expiratory force is required. This may be regulated at will. The apparatus may be used for a few minutes several times a day, and particularly in cases of long standing it is of great assistance in producing pulmonary expansion. Blowing soap bubbles often answers the same purpose.

SECTION V.

DISEASES OF THE CIRCULATORY SYSTEM.

CHAPTER I.

PECULIARITIES OF THE HEART AND CIRCULATION IN EARLY LIFE.

The Fœtal Circulation.—During the latter part of fœtal life the circulation may be briefly described as follows: The purified blood comes from the placenta through the umbilical vein. Entering the body, it divides at the under surface of the liver into two branches, the smaller one, the ductus venosus, communicating directly with the inferior vena cava; the larger branch joining the portal vein, so that its blood traverses the liver, and then enters the inferior vena cava through the hepatic vein. From the inferior vena cava the blood enters the right auricle, like that returned from the head and upper extremities by the superior vena cava. A part of the blood now passes directly into the left auricle through the foramen ovale; the remainder, through the tricuspid orifice into the right ventricle. As the requirements of the pulmonary circulation are not great, only a small part of the blood is sent through the pulmonary artery to the lungs; the greater portion passes from the pulmonary artery through the ductus arteriosus into the aorta, joining here the blood from the left ventricle. The blood thus finds its way from the right heart to the left, only in small part by way of the lungs, the greater part passing directly from the right auricle to the left, or from the right ventricle into the aorta through the ductus arteriosus. From the aorta, the blood reaches the placenta through the umbilical arteries, which are a continuation of the hypogastric arteries, which in turn are given off from the internal iliacs.

Changes in the Circulation at Birth.—With the ligature of the umbilical cord, the circulation through the umbilical vein and arteries and the ductus venosus ceases. With the establishment of respiration and the consequent increased demands made by the pulmonary circulation, the blood ceases almost at once to pass through the ductus arteriosus, and very soon through the foramen ovale. The umbilical vessels during the first few days of life are filled with small thrombi, which become organized. By the end of the first week, these vessels, as well as the ductus venosus, are usually closed at their extremities, although they may remain patulous throughout the greater part of their extent for several weeks. They subsequently atrophy to the condition of small fibrous cords. For some weeks

before birth the circulation through the foramen ovale is slight, it being gradually obstructed by the growth of a septum which nearly fills the space at birth. After the first week of extra-uterine life very little, if any, blood passes through it, although complete closure of the foramen often does not take place until the middle of the first year. In fully one fourth of the autopsies I have made upon infants under six months old, there have been found minute openings at the margin of the foramen ovale, but they are usually oblique, and closed by the valvular curtain so as effectually to obstruct the current of blood. The ductus arteriosus is first closed by a clot, which becomes organized and blends with the products of a proliferating arteritis. It is rarely found open after the tenth day, and by the twentieth it is almost invariably obliterated.

The Pulse.—The pulse in early life is not only more frequent, but it is very much more variable than in adults. The following is the average pulse-rate in healthy children during sleep or perfect quiet:

Six to twelve months	105 to 115 per minute.
Two to six years	90 " 105 " "
Seven to ten years	80 " 90 " "
Eleven to fourteen years	75 " 85 " "

The pulse is a little more frequent in females than in males, and more frequent when sitting than when lying down. Muscular exercise or excitement increases the pulse-rate by from twenty to fifty beats. Very trivial causes disturb not only the frequency but the force of the pulse. The pulse in young infants may be irregular even in health and during sleep. When rapid, it is frequently irregular without any meaning. No dicrotism is seen in the pulse wave of early infancy, according to Blanche.*

The circulation is much more active in infancy than in later childhood; thus, according to Vierordt, the entire round of the circulation is accomplished in the newly born in twelve seconds; at three years, in fifteen seconds: in the adult, in twenty-two seconds.

Size and Growth.—The relative size of the heart is slightly greater in infancy than in later life, it being smallest at about the seventh year. The average weight at the different periods of life is as follows:†

Age.	Ounces.	Grammes.	Ratio to body weight.
Birth	0·50	14	
1 year	1·25	35	1 to 225
2 years	1·87	53	
3 "	2·25	64	
7 "	2·80	80	1 to 280
14 "	5·84	166	1 to 222
Adult	8·50	241	1 to 226

* See tracings in Archives of Pædiatrics, vol. v, p. 732.

† The figures in infancy are from one hundred and fifty-five observations made in the New York Infant Asylum; the others are taken from Sahli.

The growth of the heart is rapid during the first three years, and nearly proportionate to that of the body. It is slowest from the third to the tenth year, and most rapid from the eleventh to the fifteenth year. At birth, the thickness of the right ventricle is very nearly the same as that of the left, the ratio being 6 : 7. The left ventricle, however, grows very much more rapidly than the right, so that at the end of the second year the ratio is 1 : 2, which is nearly that of the rest of childhood.

Position of the Apex Beat.—In the infant the heart is placed somewhat higher, and occupies a position a little nearer the horizontal than in the adult. This is partly due to the higher position of the diaphragm. The apex beat is therefore higher and farther to the left than in adult life. According to the observations of Wassilewski and Starck, whose combined examinations with reference to this point were made upon over 2,100 children, the apex beat is, as a rule, outside the mammary line until the fourth year; if it is less than one third of an inch beyond the nipple, it can not be considered abnormal. From the fourth to the ninth year, the apex beat is in or near the mammary line. After the thirteenth year, under normal conditions, it is invariably within that line. During the first year the apex beat is usually found in the fourth intercostal space; from the first to the seventh year, it is found with about equal frequency in the fourth and the fifth spaces; after the seventh it is usually, and after the thirteenth year it is always, when normal, in the fifth space. The position of the apex beat may be considerably modified by severe deformities of the chest resulting from rickets, Pott's disease, or lateral curvature of the spine.

Examination of the Heart.—*Inspection.*—Bulging of the præcordia is a frequent and important sign of cardiac disease during childhood. The cardiac impulse is generally weaker than in the adult, and often it is difficult to locate the apex beat owing to the thick layer of adipose tissue covering the chest.

Palpation.—This is usually a much more satisfactory method than is inspection for determining the position of the apex beat. For this purpose the child should be in the sitting posture, with the body inclined slightly forward. Great displacement of the apex beat is always significant, and should lead one to suspect pleuritic effusion; lesser degrees of displacement to the left indicate hypertrophy, especially of the left ventricle; to the right, hypertrophy of the right ventricle, usually with a congenital malformation.

Percussion.—This is best done by means of the percussion hammer. A light blow should be used, on account of the thinness and elasticity of the chest walls. The outline of the area of "relative cardiac dulness," especially in small children, is proportionately larger than in the adult. This may lead to the mistaken opinion that the heart is enlarged, when it

is really of normal size. According to Sahli,* the limits of this area are as follows: Above, the second space or lower border of the second costal cartilage; to the right, at the para-sternal line, sometimes slightly beyond it; to the left, at or slightly beyond the mammary line, this depending upon the age of the child. The lower border is indeterminable on account of the liver.

The area of "absolute cardiac dulness," or that part of the heart uncovered by the lung, resembles in shape the same area in the adult, but it is relatively larger. Its upper limit is the upper border of the third intercostal space, sometimes the third costal cartilage; it extends to the left to a point between the para-sternal and the mammary lines, and to the right as far as the left border of the sternum. These two areas will be readily understood by reference to the accompanying diagram (Fig. 102).

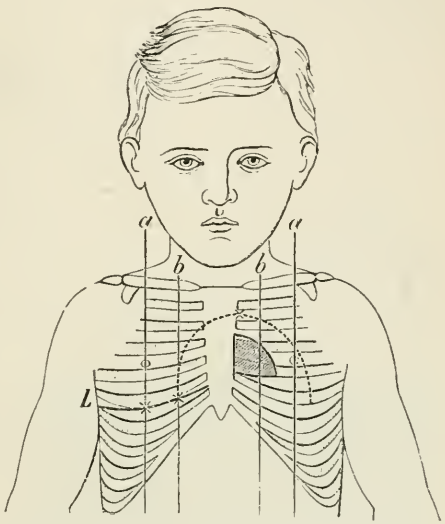


Fig. 102.—Showing areas of cardiac dulness: *a* is the mammary line; *b*, the para-sternal line; *L*, the upper border of the liver. The space enclosed by the dotted line represents the area of relative dulness; the heavily shaded area, that of absolute dulness. (After Sahli, slightly modified by Unger.)

Auscultation.—This is of little value unless the child is quiet. The preferable position is the sitting posture. For an accurate diagnosis the stethoscope is indispensable, but auscultation should always be practised with the naked ear as well. The rhythm and rapidity of the child's heart action are much more easily disturbed than are the adult's, and such disturbances are consequently much less significant. The rapidity of the heart in infancy is ordinarily so great as to make it practically impossible to distinguish between diastolic and presystolic murmurs. Normally, the loudest sound is the first sound at the apex; the weakest sound is the second sound at the aortic orifice. According to Hochsinger, the accentuation of the child's heart-sounds is upon the first sound, and not upon the second, as in the adult.

In consequence of the small size and the thin walls of the chest, all sounds, both normal and pathological, appear relatively louder than in the adult, and the area of diffusion is therefore much greater. Thus it is a frequent occurrence for murmurs to be heard all over the chest both in front and behind.

* Topographische Percussion im Kindesalter, 1882.

Reduplication of the heart sounds, in consequence of the valves of the two sides not closing exactly together, is not uncommon in children, and may be due simply to excitement. During the first four years of life nearly all the abnormal murmurs heard are systolic.

Accidental murmurs may be due to anæmia and other blood conditions, and, although not so common as in older patients, they are by no means rare even in infants.

CHAPTER II.

CONGENITAL ANOMALIES OF THE HEART.

Etiology.—The causes of congenital anomalies of the heart may be grouped under three general heads:

1. Malformations resulting from imperfect development of certain parts of the heart, most frequently one of the septa. Either the ventricular or the auricular septum may be affected, or that dividing the pulmonary artery from the aorta. Such failure in development perpetuates conditions which are normal in the early months of fœtal life. There may also be atresia of any one of the orifices, absence of one or more of the valvular leaflets, or of any one of the large vessels.

2. Fœtal endocarditis. The effects of this condition vary according to the time of its occurrence. It is almost invariably of the right side, most frequently affecting the pulmonic valves. Valvular disease in fœtal life leads not only to hypertrophy and dilatation, but also interferes with the normal development of the heart by preventing the closure of the auricular or ventricular septum or the ductus arteriosus, these being kept open by way of compensation.

3. Persistence of fœtal conditions, such as the foramen ovale or ductus arteriosus. This may be the result of valvular disease, as previously stated, or of some condition of the lungs, such as atelectasis.

Lesions.—In the following table are given the lesions found in two hundred and forty-two cases, which I have collected from medical literature:

Frequency of the different lesions in 242 autopsies upon cases of congenital cardiac anomaly.

Defect in the ventricular septum	149	cases;	the only lesion in	5	cases.
Defect in the auricular septum or patent foramen ovale	126	"	" "	9	"
Pulmonic stenosis or atresia	108	"	" "	6	"
Patent ductus arteriosus	68	"	" "	3	"

Abnormalities in the origin of the great vessels.	45	cases;	the	only	lesion in	0 cases.
Pulmonic insufficiency	17	"	"	"	0	"
Tricuspid insufficiency	6	"	"	"	0	"
Tricuspid stenosis or atresia	3	"	"	"	0	"
Mitral insufficiency	1	"	"	"	0	"
Mitral stenosis or atresia	6	"	"	"	0	"
Aortic insufficiency	1	"	"	"	0	"
Aortic stenosis or atresia	6	"	"	"	0	"
Transposition of the heart	2	"	"	"	0	"
Ectocardia	1	"	"	"	0	"

The most frequent associated lesions.

Pulmonic stenosis, with defect in the ventricular septum	92	cases;	the	only	lesion in	20 cases.
Pulmonic stenosis, with defect in the auricular septum	52	"	"	"	8	"
Defects in both septa	82	"	"	"	17	"
Pulmonic stenosis and defects in both septa	36	"	"	"	21	"

From this table it will be seen that, in the great majority of cases, several lesions are present, the most frequent combinations being pulmonary stenosis with defective ventricular septum, pulmonic stenosis with defective auricular septum, the three lesions associated, or the first two with a patent ductus arteriosus.

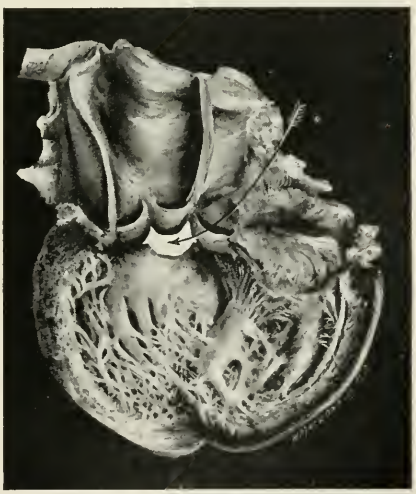


Fig. 103.—Congenital cardiac disease. The left ventricle is shown with a defect in the ventricular septum, the opening being just beneath the aortic valve. (From a patient dying in the Babies' Hospital.)

Defect in the ventricular septum.—This is the most frequent lesion in congenital cardiac disease, and in half the cases was associated with pulmonic stenosis. The defect is generally at the upper part of the septum (Fig. 103). It is usually from one fourth to one half an inch in diameter, but not infrequently there is a large defect, and the septum may be entirely absent, the heart then consisting of but three cavities—two auricles and one ventricle. If the auricular septum also is wanting, as is often the case, the heart has but two cavities. Frequently there are also abnormalities in the origin of the great vessels. The pulmonary artery and the aorta may be given off from the common ventricle, or the aorta may arise partly from one ventricle and partly from the other. If pulmonic stenosis or atresia is present, the opening in the

ventricular septum is conservative, affording a channel for the passage of blood from the right to the left side of the heart.

Patent foramen ovale, or defect in the auricular septum.—Although this is one of the most common congenital malformations, it is not one of the most important. It rarely occurs alone, but is frequently found with pulmonic stenosis or a defect in the ventricular septum. Small oblique openings in the auricular septum—usually at the foramen ovale—are not infrequently met with in autopsies upon young infants, but they are of no importance. In pathological conditions the opening is from one fourth to one inch in diameter, and there may be more than one opening. A defect in this septum is frequently secondary to pulmonic stenosis, or it may be a failure in development. A patent foramen ovale may be due to atelectasis.

Patent ductus arteriosus.—As a solitary lesion this is rare, but it is frequently associated with pulmonic stenosis, usually with a defect in one or both septa. It is then one of the channels by which the blood may find its way to the lungs when the pulmonary orifice is obstructed. It is not a malformation, but simply the persistence of a fœtal condition usually necessitated by other changes in the heart.

Pulmonic stenosis.—This is one of the most frequent and most important lesions. It may be due to fœtal endocarditis, or to a malformation. If the former, there is usually stenosis; if the latter, there may be atresia. It is often a primary lesion, and when marked it is always accompanied by other changes, most frequently by a defect in one or both septa or by a patent ductus arteriosus. This is important, as being more constantly associated with cyanosis than is any other congenital lesion. The amount of obstruction varies from a slight narrowing of the orifice to complete atresia. If there is atresia, the pulmonary artery is very small, and may be rudimentary.

Pulmonic insufficiency.—This lesion is relatively rare. It is usually the result of fœtal endocarditis, but there may be absence of the pulmonary valve. It is most frequently associated with a defect in the ventricular septum.

Tricuspid, mitral, and aortic disease are all very infrequent and usually seen in cases with multiple defects. Atresia or stenosis is much more common than insufficiency.

Abnormalities in the origin of the large vessels.—These are quite frequent; but, as will be seen from the table, they are always associated with other lesions. Three forms are seen: (1) Transposition of the large vessels—the pulmonary artery is given off from the left, and the aorta from the right ventricle. (2) Both arteries arise from a common trunk. This is usually due to an incomplete development of the lower part of the septum dividing the two arteries. Usually the pulmonary artery appears to be a branch of the aorta. This condition is frequently associated with

other abnormalities, often with so large a defect in the ventricular septum that there is really but one ventricle. (3) The aorta has an abnormal origin, arising from the right ventricle, or partly from both ventricles. This also is associated with a large defect in the ventricular septum. When described as arising from both ventricles, the aorta is usually given off directly above the line of the septum.

In addition to these main deformities, there are many others which need not be more than mentioned. An abnormality in the number of valvular segments is quite a frequent occurrence, but does not usually impair the valve's function. In rare cases a valve is rudimentary, and it may be entirely absent, generally at the pulmonic or tricuspid orifice. Absence of the right auricle and absence of the pericardium have been recorded; also opening of the pulmonary veins into the right auricle, and a single pulmonary artery. In one case in the series there was ectocardia, this being associated with a congenital fissure of the sternum.

Transposition of the heart, or true dextro-cardia, was recorded but twice in this series of cases. It was, however, simulated in several others, including one of my own, where the apex beat was to the right of the sternum. There was in this case great hypertrophy of the right ventricle with a rudimentary ventricular septum.

Secondary lesions.—Since the one condition which nearly all of the congenital malformations of the heart have in common is a persistence of one or more of the fœtal conditions in which the right ventricle does most of the work, it is usually found hypertrophied. It is in most cases accompanied by some dilatation, and often there is dilatation of the right auricle. Changes in the wall of the left heart alone are exceedingly rare. In four cases there was evidence of malignant endocarditis, which was the cause of death, all but one of these patients being adults.

Symptoms.—The symptoms of congenital cardiac disease are usually manifested soon after birth, although this is not always the case. Of 128 cases in which the time of the first symptoms was noted, they were congenital, or appeared during the first month, in 85; after one month and during the first year, in 18; from one to sixteen years, in 15; while in 10 no symptoms were observed until after puberty. Congenital cardiac disease is one of the causes, but not a frequent one, of death during the first few days of life. This may be directly due to convulsions, asphyxia, or syncope.

The most striking objective symptom is cyanosis. This was noted in 88 per cent of the cases in which histories were given. Congenital cardiac disease is very apt to be overlooked when cyanosis is absent, as it may be even with very serious lesions. Cyanosis may be slight and noticed only upon exertion, as upon coughing or crying, or it may be intense and constant, giving the skin a dark, leaden colour, and the mucous membrane of the mouth a raspberry hue. The view that cyanosis depends upon an

admixture of arterial and venous blood is generally discredited. In the great majority of the cases at least, the explanation is a deficient oxidation of the blood in the lungs, owing to some interference with the pulmonary circulation. In 63 per cent of the cases of cyanosis in the series, there was found pulmonic stenosis or atresia, or a small pulmonary artery. Cyanosis is of much value in diagnosis, as it is rarely seen in acquired cardiac disease. The degree of cyanosis and its constancy are of some importance in determining the gravity of the lesion, although these alone are not to be depended upon. Another frequent symptom is the enlargement of the terminal phalanges known as clubbing of the fingers (Fig. 104) and toes. This enlargement, which usually involves all the phalanges, is probably due to venous obstruction. Occasionally there are seen dyspnœa, œdema of the lower extremities, dropsy of the serous cavities, and hæmorrhages, particularly hæmoptysis and epistaxis.

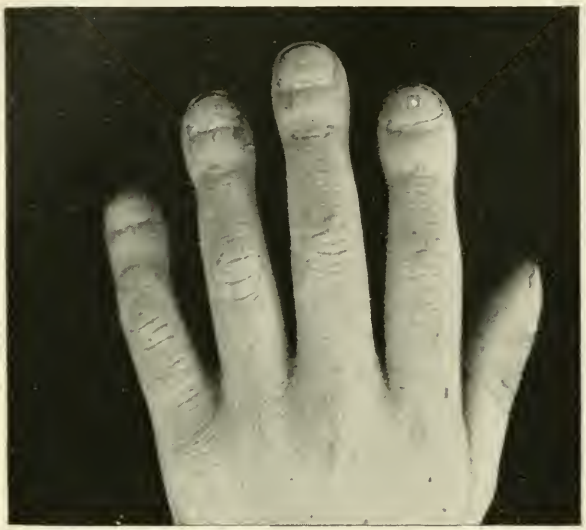


Fig. 104.—Clubbing of the fingers in congenital heart disease. (From a boy five years old.)

Diagnosis.—The most diagnostic features of congenital cardiac disease are cyanosis, the presence of cardiac murmurs, and signs of enlargement of the right heart.

Murmurs were present in four fifths of the cases in which histories were given. The most characteristic is a systolic murmur, loudest at the left base and diffused over a large area. A systolic murmur only was heard in 60 cases, a double murmur in 11, a diastolic and a presystolic in one case each. A systolic murmur may be due to pulmonic stenosis, deficient ventricular septum, patent ductus arteriosus, mitral regurgitation, tricuspid regurgitation, or aortic stenosis. Since these conditions are very often associated, it is difficult to tell upon which one the murmur depends. In over two thirds of the cases in which the murmur was localized it was at the base of the heart, and in the great majority of these it was loudest at the left base, in the second or third space at the border of the sternum and transmitted toward the left shoulder. Apex murmurs were heard in

but one fourth of the cases. The murmurs are usually loud, rough, and often out of proportion to the other signs present. Frequently they may be heard all over the chest, both in front and behind. In a young child, a very loud murmur with cyanosis is almost diagnostic of congenital disease, since in acquired disease loud murmurs are nearly always at the apex, and are accompanied by marked hypertrophy.

Enlargement of the right heart, chiefly from ventricular hypertrophy, was present in 86·5 per cent of the cases. In about one half of these there was hypertrophy of the left ventricle, but this was rarely seen alone. The signs of hypertrophy of the right ventricle are: dulness extending to the right of the sternum, displacement of the apex beat to the right, epigastric pulsation, and sometimes bulging of the lower portion of the sternum.

A diagnosis of the precise nature of the malformation is very difficult, and in the great majority of cases only a probable diagnosis is possible. Nearly all the cases are complex, and the variety of combinations is very great. A study of the histories and autopsies of the cases in this series reveals many apparently contradictory facts. Loud murmurs are sometimes heard which are difficult to explain by the lesions, and murmurs may be absent where there is every reason for expecting their presence, as in a case recently under my observation. Certain lesions like aortic stenosis, mitral stenosis, and mitral regurgitation may be accompanied by the same signs as in acquired disease. With reference to the other conditions, I can not do better than give the more frequent clinical symptoms with the results of the autopsies in the series of cases which I have collected.

A systolic murmur at the base, with cyanosis.—This is the most common combination met with, and was present in about one third of all the cases. In over 80 per cent of the cases with these symptoms, pulmonic stenosis was found. The remainder were complicated cases of quite a wide variety. Pulmonic stenosis was usually associated with a defect in one of the cardiac septa, or a patent ductus arteriosus.

A systolic murmur without cyanosis.—In the cases followed to autopsy this was not a frequent combination, being noted but six times, and usually dependent upon a defect in the ventricular septum without pulmonic stenosis, or upon tricuspid regurgitation. Judging from my own clinical experience, a systolic murmur without cyanosis is more common than is indicated by these figures.

A systolic murmur at the apex with cyanosis.—Of the six cases with this combination, all were examples of complex malformation, the most frequent lesions being a defect in the auricular septum, transposition of the great vessels, and patent ductus arteriosus.

Cyanosis without murmurs was noted fourteen times. It indicates either pulmonic atresia or the transposition or irregular origin of the great vessels.

Diastolic murmurs were heard in two cases, and depended upon pulmonic insufficiency.

A presystolic murmur was noted in a single case. It was localized at the right base, and the only lesion was a patent foramen ovale.

Absence of both cyanosis and murmurs was recorded in five cases. The lesions found were: atresia of the aorta, both arteries arising from the right ventricle, or defective septa.

It will be seen that about the only cases in which a fairly positive diagnosis can be made are those of pulmonic stenosis with a deficient ventricular septum. Enlargement of the right heart, being common to nearly all the varieties, is of no diagnostic value.

Diagnosis of congenital from acquired disease.—Congenital disease may be suspected if the patient is under two years of age; if there is no history of previous rheumatism; if the murmur is atypical in its location, character, or transmission; if there is a very loud murmur at the base; if there is cyanosis; and if there is evidence of enlargement of the right heart.

Diagnosis of congenital from anæmic murmurs.—This is often a more difficult matter than to decide between congenital and acquired disease. From a murmur alone one should be very cautious in making a diagnosis of cardiac malformation in a very anæmic infant. Anæmic murmurs are systolic, basic, unaccompanied by enlargement of the heart; usually heard in the carotids, often in the subclavian arteries, but are seldom so loud as those due to malformations. In some cases it may be necessary to watch the effect of treatment or the course of the disease before deciding the question.

Prognosis.—Of 225 cases, 60 per cent were fatal before the end of the fifth year, and nearly one half of these during the first two months; while 16 per cent of the cases lived over sixteen years, and 8 per cent over thirty years. The prognosis in any given case is to be made from the general condition of the patient and how well the circulation is carried on, rather than from the intensity of the cyanosis or the character of the murmur, although extreme cyanosis is always unfavourable.

In the cases fatal soon after birth the usual lesions are large defects in the septa, transposition of the great vessels, or pulmonic atresia. In five of twenty-three cases dying thus early, the heart had but two cavities. Lesions which are compatible with the longest life are minor septum defects, and pulmonic stenosis which can be compensated for by hypertrophy of the right ventricle. Many exceptional instances are recorded in which patients have lived a long time in spite of extreme deformities. One child with transposition of the pulmonary artery and aorta lived two and a half years. Tiedmann's case lived eleven years with a heart consisting of three cavities —two auricles and one ventricle—and with constant cyanosis. In three cases reported by Rokitansky, the patients lived over forty years with rudi-

mentary auricular septa and no cyanosis mentioned. Gelpke's case had cyanosis, and lived twenty-seven years with rudimentary auricular and ventricular septa, and with no tricuspid opening.

Treatment.—No treatment is of the slightest avail in diminishing the amount of deformity or promoting the closure of any of the abnormal openings. All cases are to be treated symptomatically.

CHAPTER III.

PERICARDITIS.

Inflammation of the pericardium is a rare disease in infancy and early childhood, only two cases being seen in seven hundred and twenty-six consecutive autopsies at the New York Infant Asylum. In later childhood the disease is more frequent. In its etiology, symptoms, and course it resembles quite closely the same disease in adults.

Etiology.—Of 69 cases of pericarditis in children under fourteen years of age, 24 occurred before the third year, 12 between the third and seventh years, and 33 between the seventh and fourteenth years. It has been seen in the newly born, and has been found even in the fœtus.

Pericarditis is almost invariably a secondary disease, following (1) pleurisy or pleuro-pneumonia; (2) acute rheumatism; (3) acute infectious diseases, especially scarlet fever; (4) pyæmia; (5) tuberculosis; (6) local causes. The relative importance of these causes differs with the age of the child. In infancy and early childhood most of the cases complicate disease of the lung or pleura, usually of the left side. After the fourth year rheumatism takes the first place as an etiological factor. Pericarditis is then generally associated with endocarditis, and may precede or follow the articular manifestations of rheumatism. Following scarlet fever, pericarditis generally occurs in connection with nephritis or multiple joint inflammations. In typhoid fever, also, it is usually associated with pneumonia or joint lesions. Pyæmia may be a cause in the newly born, or it may occur in connection with disease of the bones or joints in older children; in both it is usually associated with similar lesions of other serous membranes. Tuberculous pericarditis is more frequent after the third year, and is generally secondary to pulmonary tuberculosis. Among the local causes may be mentioned traumatism, ulceration of a foreign body from the œsophagus into the pericardium, disease of the sternum, ribs, or vertebræ, and abscesses resulting from cheesy bronchial lymph nodes.

Lesions.—1. *Pericardial transudations*, or an increase in the normal pericardial fluid, are met with in many conditions in which there is a

very marked degree of anæmia, general dropsy, or a weak heart, particularly of the right side. Generally from one and a half to two ounces of a clear serum are found in the pericardial sac.

2. *External or mediastinal pericarditis* is always associated with mediastinal pleurisy, and results in more or less extensive adhesions of the pericardial and pleural surfaces, with an increase in the connective tissue of the mediastinum. It is often a tuberculous process. When severe, it may cause compression of the large blood-vessels, and seldom in any other way produces symptoms. With this form there may be inflammation of the internal layer of the pericardium. It is only inflammation of the internal layer which is ordinarily considered as pericarditis, the other form being preferably classed as *mediastinitis*.

3. *Dry pericarditis.*—This may be either general or localized. If the latter, it is more often seen at the base than at the apex of the heart. The two opposing surfaces are usually involved. As a result of the inflammation they are coated with fibrin, which may be partly absorbed, but usually leaves behind bands of adhesions of greater or less extent. From repeated attacks there may result complete obliteration of the pericardial sac.

4. *The sero-fibrinous form—pericarditis with effusion.*—This is the most common variety. The heart appears roughened from the exudate which often completely covers it, forming bands which extend from one surface to the other. The serum may be clear, or contain flakes of lymph, and varies in amount from a few ounces to a pint. In cases terminating in recovery there is gradual absorption of the serum and part of the fibrin, but adhesions more or less extensive always remain.

5. *Purulent pericarditis.*—If the inflammation is set up by a foreign body ulcerating into the sac, by the rupture of a mediastinal abscess, or by general pyæmia, the process may be purulent from the outset. More frequently, however, in purulent pericarditis there is first an abundant exudation of fibrin with pus cells in its meshes, and subsequently the pouring out of fluid pus, precisely as in empyema, with which it is very often associated. If death occurs in the early stage, both surfaces of the pericardium are found coated with a thick exudate of greenish-yellow lymph, but little or no fluid pus may be present. At a later period the pericardial sac contains pus, which may vary in amount from a few ounces to one or two pints. Purulent pericarditis, which is secondary to pneumonia or pleurisy, is usually due to the pneumococcus. In other cases any of the pyogenic germs may be found.

6. *Pericarditis with an effusion of blood* is very rare in children. It may occur from the rupture of organized adhesions or in certain blood states such as purpura, and very rarely in tuberculosis.

Pericarditis complicating pneumonia and pleurisy is generally fibrinous or fibrino-purulent; that with rheumatism is sero-fibrinous, and often accompanied by endocarditis. With acute tuberculosis there is usually

only a deposit of miliary tubercles, or there may be a small serous or serosanguinolent effusion. In chronic cases there may be a tuberculous inflammation with the formation of caseous nodules, new connective tissue, and extensive adhesions. This generally occurs in connection with pulmonary tuberculosis—sometimes with tuberculous peritonitis.

In any form of pericarditis complete recovery, so far as pathological conditions are concerned, is rare—if, indeed, it ever occurs. Generally adhesions remain, which may be in the form of a few thin connective-tissue bands, or so extensive as to produce almost entire obliteration of the pericardial sac. Such adhesions are usually followed by secondary changes. The growth and development of the heart are interfered with, and there may be sufficient pressure upon the coronary vessels to lead to degeneration of the muscular walls and dilatation of the heart. With large fluid exudations there may be an interference with the systemic circulation, enlargement of the spleen and liver, and sometimes general dropsy.

Symptoms.—A pericardial transudation, or dropsy of the pericardium, is very rarely large enough to make a diagnosis possible.

External pericarditis is seldom recognised during life, there being no symptoms except those of the pleurisy with which it is associated. Occasionally there may be heard, particularly if the inflammation is anterior, a pleuritic friction sound which is increased with the systole of the heart. The pulse may be weak during inspiration, and there may be an increased area of cardiac dulness. If the inflammation is chiefly posterior, it causes only the symptoms of mediastinitis, which is recognised principally by its pressure effects upon the great vessels. It may produce œdema of the face or of the lower extremities, ascites, enlargement of the liver and spleen, but rarely albuminuria. It is usually progressive, and lasts from a few months to two or three years, according to its cause.

Inflammation of the internal layer is the only form usually described as pericarditis. This is very frequently overlooked, not only on account of its rarity, but from the obscurity of its symptoms. The difficulty in diagnosis is particularly great in young children. The symptoms are few, and many of them are equivocal. As this disease is nearly always secondary, the physician should be on the watch for it in infants with pleurisy or pleuro-pneumonia of the left side, and in older children in the course of articular rheumatism. Localized pain and tenderness may be present, and also a certain amount of embarrassment of the heart's action, usually manifested by præcordial distress, palpitation, and slight irregularity of the pulse. There may be dyspnœa, and if there is a large effusion present there may be orthopnœa and cyanosis. Sometimes there is delirium. When pericarditis follows pleurisy or pleuro-pneumonia there are frequently no new symptoms added.

The physical signs in older children resemble those in adults. In dry pericarditis there is usually heard a double friction sound over the præcor-

dial space, the area being generally small and near the base of the heart. The sound is not transmitted, and bears no relation to the respiratory movements. After effusion has taken place the apex beat may be displaced upward, diffused, and somewhat indistinct, or it may not be found at all. There may be bulging of the chest wall. On palpation, there is an absence of vocal fremitus over an area usually occupied by the lung. Percussion gives an area of marked dulness or flatness of triangular shape, the base being below and the apex above. The normal area of cardiac dulness is increased in all directions, and this dulness extends beyond the limits of the heart. On auscultation, the heart sounds are feeble and distant. Friction sounds disappear as serum is poured out, and reappear as it is absorbed. Endocardial murmurs may also be present. In infants, physical signs are often entirely wanting, or the normal sounds may be feeble, distant, or absent.

The usual duration of acute pericarditis is from one to three weeks. The ordinary dry form, with its resulting adhesions, may be followed by a subacute or chronic form of the disease. In the sero-fibrinous form the serum is usually absorbed quite promptly, and only adhesions are left, or a chronic inflammation follows, with exacerbations in each recurrence of rheumatism. In the purulent form of the disease in young children, death is the most frequent termination. If the pus is evacuated, or spontaneous opening takes place, there may be recovery, but always with more or less extensive adhesions remaining.

Prognosis.—Of thirty-five cases in Steffen's collection, only six recovered. This statement is to be taken rather as evidence of the great difficulty of diagnosis than of a very high mortality, although the disease is always a serious one. The prognosis depends chiefly upon the exciting cause. When due to pyæmia or the acute infectious diseases, or when extending from pleurisy or pneumonia, the prognosis is bad. Here it is usually the primary disease rather than the pericarditis which is the cause of death; the latter may be the case, however, if the effusion is large. The cases in which the pericarditis itself is the most important disease are those depending upon rheumatism. Although immediate danger to life may not often be great, yet convalescence is slow, and the remote consequences of the disease, by reason of adhesions, may be very serious.

Diagnosis.—Owing to the very rapid action of the heart in children, acute dry pericarditis presents difficulties of diagnosis in early life which are not met with in the adult. The disease is fortunately so rare under three years, that in ordinary practice it need seldom be considered. In older children the diagnosis is to be made by essentially the same signs as in adults. Pericarditis with effusion is to be diagnosticated from dilatation of the heart and from pleuritic effusions. From dilatation, the diagnosis is not often difficult in childhood, for this is not a common condition, and is rarely extreme except in advanced valvular disease. From

pleuritic effusions the diagnosis is at times almost impossible. Signs pointing to a sacculated empyema of the left side anteriorly should always be regarded with suspicion, particularly if the apex beat is not displaced to the right, and if the heart sounds are very feeble. When empyema and pericarditis coexist, it may be impossible to recognise the condition. The diagnosis between serous and purulent effusions can be made only by aspiration. Fluid effusions in infants are almost invariably purulent, and so also are they in the majority of cases in older children, unless due to rheumatism.

Treatment.—In the early part of an attack of acute pericarditis the patient should be kept in bed and as quiet as possible, and hot poultices or counter-irritation by mustard used over the heart. Sometimes an ice bag may with advantage be substituted. Excessive heart action may be controlled by aconite, and severe pain may require opium. If the disease is due to rheumatism, anti-rheumatic remedies should be employed. Serous effusions usually subside under simple tonic treatment. If absorption is slow, it may be hastened by counter-irritation. When a large effusion forms rapidly there may be danger of death from syncope. Symptoms which indicate an unfavourable termination are cyanosis, weak, irregular pulse, and great dyspnœa, or orthopnœa. Under these conditions aspiration may afford temporary relief, and free diuresis should be induced by citrate of potash and caffein. The inhalation of oxygen is at times of great value in cases presenting such urgent symptoms. If pus is shown to be present by puncture, incision and drainage should be practised, as in empyema. The results of aspiration in such cases are extremely unfavourable. Of eighteen cases of aspiration of the pericardium collected by Keating, only four recovered. In puncturing the pericardium the point usually selected is a little to the left of the border of the sternum in the fifth intercostal space, the needle being directed upward and outward.

CHRONIC PERICARDITIS WITH ADHESIONS.

This is not a very uncommon condition. It may be general or localized. The youngest case which has come under my observation was in a female child sixteen months old, who died from acute broncho-pneumonia. The adhesions were old and general, the pericardial sac being completely obliterated. There was also some old pleurisy present. The history threw no light upon the lesions. As already stated, such adhesions may follow single, but more frequently recurrent, attacks of rheumatic pericarditis. Sometimes the process may be tuberculous. The adhesions may increase until they are one eighth or even one fourth of an inch in thickness. Adhesive pericarditis is usually accompanied by some dilatation of the heart, which may be preceded by hypertrophy, and there may or may not be valvular disease.

Partial adhesions cause no symptoms by which they can be recognised,

and even general adhesions sufficient to obliterate the pericardial sac are found at autopsy where not suspected during life. This is one of the conditions in which, after it has led to considerable dilatation of the heart, sudden death sometimes occurs. It often happens that the only cardiac symptoms present are such as could be explained by functional disturbance. The heart is almost invariably enlarged. On inspection, there is seen bulging of the chest wall, with a strong and somewhat diffused apex beat. One of the most characteristic signs is that during systole there occurs a retraction of the chest over a small area at or near the apex of the heart, sometimes at the tip of the sternum, and sometimes at the epigastrium. This is often better appreciated by palpation than by inspection. It is followed by a rapid rebound, associated with diastolic collapse of the jugular veins. A similar retraction, according to Broadbent, is to be seen behind in the infrascapular region, sometimes on the left and sometimes on the right side. Percussion shows an increase in the cardiac dulness in all directions, but particularly upward. Hale White has called attention to the frequency of a presystolic murmur of a "blubbering" character in these cases. The diagnosis of adherent pericardium always presents difficulties, but it can be made with tolerable certainty in a considerable proportion of the cases. On account of the enlargement of the heart and the frequency of the murmurs, it is usually mistaken for valvular disease. The lesion is a permanent one, and tends to increase. The treatment is symptomatic.

CHAPTER IV.

ENDOCARDITIS AND VALVULAR DISEASE.

ACUTE SIMPLE ENDOCARDITIS.

Acute endocarditis may occur even in fœtal life. At this period it usually affects the right side of the heart, and is one of the important causes of congenital malformations. In infancy, acute endocarditis is exceedingly rare, not a single instance being found in over one thousand autopsies upon children under three years of age of which I have records. From the third to the fifth year it is not so rare, and after this period it is quite common. Of 95 cases observed by Steffen, 15 occurred before the sixth year, and 80 between the sixth and fourteenth years.

Acute endocarditis may be primary, but it is much more frequently a secondary disease. The primary cases have been the subject of much discussion, but I agree with those who regard the great majority of these as rheumatic. Cheadle (London) has well said that we are to look upon endocarditis in children not as a complication of rheumatism, so

much as a manifestation—often the first—of that disease. Sometimes endocarditis occurs alone, and sometimes it is associated with chorea without articular symptoms; but the latter almost invariably appear sooner or later. Endocarditis is seen as a frequent complication both of acute and of subacute articular rheumatism. The proportion of rheumatic cases in which it occurs is much larger in children than in adults. Compared with rheumatism, all other causes of acute endocarditis are very infrequent. It is seen occasionally in the course of nearly all the acute infectious diseases, most often with scarlet fever, and it sometimes complicates pleurisy and pneumonia, being usually associated with pericarditis. In infectious diseases, and in pleurisy and pneumonia, the endocarditis is probably excited by pathogenic germs. Fraenkel and Sanger have found the staphylococcus in cases of simple endocarditis, and cultures by others have shown the presence of other pyogenic organisms, including the pneumococcus.

Lesions.—Acute inflammation may affect any part of the endocardium, but in extra-uterine life it usually affects the valves of the left side, involving the mitral much more frequently than the aortic valve. Steffen's figures give only four examples of aortic disease in ninety-five cases. (Compare statistics of valvular disease, page 583.)

The pathological changes consist first in an extensive growth of new connective-tissue cells and an infiltration of round cells beneath the endothelial layer. This results in the formation of small masses of granulation-tissue upon the valves or the endocardium of the heart wall, and upon these there is deposited fibrin from the blood. In this way the tiny wart-like excrescences known as vegetations are produced. Bacteria may also be caught in the exudate. As a consequence of the inflammation, the valve is swollen, somewhat shortened, and consequently insufficient. The results of the process may be ulceration of this new-formed tissue, which in ordinary cases is small in amount, or organization and cicatrization. Masses of fibrin may be detached from the vegetations and swept into the general circulation, lodging as emboli in the kidneys, spleen, brain, or other organs. This is not common in acute endocarditis, at least not in the first attacks.

In the milder forms of inflammation it is possible for complete recovery to take place, with the exception of a slight valvular thickening, not enough, however, to interfere in any way with the function of the valves. But this result is rare. In most cases they remain slightly insufficient, as the least serious consequence of the inflammation. Unfortunately, it more often happens that an acute inflammation which may not be at first serious, proves the beginning of the progressive changes of a chronic inflammation, the full effects of which are not seen for years. Chronic inflammation may follow the first attack immediately, or after a considerable interval, or occur after several acute attacks.

Symptoms.—When acute endocarditis occurs as a primary disease, or when it is the only manifestation of rheumatism, it usually begins abruptly with rather severe general symptoms—high temperature, often 102° to 105° F., prostration, exaggerated heart action, restlessness, and sometimes dyspnœa. There is nothing distinctive about these symptoms, and it is not until the heart is examined that the disease is recognised. If the heart is not watched, the diagnosis is not made, and there may be no suspicion of the nature of the attack until some time afterward, when the existence of valvular disease is discovered. If the heart is carefully examined from day to day, nothing abnormal may be found until the third or fourth day, or even later, when there is heard the characteristic soft, blowing, systolic murmur at the apex. The murmur is generally transmitted to the left. It may be accompanied by a thrill and by an accentuated pulmonic second sound, and later there may be evidence of slight dilatation with the usual signs of some degree of cardiac insufficiency. The murmur gradually increases in intensity until the maximum is reached, and then in most cases somewhat subsides.

Acute endocarditis sometimes occurs in the course of, or simultaneously with, an attack of chorea, with symptoms quite similar to those described. Finlayson (Glasgow) has called attention to endocarditis as a frequent cause of obscure fever in choreic patients, either when occurring alone or with articular symptoms. It may develop at any time during the choreic attack or subsequent to it. When endocarditis occurs as a complication of articular rheumatism, there may be an increase in the temperature and in the severity of the general symptoms, but rarely anything more definite. Endocarditis complicating other diseases is recognised only by the physical signs.

The usual duration of acute endocarditis is from one to three weeks, the febrile symptoms frequently subsiding in a few days and the cardiac symptoms slowly diminishing.

The attack may terminate fatally in the course of a few weeks, owing to the rapid development of acute dilatation, accompanied by the usual signs of cardiac insufficiency, with dropsy, cyanosis, and often pulmonary complications. Cerebral embolism may occur, which usually produces hemiplegia, but rarely results fatally. If emboli lodge in the spleen or kidneys, they may lead to swelling of the spleen or to hæmaturia. The patient may recover with a murmur which lasts but a few weeks and gradually disappears—a rare result. Usually there is a persistent murmur, with the subsequent development of the ordinary signs of valvular disease. Lastly, there may be recurrent attacks of inflammation, with the ultimate development of chronic valvular disease.

Diagnosis.—The diagnosis of acute endocarditis is very frequently not made; not because it is difficult, but because in young children the heart is not examined as frequently and as carefully as it should be. The symp-

toms are few and not diagnostic. It is therefore of the greatest importance that not only in chorea and rheumatism, but in all acute febrile attacks, particularly those of obscure origin, the heart should be closely watched. Endocarditis affecting the wall of the heart can not be diagnosticated. The murmur of valvular endocarditis may be confounded with pericarditis, or with functional or blood murmurs occurring in the course of acute febrile attacks, or with those of anæmic origin. From pericarditis it is distinguished by the fact that the murmur is single, has a soft blowing character, is usually located at the apex, is transmitted beyond the border of the heart, and is diminished by a full inspiration. Functional murmurs in febrile diseases are quite frequent in young children, and may at first be difficult to distinguish from those of endocarditis. Usually, however, the former are at the base rather than at the apex. They are more irregular, both as to time, transmission, and constancy, than are murmurs resulting from acute endocarditis. The same may be said of anæmic murmurs, which, as in adults, may be heard in the carotids, and sometimes over any of the large arteries.

Prognosis.—The danger to life in acute endocarditis is not often great, as the disease seldom proves fatal. However, death may occur when it is associated with chorea, but here usually when an acute process is ingrafted upon an old valvular disease. In other cases, death results from complications, particularly pneumonia. Only the progress of the case enables one to decide how extensive is the damage which has been done to the valves. There is always the danger of recurrent attacks.

Treatment.—All the so-called primary cases, as well as those occurring with chorea and articular symptoms, should have the benefit of antirheumatic remedies, as this is the only plan which offers any chance of limiting the inflammation, although the effect upon the heart is rarely striking. Excessive cardiac action is sometimes allayed by aconite, sometimes best by opium. The most important thing in the management of these cases, and the one frequently overlooked, is to secure for the heart as complete rest as possible, not only during the period of acute inflammation, but for several succeeding weeks. Patients should be kept in bed for at least a month, and only the slightest exertion permitted for many weeks. It is during this early period of the disease that changes take place most rapidly in the heart walls, and the gravest results sometimes follow the neglect of these precautions. Children are often allowed out of bed as soon as the fever has subsided, and the heart disease is unnoticed until a grave amount of dilatation has developed, with dropsy, palpitation, shortness of breath, slight cyanosis, irregular pulse, and cough. All children who have once suffered from endocarditis should be protected as much as possible against subsequent attacks of rheumatism.

MALIGNANT ENDOCARDITIS.

Malignant or ulcerative endocarditis is a rare disease in childhood. The youngest case I have found reported is that of Harris, which occurred in a boy four years old, and affected the right side of the heart. It was secondary to a cardiac malformation. Of the cases thus far reported in early life, about twenty-five in number, the great proportion have been in children over ten years of age, in whom the disease does not differ essentially from the adult type. For the most exhaustive study of this subject we are indebted to Osler's Gulstonian Lectures.

Malignant endocarditis rarely occurs as a primary affection. Of the acute diseases, it is most frequently secondary to pneumonia, next to rheumatism and meningitis. It may be met with in any infectious disease or septic process. In 75 per cent of the cases, according to Osler, it is ingrafted upon a previous valvular disease. In my series of collected cases of congenital malformations of the heart, there were four deaths from malignant endocarditis, all but one, however, occurring in adult life.

The bacteria most frequently associated are the staphylococcus and streptococcus, and, in the cases complicating pneumonia, the pneumococcus. These micro-organisms are believed to play an important part in the production of the disease. Circulating in the blood, they lodge upon the endocardium of the valves, all the more readily when they are previously diseased.

Lesions.—Malignant endocarditis may result in the production of vegetations which subsequently break down, or there may be superficial ulceration affecting only the endocardium, or deeper ulceration involving the valve, the septum, or even the heart wall. In other cases there is suppuration of the deeper tissues of the valve first affected, with the production of small abscesses at the base of the vegetations. These conditions may lead to large perforations, or even to the destruction of the valve, to valvular aneurisms, or abscesses of the heart wall. According to Osler, the different parts of the heart are affected in the following order: mitral valve; aortic, mitral and aortic combined; tricuspid and pulmonic valves; and the cardiac wall. The secondary lesions of malignant endocarditis are due to emboli. These are most frequent in the spleen and kidney, next in the brain, intestines, and skin, and, if the right side of the heart is diseased, in the lungs. These emboli lead to the formation of red or white infarctions, to hæmorrhages, or to multiple abscesses in the various organs and tissues in which they lodge.

Symptoms.—Malignant endocarditis presents a great variety of symptoms, making the diagnosis extremely difficult in perhaps the majority of cases. There is generally a remittent type of fever, sometimes repeated rigors, profuse sweating, low delirium, stupor or coma, and extreme prostration. In many cases there is a fine petechial eruption upon the skin;

diarrhœa is also frequent. The cerebral symptoms may be so prominent as to suggest meningitis. There is usually a cardiac murmur, the location of which depends upon the seat of disease. It is most frequently the murmur of mitral regurgitation. This murmur is sometimes faint, and may be absent. The spleen is in most cases enlarged. From the emboli there may be hemiplegia, rapid swelling of the spleen, bloody urine, cough, and symptoms of pneumonia. The disease lasts from a few days to six weeks, death being the almost invariable termination. It is due to exhaustion or to some embolic process.

Diagnosis.—The most characteristic features of malignant endocarditis are the development of pyæmic or typhoid symptoms with a petechial eruption, in a patient who has previously had valvular disease. Malignant endocarditis is differentiated from typhoid fever by its sudden onset, irregular temperature, recurring chills, profuse sweats, petechial eruption, and dyspnœa. It may be confounded with malarial fever.

Treatment.—This is entirely symptomatic; no known measures have any influence upon the disease itself.

CHRONIC VALVULAR DISEASE.

Chronic valvular disease of the heart in children is usually the result of endocarditis; in a small number of cases it depends upon congenital malformation; but the degenerative lesions to which many adult cases are due have no place in early life.

Lesions.—The changes of chronic endocarditis may be briefly described as follows: The valvular segments are thickened by the production of new connective tissue, the contraction of which results in retraction, shortening, puckering, and imperfect closure of the valves. The valvular leaflets may adhere to each other, so that the opening is very much narrowed. This is sometimes reduced to a funnel-shaped orifice barely admitting the tip of the finger, and it may even be much smaller. The leaflets are sometimes adherent to the wall of the heart; the chordæ tendineæ are shortened, and sometimes entirely disappear; and, finally, the valves may be the seat of calcareous deposits. These changes take place very slowly, requiring many years for their full development. From time to time there may be attacks of acute inflammation. The changes described may bring about (1) valvular insufficiency, owing to imperfect closure, causing a regurgitation of blood through the opening guarded by the valve; or (2) stenosis, with such a narrowing of the opening that the outflow of blood is obstructed. In early life it is usually the mitral valve that is affected.

Of 141 cases in children under fourteen years old, observed clinically by Dr. F. M. Crandall and myself, the mitral valve was alone affected in 79 per cent; the aortic valve alone in 3 per cent; and both were associated in 18 per cent. Lesions of the aortic valve in early life are therefore comparatively rare.

Following valvular lesions, important changes take place in the wall and cavities of the heart: these are hypertrophy and dilatation.

Hypertrophy.—This consists in an increase in the thickness of the heart wall, due to an increase in the size and number of the muscular fibres. It is principally of the ventricles, and is always conservative. It may continue indefinitely, or it may be followed by degeneration and dilatation. Hypertrophy occurs as a result of any obstructive lesion at one of the cardiac orifices, in renal disease when the obstruction is in the small arteries, also when extra work is thrown upon the ventricles as a result of regurgitation, and it may follow primary dilatation.

Dilatation.—This consists in an enlargement of the cavities of the heart, usually with thinning of their walls. It is generally most marked in the auricles. Primary dilatation is produced by regurgitation of blood into any of the cavities as a result of valvular insufficiency. This may to a slight extent be regarded as a conservative lesion. Secondary dilatation, or that resulting from degeneration of the cardiac muscle, is always injurious. It is usually caused by imperfect nutrition of the heart which may be due to local or general causes. In most of the cases both hypertrophy and dilatation continue for a long time. So long as hypertrophy predominates, the circulation may be well carried on; but when dilatation comes to exceed hypertrophy, there are signs of great embarrassment to the circulation and of cardiac insufficiency.

There are other lesions accompanying chronic valvular disease, depending upon obstruction to the venous circulation. If this obstruction is in the pulmonary veins, it leads to congestion of the lungs, chronic bronchitis, or chronic pneumonia; if of the systemic venous circulation, it leads to chronic congestion of the spleen, liver, kidneys, peritonæum, and sometimes to general dropsy.

Etiology.—The following table gives the age and sex in the cases observed by Dr. Crandall and myself:

	1 year.	2 years.	3 years.	4 years.	5 years.	6 years.	7 years.	8 years.	9 years.	10 years.	11 years.	12 years.	13 years.	14 years.	
Males.....	..	1	2	2	4	6	4	9	8	6	5	7	6	1	= 55, or 38%
Females...	..	1	3	5	7	9	10	3	11	12	14	4	2	3	= 90, " 62%
Total....	..	2	5	7	11	15	14	12	19	18	19	11	8	4	= 145

The difference in sex is very nearly the same as was found in my cases of rheumatism. Sturges, in 100 cases, gives 56 per cent females and 44 per cent males. Sansom's figures alone give a predominance of males.

The chronic endocarditis of early life is, as a rule, secondary to the acute or subacute form. Its etiological factors are therefore those of acute endocarditis. Of 117 cases in my own series, 93, or 80 per cent, gave a history of previous rheumatism—7 cases of chorea without articular symptoms being included as rheumatic. Of the 31 cases which

at the first examination gave no history of rheumatism, 8 subsequently developed articular rheumatism, and 2 chorea, so that nearly 90 per cent of this series of cases presented, to my mind, conclusive evidence of a rheumatic diathesis. Thirty per cent had chorea previously, or developed it while under observation. The more closely I study cases of rheumatism, chorea, and valvular disease, and the longer the patients are kept under observation, the deeper becomes my conviction of the very close relationship between these three conditions in childhood. The percentage of rheumatic cases in this series is considerably larger than that given by many writers, but it corresponds very closely with Cheadle's careful observations. Valvular disease is occasionally traced to an attack of endocarditis complicating scarlet fever, and in rare cases to that occurring with other infectious diseases.

Symptoms.—The symptoms of chronic valvular disease in most cases come on slowly, often insidiously, and frequently there are none until the disease has lasted a long time, the condition being discovered by accident. The course of valvular disease is usually divided into two periods, the first being that while compensation is present, and the second after compensation has failed. The duration of the stage of compensation is indefinite; it may last a lifetime. The only subjective symptom that is of much diagnostic value is shortness of breath on exertion. Occasionally other symptoms are present, such as præcordial pain, attacks of palpitation, headache, epistaxis, anæmia, and cough. These are rarely constant, but come on when the patient's general condition for any reason is below normal. As a rule, there is in young subjects a tendency to an increase in the disease, although this is often slow, and may be interrupted by long periods in which the process appears to be stationary. At such times the patients either have no symptoms, or suffer only from a slight amount of inconvenience on marked exertion.

Failure in compensation is generally brought about by one of the following causes: There may be an intercurrent attack of acute endocarditis, which in a short time leads to a very great increase in the heart's disability. It may be due to additional work thrown upon the heart from excessive muscular exertion, or to the strain of a prolonged attack of some acute illness, especially one that is liable to produce changes in the heart muscle, such as typhoid or scarlet fever. It is sometimes the increased work which is physiologically thrown upon the heart at the time of puberty, owing to the rapid growth of the body. It may result from any cause which seriously affects the patient's general nutrition, particularly when this is associated with marked anæmia.

The symptoms indicating failure of compensation are those depending upon a weak heart, with imperfect filling of the arteries and overfilling of the veins. The embarrassment of the pulmonary circulation leads to constant dyspnœa or orthopnœa and cough, sometimes accompanied by profuse

expectoration, which may be bloody, and in rare cases there may be larger pulmonary hæmorrhages. The obstruction to the systemic venous circulation leads to dropsy, which begins in the feet. There may be general anasarca and dropsy of the serous cavities, especially the peritonæum and pleura; also enlargement and functional disturbances of the liver, enlargement of the spleen, dyspeptic symptoms, and chronic congestion of the kidney, with scanty urine and albuminuria. There may be dilatation of the superficial veins, with clubbing of the fingers, and cyanosis; and there may be cerebral symptoms, such as headache, dizziness, and fainting attacks. The pulse is small and soft, and the heart's action rapid and irregular.

It is rare to see all the symptoms of cardiac failure in children under ten years, but about the time of puberty they are not uncommon. The symptoms may increase in severity until death occurs, or they may be severe for a time and then nearly disappear, to return again after a longer or shorter interval.* Death may be due to sudden cardiac paralysis,

---

* The course and termination of these cases of chronic valvular disease is well illustrated by the following history of a little girl who was under observation for nine years: When first seen she was seven years old, and gave a history of cardiac symptoms for one year. There was then present a loud mitral regurgitant murmur, with considerable hypertrophy. There was general dropsy, and all the symptoms pointed toward acute dilatation. Under treatment, the dropsy and other symptoms disappeared, and she went on comfortably for over a year. In her eighth and ninth years there were frequent attacks of subacute rheumatism, during which time the heart lesion steadily increased in severity. At twelve years there was an eruption of subcutaneous tendinous nodules, which remained for over two years. During this year there was heard for the first time a mitral direct murmur, accompanied by a very marked thrill, mitral stenosis having been gradually brought about by the slowly progressing endocarditis. This murmur gradually increased in intensity from that time, while the mitral regurgitant murmur became less distinct. The apex beat at this time was in the sixth space, two and a half inches to the left of the nipple. From the twelfth to the fifteenth year she grew very little in height or weight, and showed no signs of maturity, the cardiac symptoms being nearly stationary. In the fifteenth year she developed a marked enlargement of the liver and spleen with general dropsy and all the symptoms of cardiac insufficiency, these being the first symptoms of this character since she was seven years old. There was now heard for the first time an aortic regurgitant murmur in addition to the others formerly present. The symptoms disappeared under treatment in the course of a few months, but six months later returned with greater severity and were accompanied by albuminuria, the patient dying from heart failure in a few weeks. During the last exacerbation there was heard a double aortic as well as a double mitral murmur.

At autopsy the heart weighed fifteen ounces. There was a very great hypertrophy, especially of the right ventricle, which was as thick as the left. All the cavities were much dilated. The most important valvular lesion was mitral stenosis, the orifice not admitting the end of the little finger. The valves were the seat of calcareous deposits. The curtains of the aortic valve were thickened and adherent; there was also thickening of the pulmonic and tricuspid valves.

to intercurrent nephritis, pneumonia, embolism, inflammation of the serous membranes, or to œdema of the lungs.

Clinical Varieties.—Of the 141 cases of valvular disease in children under fourteen years, previously referred to, the following were the forms and combinations recorded. It is to be noted that these figures are based upon clinical and not pathological examinations:

Mitral insufficiency	131 cases;	alone in 99 cases.
Mitral stenosis	17 "	" " 4 "
Aortic insufficiency	9 "	" " 0 "
Aortic stenosis	28 "	" " 3 "
Double mitral	8 "	
Double aortic	1 case.	
Double mitral and double aortic	3 cases.	
Mitral insufficiency and double aortic	3 "	
Mitral insufficiency and aortic stenosis	18 "	
Mitral stenosis and aortic insufficiency	2 "	

Mitral insufficiency.—This is usually the result of attacks of acute endocarditis. It is by far the most frequent form of valvular disease in early life, occurring in 93 per cent of the above cases, and alone in 70 per cent. In mitral insufficiency there is regurgitation of blood from the left ventricle into the left auricle during systole. This is compensated for by hypertrophy of both ventricles. It causes dilatation of the left auricle, increased pressure in the pulmonary veins, afterward in the pulmonary arteries, hypertrophy of the right ventricle, and, finally, there is dilatation of the right ventricle, tricuspid insufficiency, dilatation of the right auricle, and general systemic venous obstruction. Coincident with the changes in the right heart there is hypertrophy of the left ventricle, followed by dilatation.

In mitral insufficiency there is heard a systolic murmur which is synchronous with the apex impulse and with the first sound of the heart, and may in part replace the first sound. It is loudest at the apex, transmitted to the left, and heard with almost equal distinctness at the inferior angle of the left scapula. This is a very diffusible murmur, and may be audible all over the chest. It is accompanied by an accentuation of the pulmonic second sound heard at the left border of the sternum in the second space, and by signs of hypertrophy of the heart. When both these signs are wanting, the existence of mitral insufficiency is somewhat doubtful, as a similar murmur may be of functional or accidental origin. In the early stages of the disease the signs of hypertrophy predominate; in the later stages, those of dilatation.

In hypertrophy of the left ventricle or of the whole heart, the apex beat is displaced downward and to the left.* It may be in the fifth or

* For normal position of the apex in childhood, see page 560.

the sixth space, but rarely lower, and as far to the left as the axillary line. There is often bulging of the præcordia, so marked as to cause a deformity of the chest. The impulse is forcible and heaving, and over a larger space than normal. The area of cardiac dulness is increased in all directions, but particularly downward and to the left. In hypertrophy involving chiefly the right ventricle, there may be bulging of the lower part of the sternum, and the area of dulness is increased to the right, in extreme cases extending from one to one and a half inches beyond the right border of the sternum. The heart sounds in hypertrophy are loud and distinct, and often have a somewhat metallic character. With hypertrophy of the right ventricle there may be reduplication or accentuation of the second sound. The pulse is full and strong.

In dilatation the apex beat is indistinct, diffuse, and undulatory. There is an increase in the area of cardiac dulness, the outline being nearly square. The cardiac sounds are feeble, and murmurs previously present may be lost. The heart's action is irregular, and the pulse small and weak.

Mitral stenosis.—This is apt to occur from repeated attacks of subacute rheumatism, with a slowly progressing endocarditis. It is usually associated with mitral regurgitation, but may occur alone. There is with this lesion obstruction to the flow of blood from the left auricle into the left ventricle. It is mainly compensated for by hypertrophy of the right ventricle, but to a certain degree by hypertrophy of the left auricle. The secondary changes following the lesion are hypertrophy of the left auricle followed by dilatation, increased pressure in the pulmonary veins, followed by hypertrophy and dilatation of the right ventricle. The left ventricle is usually normal or small.

Mitral stenosis produces a presystolic murmur which is somewhat prolonged, usually rough in character, and terminates sharply with the first sound of the heart. It is loudest at or near the apex, but is audible over only a small circumscribed area. Quite as constant and important for diagnosis is the presence of a "purring thrill," which is very distinct upon palpation, and terminates sharply as the apex strikes the chest wall. The pulse of mitral obstruction is usually small. The symptoms are few, but those which are present depend chiefly upon pulmonary congestion.

Aortic stenosis.—This is not very common in early life, and rarely occurs as the only murmur, being most frequently associated with mitral insufficiency. It is sometimes a congenital murmur. Aortic obstruction is compensated for by hypertrophy of the left ventricle, which may be complete for a long period, but ultimately it is followed by dilatation of the left ventricle, with mitral insufficiency and its consequences. In aortic obstruction there is an interference with the outflow of blood from the left ventricle into the aorta. It causes a systolic murmur, which is usually loudest at the right border of the sternum in the second space,

and is transmitted upward, being distinct in the carotids. The second sound is generally weak. There are associated the signs of marked hypertrophy of the left ventricle.

Aortic obstruction is more frequently confounded with conditions giving accidental or functional murmurs than is any other valvular lesion. Without the signs of hypertrophy of the left ventricle, a positive diagnosis should not be made. On account of the almost perfect compensation, this form of the disease causes fewer symptoms than any other variety, possibly excepting mitral obstruction. The danger of embolism is somewhat greater than in mitral disease.

Aortic insufficiency.—This is one of the rarest valvular lesions in children. In no case on my list did it occur as the only lesion. It causes a regurgitation of blood from the aorta into the left ventricle during diastole. It is compensated for by dilatation and hypertrophy of the left ventricle. The order in which the secondary changes take place is: dilatation followed by hypertrophy of the left ventricle, ultimately followed by further dilatation due to degeneration, this leading to mitral insufficiency with all its remote consequences. The signs of aortic insufficiency are a prolonged diastolic murmur, with, or taking the place of, the second sound of the heart, generally loudest at the left border of the sternum in the second space, and transmitted downward to the apex of the heart or the ensiform cartilage. This is invariably accompanied by signs of hypertrophy and dilatation of the left ventricle, these being usually marked. In the stage of compensation the signs of hypertrophy predominate, and when compensation has failed, the signs of dilatation. A characteristic symptom is the intense throbbing of the carotids, with the sudden distension and complete collapse of their walls, and the "ball-pulse" of Corrigan. Early in the disease there may be headache, flashes of light before the eyes, and other evidences of cerebral congestion. In the late stages there may be fainting attacks. With this lesion compensation may be complete for a long time.

Tricuspid insufficiency.—This is usually secondary to disease of the left side of the heart, occurring in its late stages. It most frequently follows mitral insufficiency, where it is usually due to dilatation of the right ventricle without changes in the valves. It may be secondary to certain diseases of the lungs, such as emphysema, chronic interstitial pneumonia, or chronic pleurisy, and it may be due to congenital malformation. Tricuspid insufficiency gives a systolic murmur, loudest over the lower part of the sternum, but heard usually over a small area. It is generally associated with signs of dilatation of the right ventricle. The jugular veins stand out prominently, and often show systolic pulsation, especially upon the right side. The symptoms associated with tricuspid regurgitation are due to general systemic venous obstruction, already mentioned in connection with mitral insufficiency.

Tricuspid stenosis, pulmonic stenosis, and pulmonic insufficiency are practically unknown in childhood, except in congenital cardiac disease.

Prognosis of Valvular Disease.—Complete recovery from valvular disease is possible only when the lesions are very slight. Few children die from cardiac disease before reaching the age of fourteen years, sudden death being extremely rare. A large proportion of the cases do fairly well up to about the time of puberty, when they begin to lose ground, often failing rapidly. Others do well until a fresh endocarditis is lighted up by an intercurrent attack of rheumatism, after which the disease may make rapid progress. The proportion of children who have serious cardiac lesions before the age of eight years, and reach adult life in good condition is comparatively small.

There are several features of cardiac disease in children, in consequence of which, serious lesions tend to progress more rapidly than in adults. The muscular walls are less resistant, and hence rapid dilatation occurs much more readily than in adult life. The heart must provide not only for constant needs, but for the growth of the body. If the patient's general nutrition is poor during the period of most rapid growth, this tells quickly and seriously upon the heart, and dilatation makes rapid progress; but if the general nutrition continues good the heart may do more than hold its own throughout childhood. The demands made upon the heart at puberty are especially severe, by reason of the rapid growth of the body and the frequency of anæmia and malnutrition. There is always present the danger of rapid advances in the disease from intercurrent attacks of rheumatism, from which children are more likely to suffer than are older subjects. Extensive pericardial adhesions are not infrequent, and seriously handicap the heart, greatly increasing the tendency to dilatation. The effect upon the heart of poor food, unhygienic surroundings, and general malnutrition is much more marked than in adults.

These unfavourable conditions are in part offset by others in which the child has an advantage over the adult. Disease of the coronary arteries is very rare, and the valvular lesions which are most frequently met with—mitral insufficiency and aortic obstruction—are those which admit of the most complete compensation.

In making a prognosis in any given case, the amount of hypertrophy or dilatation which exists is of much more importance than the location or the special character of the murmur. The condition of the arterial and venous circulation must also be taken into consideration; also how rapidly the disease is progressing, the condition of the patient's general health, and how well circumstances will admit of proper hygienic and general management. The presence of valvular disease in childhood increases the danger from every acute disease, especially pertussis, diphtheria, and scarlet fever.

Diagnosis.—Valvular disease is to be particularly distinguished from conditions in which there are heard functional or accidental murmurs. According to my own experience the latter are quite common even in young children. Mistakes usually arise from attaching too much importance to the presence of murmurs, and too little to the changes in the walls and cavities of the heart, with which valvular disease is almost invariably associated. It is not always possible to decide whether a murmur is organic or functional until the patient has been for some time under observation and treatment, particularly when anæmia is present. The diagnostic points, so far as the murmurs are concerned, are mentioned in connection with anæmic murmurs (page 590).

Treatment.—A child who is the subject of chronic valvular disease should be under constant medical supervision. Irreparable harm often results from wilful, but more frequently from ignorant, disregard of the simplest and most important rules of life for these patients. The facts should be plainly stated, the course of the disease and the dangers fully explained to parents, and, when old enough, to the child himself. At the very least the patient should be carefully examined three or four times each year, in order that the physician may note the progress of the disease, and be able to modify the child's occupation, exercise, and surroundings, in order to meet, so far as possible, the changing conditions. Few patients need more watchful oversight than children with cardiac disease. The greatest care should be exercised, especially in all recent cases, not to overtax the heart.

During the stage of compensation, treatment directed especially to the heart is rarely necessary. The main purpose should be to maintain the patient's general nutrition at the highest possible point during the period of active growth. To this end, diet, sleep, study, and exercise should receive the most careful attention. If malnutrition and anæmia are allowed to go on unchecked until they have become severe, the cardiac disease may make rapid strides, and as much harm be done in a few months as otherwise might not occur in years. The special symptoms of malnutrition and anæmia should be met as they arise, by the same means as when they occur under other conditions. The question of exercise and recreation is always a difficult one to settle. Often too little latitude is given, and the heart, like the voluntary muscles, loses its tone. Every form of exercise requiring a prolonged severe strain should be forbidden, particularly swimming and competitive games, like ball and tennis, and others requiring much running; but skating, rowing, mountain-climbing, horseback exercise, gymnastics, and even cycling on the level—all in moderation—may be allowed not only without harm, but with the greatest benefit; but any of these, used immoderately, may be productive of great injury. All exercise should be taken with regularity and system, the amount being carefully measured by the child's condition. If the patient

is a boy who must earn his own living, the physician should see to it that the occupation chosen is not one liable to make special demands upon the heart.

Special watchfulness is required at the time of puberty to prevent overpressure in schools, and the development of anæmia or chlorosis. The first symptoms of these conditions should be treated energetically, and if the heart seems to be overtaxed the child should be put to bed. Patients should be so far as possible removed from conditions liable to induce fresh attacks of rheumatism. To this end, if possible, they should spend the winter and spring months in a warm, dry climate.

In the stage of failing compensation, the same general conditions are present as in adults, and they are to be managed in pretty much the same way. When such symptoms are first seen, prolonged rest in bed should be insisted upon as the thing most likely to restore the normal conditions. Cardiac dropsy with low arterial tension and weak pulse, calls for digitalis. An overloaded venous circulation may be relieved by diuretics, or, better, by saline purgatives. Iron and tonics generally are indicated, particularly strychnine and cod-liver oil. In cases of sudden heart failure, nitroglycerin, ether, and ammonia are as valuable as in adults; but better, probably, than any of these is the use of strychnine hypodermically.

MYOCARDITIS.

Disease of the muscular wall of the heart is rare in children, and of comparatively little importance, except in connection with the acute infectious diseases. Myocarditis may, however, occur at any age, even in fœtal life. As seen in children, it is almost invariably a secondary lesion, usually the result of some infectious process. The two diseases which furnish most of the cases are scarlet fever and diphtheria. The most important local cause is pericarditis with adhesions.

Lesions.—In extra-uterine life, myocarditis, as a rule, affects the wall of the left ventricle, the papillary muscles, or the septum. The heart is pale or of a yellowish-white colour, very soft and flabby, and there is frequently dilatation of the cavities. Small ecchymoses may be seen beneath the pericardium.

Two varieties of myocarditis are described: In the parenchymatous form there is a degeneration of the muscle fibre which, according to Romberg, is most frequently albuminous, next fatty, and least frequently hyaline. There is a loss of the transverse striations, and there may be complete disintegration of the fibres. This process may be circumscribed, but it is usually diffuse. In the interstitial form the lesion usually occurs in small, circumscribed areas. There is an infiltration of round cells between the muscular fibres of the heart. The process, when acute, may result in absorption or in the production of small abscesses. There may also be congestion and minute blood extravasations. In chronic cases it may

lead to the formation of larger or smaller areas of dense connective tissue resembling cicatrices, in the heart wall. Either the interstitial or the parenchymatous form may occur alone, but in most of the acute cases they are combined. In addition, there is usually some degree of mural endocarditis and inflammation of the pericardium next to the heart wall. Dilatation frequently follows; rarely abscesses may form, which may open into the heart or into the pericardium. Cardiac aneurism, and even rupture, have been known to occur in a child of six years (Hadden's case).

Symptoms.—These are very rarely sufficiently marked to enable one to make a positive diagnosis. In many cases in which advanced lesions have been found at autopsy there have been no symptoms during life, and in others none until the occurrence of sudden death. This is usually from cardiac paralysis, rarely from rupture. In eight cases studied by Romberg, which occurred in the course of diphtheria, not one had cardiac symptoms during life and two died suddenly. When symptoms are present, they are generally those of feeble heart action—a faint apex impulse, a slow, weak pulse of irregular rhythm, pallor, dyspnœa, and attacks of syncope. In the late stages there may be the physical signs of dilatation, with dropsy of the feet or the serous cavities, and scanty urine, sometimes containing albumin.

Diagnosis.—A positive diagnosis of myocarditis is impossible. It may be suspected in the course of diphtheria, scarlet or typhoid fever, when cardiac symptoms like those mentioned occur, and when pericarditis and endocarditis can be excluded by the physical examination.

Treatment.—This is mainly symptomatic. After severe attacks of those infectious diseases in which myocarditis is liable to occur, and at any time when it is suspected, patients should be kept recumbent for several weeks, and special care exercised to prevent any sudden exertion, as death has occurred from so slight a thing as suddenly sitting up in bed. Iron, alcohol, and tonics should be given, the best of all of these being strychnine. Digitalis should be used with caution, and never in large doses. In some cases with symptoms indicating imminent heart failure, more striking benefit follows the use of morphine hypodermically than any other plan of treatment.

ANÆMIC MURMURS.

As already stated, these are not uncommon even in infancy. They may be confounded with organic murmurs, either from congenital malformations or acquired disease. I have several times found the heart normal at autopsy in cases where a diagnosis of congenital disease had been unhesitatingly made during life, the murmur having been of anæmic origin. In any anæmic infant, as well as older child, one should hesitate to make a diagnosis either of congenital or acquired organic disease, from the mere presence of a murmur.

An anæmic murmur is usually systolic, heard at the base of the heart, also in the carotids, often in the subclavian arteries, and occasionally over any of the large trunks of the body. The murmur varies from day to day, and sometimes it is altered by changing the position of the patient. It may be loud enough to be heard over a great part of the chest in front, and even behind. There is frequently present a venous hum in the neck. There are no signs of hypertrophy, nor is there the accentuated second sound so characteristic of mitral disease. The pulse is not usually strong. Anæmic murmurs diminish in intensity and ultimately disappear with improvement in the general condition of the patient. In some cases one must wait for the effects of treatment before giving a positive opinion.

FUNCTIONAL DISORDERS OF THE HEART.

Disturbances in the heart's action unconnected with organic disease, are rare in infants and young children; but after the seventh year they are not uncommon, becoming in fact quite frequent as puberty approaches. One of the most important causes is indigestion; another is overpressure in schools, or anything else leading to nervous exhaustion. In these circumstances it is usually associated with other mental or psychical disturbances. An important predisposing cause is the demand made upon the heart by the rapid growth of the body about the time of puberty, particularly when this is associated with anæmia. In some of the cases there is a definite exciting cause, such as fright or great excitement, and it may be due to the excessive use of tea, coffee, or tobacco, especially in the form of cigarette-smoking. In a few instances it has been traced to masturbation. It may follow any acute disease, such as typhoid fever, malaria, or one of the exanthemata, and occasionally it occurs in the course of these diseases, or with bronchitis or pneumonia.

Symptoms.—The usual manifestations are attacks of palpitation; less frequently there is tachycardia (rapid heart) or bradycardia (slow heart). The majority of children complain more with functional disturbances than with organic disease, certainly while the latter is accompanied by compensation. Attacks of palpitation occur in paroxysms. In the severe form there is usually a sense of oppression in the region of the heart, with some dyspnœa, or even orthopnœa. The pulse is usually rapid, from 120 to 130, and is irregular both as to force and rhythm. The carotids pulsate strongly. The apex impulse is felt over an increased area, the heart sounds are usually strong but irregular, and sometimes a slight murmur is heard. The face is pale or flushed. There may be headache, vertigo, spots before the eyes, and noises in the ears. Sometimes there is slight cyanosis with cold hands and feet, and general perspiration. The frequency of these attacks depends upon the nature of the exciting cause. Their duration is from a few minutes to several hours.

Diagnosis.—Functional disorders are differentiated from organic cardiac disease only by careful and repeated examinations of the heart. In the diagnosis of functional disturbance especial importance is to be attached to a neurotic or neurasthenic condition of the patient, to the presence of some adequate exciting cause, the absence of evidence of enlargement of the heart, and the fact that the pulmonic second sound is not increased.

Prognosis.—This in most cases is favourable, for with improvement in the patient's general condition, with the growth of the body, and in girls with the establishment of menstruation, the attacks usually disappear.

Treatment.—During the attacks, digitalis in moderate doses should be given, also bromides or valerian. The curative treatment is to be directed toward the cause. Where no special cause can be discovered a general tonic plan of treatment should be adopted, with careful regulation of the patient's diet, exercise, and mode of life. All stimulating food, tea, coffee, and tobacco should be prohibited. Anæmia should receive its appropriate remedies. The hours of sleep and study, and the amount and character of exercise allowed, should be carefully regulated. During the intervals no treatment of the heart is necessary.

DISEASES OF THE BLOOD-VESSELS.

Abnormally Small Arteries (*Arterial hypoplasia*).—This condition is not a very common one, but it has attracted a good deal of attention, having been studied especially by Virchow. The only thing which is abnormal in the circulatory system may be that the aorta, and sometimes all the large vessels are only two thirds or three fourths their usual calibre, or even less. This may interfere seriously with the growth and development of the body, especially of the genital organs, although this result is not a constant one. The condition is found occasionally in cases of chlorosis, and in the congenital cases it may be the chief cause. There is usually associated a certain amount of hypertrophy of the heart. The other symptoms are anæmia, and sometimes an imperfect development of the body. A positive diagnosis during life is impossible.

Aneurism and Atheroma.—In early life chronic disease of the blood-vessels is exceedingly rare, yet a sufficient number of observations have been recorded to show that even young children are not exempt from this form of disease. There had been reported up to 1890 twenty-eight cases of aneurism in patients under twenty years of age (Jacobi).* Of these, however, only twelve were under fourteen years. Sanné † records the youngest case, which occurred in a fœtus born at about the eighth month,

* A. Jacobi. Archives of Pædiatrics, vol. vii, p. 161.

† Sanné, Revue Mensuelle des Maladies des l'Enfance, vol. v, p. 56. In these articles will be found references to most of the reported cases.

in whose body there was found a large aneurism of the abdominal aorta just below the origin of the renal arteries. Of the eleven remaining cases occurring in children under fourteen years, in over one half the number the arch of the aorta was the part affected. In one case the seat was the femoral artery, in another the external iliac, and in still another the abdominal aorta.

Probably the most important etiological factor, as in adult life, is syphilis, but in only a few of the cases reported was the evidence of syphilis conclusive. In two cases there was general tuberculosis. In addition to these general causes, aneurism may be due to some local condition, such as an erosion from bone, an abscess in the neighbourhood, or to embolism. The symptoms and course of aneurism in young children do not differ essentially from those of the disease as seen in adults.

In addition to the cases of aneurism referred to above, I have found reports of seven cases of atheroma in very young subjects. In Sanné's case the patient was but two years old, and patches of atheromatous degeneration were found in several places in the aorta. In Hawkins's case, eleven years old, there was found extensive atheromatous disease of the aorta, subclavian and carotid arteries. In Filatoff's case, atheromatous degeneration affected the arteries at the base of the brain, causing death from cerebral hæmorrhage. It is interesting to note that in this patient, who was only eleven years old, there was also present chronic diffuse nephritis with contracted kidneys. A similar condition of the kidneys and arteries was observed by Dickinson in a girl of six years.

Embolism and Thrombosis.—Embolism has already been referred to in connection with acute endocarditis. It may be seen at any age, even in infancy, but generally occurs in patients over five years old. The emboli are usually swept into the circulation from vegetations upon the valves of the heart. The symptoms which they produce will depend upon the nature of the emboli and the vessels occluded by them. If they lodge in the brain they may cause paralysis or convulsions; if in the spleen, pain and swelling of this organ; if in the kidneys, pain, tenderness, and sometimes hæmaturia; if in the lungs, cough, sometimes accompanied by hæmoptysis and occasionally by a sharp thoracic pain. If the emboli are infectious, they may give rise to abscesses. The pathological results following embolism are similar to those which are seen in adults.

The most frequent form of thrombosis, that occurring in the sinuses of the brain, is discussed in connection with Diseases of the Nervous System. Cardiac thrombi, especially of the right side of the heart, are not infrequently found in patients dying from heart disease, pneumonia, and occasionally also from other acute inflammatory processes and acute infectious diseases, particularly diphtheria. These thrombi are in most cases produced during the last few hours of life, or just at the time of death, and are of no clinical importance. They frequently extend from the heart into the

large blood-vessels, particularly the pulmonary artery. Thrombosis occasionally occurs in all the large vascular trunks in childhood as well as in adult life.

Thrombosis of the internal jugular vein.—Pasteur * reports a case in a child two and a half years old, in which the middle of the vein was filled with an organized thrombus, and the lower portion obliterated and reduced to a fibrous cord. The symptoms were swelling, œdema, and cyanosis of the face, and dilatation of the facial vein, but not of the external jugular. There were clubbing of the fingers and œdema of the feet, but not of the arm. The heart was found to be dilated and hypertrophied, but was not the seat of valvular disease. The symptoms had existed since an attack of pneumonia, eighteen months before death.

Thrombosis of the vena cava.—Quite a number of cases are on record where this has occurred as the result of pressure from large abdominal tumours; it has followed new growths of the kidney and large masses of tuberculous lymph nodes. Neurutter and Salmon have recorded a case of thrombosis, apparently of marantic origin, in a child seven years old. The thrombus filled the vena cava, and extended to the origin of the hepatic veins and into both femorals. Death occurred from tuberculosis. In Scudder's case (seventeen years old) there was apparently obliteration (probably congenital) of the inferior vena cava; there was an extensive varicose condition of all the abdominal veins. The symptoms of thrombosis of the vena cava are swelling and œdema of the feet—sometimes of the abdominal walls and the groin—and very great dilatation of the superficial abdominal veins.

Thrombosis of the aorta.—A case has been reported by Leopold in a newly-born child which was delivered by version. The thrombus was of recent origin, and filled the lower aorta, extending into the femoral artery. A case of thrombosis of the aorta occurring in a girl of thirteen years has been reported by Wallis. The aorta was very narrow, and probably the seat of syphilitic disease. The thrombus extended from the origin of the renal arteries to the cœliac axis.

Thrombosis in infectious diseases.—There is occasionally seen in typhoid fever, but more frequently in diphtheria, thrombosis of some of the large venous trunks, usually of one of the lower extremities. The symptoms are pain, localized swelling, and partial paralysis. If the artery is affected, there may be gangrene.

---

* Lancet, February 11, 1888.

SECTION VI.

DISEASES OF THE URO-GENITAL SYSTEM.

CHAPTER I.

THE URINE IN INFANCY AND CHILDHOOD.

While a study of the urine is of much less importance in early life than of the symptoms referable either to the digestive or respiratory system, it is deserving of much more attention than it has generally received. In infancy especially it is attended with difficulty, owing to the fact that it is by no means an easy matter to secure a specimen for examination.

Methods of Collecting Urine.—In male infants this may be done by placing the penis in the neck of a small bottle which lies between the thighs and is secured in position by pieces of tape passing over the hips and beneath the perinæum. A still better plan is to use a condom in the place of a bottle. The urine of female infants can sometimes be collected in a similar way by placing a small cup over the vulva and holding it in place by the napkin. In either sex, if the infant is placed upon a chamber regularly every ten or twenty minutes for a few hours, it is rarely difficult to secure the urine, especially if at the same time a cold hand or a cold compress be placed over the bladder; sometimes hot applications will answer the purpose better. A small amount, sufficient to test for albumin, may often be obtained by placing absorbent cotton over the vulva or penis. The most certain of all means, however, is catheterization; in females sometimes nothing else will answer the purpose. A soft rubber catheter, size 6 or 7, American scale (9 or 11 French), should be used for infants.

Daily Quantity.—This is relatively much larger in infants than in older children and in adults, on account of the more active metabolism of the young child and the large amount of water taken with the food. The quantity fluctuates widely from day to day according to the amount of fluid food taken and the activity of the skin and bowels. The following figures are the averages obtained by combining the results of the investigations of Schabanowa, Cruse, Camerer, Pollak, Martin-Ruge, Berti Schiff, and Herter:

Average Daily Quantity of Urine in Health.

Age.	Grammes.	Ounces.
First twenty-four hours	0 to 60	0 to 2
Second twenty-four hours	10 " 90	½ " 3
Three to six days	90 " 250	3 " 8
Seven days to two months	150 " 400	5 " 13
Two to six months	210 " 500	7 " 16
Six months to two years	250 " 600	8 " 20
Two to five years	500 " 800	16 " 26
Five to eight years	600 " 1,200	20 " 40
Eight to fourteen years	1,000 " 1,500	32 " 48

Frequency of Micturition.—This is greatest in young infants, and diminishes steadily as age advances. In the first two years, during the waking hours, the urine is generally passed as often as twice an hour, while during sleep it is retained from two to six hours. By the third year the urine may be held during sleep for eight or nine hours, and at other times for two or three hours. Such control of the sphincter of the bladder is often obtained at two years, and sometimes even at an earlier period. From slight nervous disturbances or minor ailments of any kind, this control is impaired, and the water may be passed by children of four or five years with the frequency seen in infants.

Physical Characters.—The urine of the newly born is usually highly coloured. During later infancy it is pale and frequently turbid, even when practically normal, owing to the presence of mucus; this turbidity often no amount of filtration will entirely remove. Less frequently turbidity depends upon urates. The urine of the first few days of life often shows a deposit of urates or uric acid in the form of a reddish-yellow stain upon the napkin. The reaction of the urine at this time is usually strongly acid, but throughout the rest of infancy it is faintly acid or neutral.

The specific gravity is higher during the first two days than at any time in infancy on account of the scanty supply of fluid taken; it is usually lowest from the third to the sixth day, but from this time it rises steadily until puberty is reached. The specific gravity will of course vary with the quantity. From the writers already referred to the following figures are taken:

	Specific gravity.
First to third day	1·010 to 1·012
Fourth to tenth day	1·004 " 1·008
Tenth day to sixth month	1·004 " 1·010
Six months to two years	1·006 " 1·012
Two to eight years	1·008 " 1·016
Eight to fourteen years	1·012 " 1·020

Microscopically, the urine of the newly born shows the presence of many squamous epithelial cells, mucus, granular matter, and crystals of

uric acid and amorphous or crystalline urates. It is not uncommon to find hyaline and even granular casts. Martin-Ruge found hyaline casts in the urine of fourteen out of twenty-four healthy nursing infants examined during the first week. Granular casts were much less frequent. The microscopical appearances of the normal urine of later infancy and childhood present no peculiarities.

Composition.—*Urea.*—The following figures show the average daily quantity of urea eliminated at the different ages:

Age.	Daily quantity of urea.
First day	0·076 to 0·114 gramme.
Second to seventh day	0·140 " 0·660 "
One to two months	0·90 " 1·40 "
Three to five years	13·09 " 14·01 grammes.
Five to thirteen years	16·05 " 21·03 "

Uric acid.—Few observations have been made upon the elimination of uric acid, but all authorities agree that it is much higher in the newly born than at any subsequent period of life. The quantity is better appreciated by giving the ratio between the uric acid and urea than by the absolute quantity of the former. The figures here given for the newly born are taken from Martin-Ruge; the others are from Herter.

Ratio of Uric Acid to Urea.

In the newly born	1 to 14
Under one year	1 " 60–80
From two to five years	1 " 50–70
From five to fifteen years	1 " 45–60

The inorganic salts (phosphates, chlorides, sulphates) are all present in the urine of the newly born, but in relatively small quantity, increasing as age advances. The colouring matters are also less abundant.

Albumin is often present in the urine during the first days, but usually in small amount. Cruse found it twenty-eight times in ninety observations upon healthy infants; usually the quantity was small, amounting to traces only, but in two cases it was quite large upon the second day. These observations are confirmed by the investigations of Martin-Ruge, and also of Pollak.

Sugar is frequently found in the urine of healthy infants during the first two months. This subject is referred to later under the head of Glycosuria.

FUNCTIONAL OR CYCLIC ALBUMINURIA.

Etiology.—This condition, although a rare one in young children, is quite common between the ages of ten and sixteen years. I shall not in this connection include cases sometimes classed as febrile albuminuria, in which there is usually present the condition described as acute degeneration of the kidneys.

The causes of functional or physiological albuminuria, and the circumstances in which it has been observed, are many and varied. It is much more common in males than in females. In many patients it is regularly cyclic in character, albumin being absent in the urine passed during the night or early morning, but present during the day, diminishing in the evening and absent at bed-time. In a case reported by Tiemann, the morning urine showed no trace of albumin in seventy-eight of eighty-four examinations. At noon albumin was present in ninety-eight of one hundred and thirteen examinations. In certain cases albuminuria is distinctly traceable to cold bathing; in others, to fatigue following excessive muscular exercise; in still others, to dyspeptic conditions. It may be associated with a diet rich in nitrogenous food. In other cases none of these conditions exist, and there is simply the occasional presence of albumin in the urine.

Many theories have been advanced in explanation of cyclic albuminuria. Sometimes it appears to be clearly traceable to irritation of the kidney by uric acid, urates, or oxalates. Kinnicutt believes this to be one of the prominent causes, and that albuminuria is due to vaso-motor disturbances in the kidney. Delafield compares the exudation of serum from the vessels of the kidney to the dropsy of the feet seen in anæmia. Da Costa believes that it always depends upon slight changes of an evanescent character in the kidney.

Symptoms.—Many of the patients exhibiting cyclic or periodical albuminuria are well nourished, and have no other signs of disease; others show dyspeptic symptoms, and are anæmic and poorly nourished, suffering from headaches and other neuroses. In the cases distinctly periodical the amount of albumin is commonly small. It is not infrequently associated with temporary glycosuria. As a rule, casts are absent, although it is not uncommon to find a few hyaline casts, and occasionally granular casts are also present. A gouty family history exists in a certain proportion of the cases, and some of the patients themselves present other evidences of this diathesis.

Diagnosis.—Pavy mentions the following points as characteristic of physiological or functional albuminuria: (1) The time of its occurrence. The absence of albumin early in the morning, its presence in the forenoon, and diminution toward evening. When this is repeated day after day the diagnosis is, he believes, quite positive. (2) The absence of serious impairment of the general health and of the characteristic symptoms of nephritis, such as dropsy, cardiac hypertrophy, a pulse of high tension, retinal changes, etc. (3) The fact that casts are, as a rule, absent. (4) That crystals of oxalate of lime are present, and the urine is of high specific gravity.

Too much stress is certainly laid by Pavy and many other writers upon the fact that the albumin is found in the urine only at certain

times in the day. This is not characteristic of functional albuminuria, as the same thing occurs in many cases of chronic nephritis, especially in the early stages when the amount of albumin present is small. All these cases must be carefully watched for a long time and many observations made, before nephritis can positively be excluded.

Prognosis.—The prognosis in cases of purely functional albuminuria is good. It is to be remembered that patients who for a considerable time have been regarded as having only functional albuminuria have ultimately developed nephritis; hence an absolutely favourable prognosis is possible only after a long period of observation. If albumin is constantly present it is probably pathological, and the longer it continues the more serious is the outlook.

Treatment.—This is to be directed toward the patient's general condition rather than to the kidneys and the urine. The dyspeptic symptoms must be relieved, the patient's mode of life regulated, only moderate exercise allowed, and a simple diet given which does not consist too largely of nitrogenous food. If the urine is of high specific gravity, and contains oxalate-of-lime crystals, alkalies and mineral waters should be given in addition. Iron is indicated if there is anæmia present.

HÆMATURIA.

Hæmaturia is characterized by the presence of red blood-cells in the urine, and is to be distinguished from hæmoglobinuria where only blood pigment is present.

Hæmaturia may result from local or general causes. In infancy it may be due to new growths of the kidney. In such cases the hæmorrhages are often abundant, and may be the first symptom of the condition. Hæmaturia may occur also as a symptom of acute nephritis, especially that complicating scarlet fever, or it may result from the irritation of a calculus in the kidney, the ureter, or the bladder. In rare instances its cause is a new growth of the bladder, and it may be due to traumatism. Among the general causes the most important are: the hæmorrhagic disease of the newly born; the blood dyscrasiæ, such as scurvy, purpura, and hæmophilia; and infectious diseases, particularly malaria, typhoid, variola, scarlet fever, and influenza. In most of these cases the amount of blood passed is small. When it is large it may appear in the urine as clear blood, or as clots, or it may impart simply a reddish or smoky colour to the urine. The colour, however, is not a reliable guide; the best of all is the microscopical examination. For a simple chemical test guaiacum may be used.

To discover the source of the blood may be quite difficult. Large hæmorrhages are much more likely to come from the kidneys than from the bladder. The presence of blood casts from the renal tubules, or larger

ones from the ureter, are conclusive evidence of the renal origin of the hæmorrhage.

In children, renal hæmorrhage in itself rarely requires treatment; when it does, the same remedies are indicated as in the adult, viz., ergot, gallic acid, and rest in bed. Some obstinate cases have been cured by drinking water from alum springs.

HÆMOGLOBINURIA.

In this condition blood pigment appears in the urine in large quantity, but red blood-cells are very few in number, or are absent altogether. In severe cases the urine may be almost black. There is commonly a small amount of albumin. This condition may be recognised by the appearance of granules of pigment under the microscope, or by Heller's test; the most conclusive means of diagnosis, however, is the spectroscope.

Epidemic hæmoglobinuria (Winckel's disease) has already been described in the chapter on Diseases of the Newly Born. Hæmoglobinuria may be due to certain poisons, as carbolic acid or chlorate of potash, or to certain infectious diseases, as scarlet fever, typhoid fever, malaria, syphilis, and erysipelas.

Paroxysmal hæmoglobinuria occurs in childhood, although it is an exceedingly rare condition. A typical case in a child of four and a half years has been reported by Mackenzie. This was a delicate child of syphilitic parents; the hæmoglobinuria was preceded by fever and chills, without any other evidence of the presence of malaria.

The exact pathology of hæmoglobinuria is at present unknown, and its treatment is very unsatisfactory.

GLYCOSURIA.

By this term is understood the occasional or transient appearance of sugar in the urine. This is not very infrequent in children, and may be met with even during the first month of life. Grósz has published some careful investigations upon the glycosuria of early infancy.* He made many observations upon fifty infants during the first month of life, from which the following conclusions were drawn: Glycosuria is not uncommon in nursing infants; but it is not seen in nursing infants who are perfectly healthy. It occurs particularly with certain disturbances of digestion, whether functional or inflammatory. The sugar found in the urine under these conditions reacts strongly to the reduction test (Fehling's), but not to the fermentation test; sometimes the polariscope shows that it has the power of dextro-rotation. This is believed to be milk sugar, or one of its derivatives. It is not of constant or regular occurrence. It may be

* Jahrbuch für Kinderheilkunde, Bd. xxxiv, p. 83.

produced artificially by increasing the amount of milk sugar above that which can be normally absorbed. This quantity Grósz places at 3·3 grammes for each kilogramme of the body weight. If more than this is given, or if there is diminished capacity for the absorption of sugar, glycosuria occurs.

Koplik has made some observations upon the urine of patients fed chiefly upon infant foods composed largely of sugar. He found sugar in five out of ten cases examined; in three, the sugar responded both to Fehling's and the fermentation test; in two cases to Fehling's test only.

There seems to be no doubt regarding the existence of dietetic glycosuria in infants and in older children. Repeated examinations of the urine are, however, necessary in order to exclude more serious disease.

PYURIA.

Pus in the urine may exist as an acute or a chronic condition. In either case, in a child, it is much more likely to come from the pelvis of the kidney than from any other source. It may, however, come from any part of the genito-urinary tract—the kidney or its pelvis, the ureters, the bladder, the urethra, or the vagina. Sometimes it comes from an outside source, as when an abscess from perinephritis, appendicitis, or caries of the spine opens into the urinary tract.

Coming from the pelvis of the kidney, pus may indicate, if the condition is an acute one, pyelitis, pyelo-nephritis, or pyonephrosis; if it is chronic, it points to renal tuberculosis or calculus. The amount of pus in any of these conditions may be quite large. The urine is turbid and usually acid in reaction. It contains many epithelial cells of the transitional forms described in the article on Pyelitis. The urine when containing much pus is always albuminous. A turbidity due to pus may be mistaken for an excessive deposit of urates, but a microscopical examination quickly reveals its true nature. It is rare that pus comes from the ureters except in connection with congenital malformations or the impaction of calculi. Pus from the bladder is usually in small quantity, especially in young children, and it is mixed with mucus. The urine may be alkaline or acid in reaction; there are associated the symptoms of vesical irritation or of cystitis. Pus from the lower genital tract is rare in children, but its causes are usually easily recognised by a local examination. When the cause of pyuria is the opening of an abscess into the urinary tract there is generally a sudden appearance of pus in large amount. It is in most cases of short duration, possibly only a few days, and it may disappear quite rapidly.

The treatment of pyuria depends altogether upon its cause. Improvement in the symptoms sometimes follows the use of benzoic acid or ben-

zoate of ammonia in doses of from two to five grains every three hours to a child of five years. It is especially indicated where the urine is strongly alkaline.

LITHURIA.

Lithuria is a condition in which there is an excessive elimination in the urine of uric acid or of urates. The amount of nitrogen compounds eliminated by the kidneys as uric acid and urea, varies much from day to day with the nature of the food and other conditions. Hence in estimating an excess of uric acid, the absolute quantity eliminated in twenty-four hours is much less significant than the ratio of the uric acid to the urea (page 596). Whenever this ratio is continuously disturbed, the excretion of uric acid may be considered abnormal, except, of course, in grave pathological conditions of the kidney, where there is an insufficient elimination of urea. Regarding the source of uric acid, the theory of Horbaczewski is that most widely accepted, viz., that it results from the destruction of the nuclein of the cells of the body, particularly of the white blood-cells.

For accurate knowledge as to the amount of uric acid eliminated, nothing short of a quantitative chemical analysis can be depended upon. But if amorphous urates are deposited in large amount, uric acid may be considered excessive if the specific gravity is not high (above 1.025). If the specific gravity is high, the precipitation may be explained simply by the concentration of the urine. The deposition of the crystals of uric acid, forming the familiar brick-dust deposit, is not evidence of excessive elimination. For a quantitative clinical test, that of Haycroft is probably the best.*

Lithuria is not a specific condition, but rather a very general symptom associated with many kinds of disturbances of nutrition. It may be found in anæmia, malnutrition, chorea, rheumatism, chronic dyspepsia, and in a great variety of other disorders. Regarding the significance of lithuria, thus much may be positively asserted: The excessive elimination of uric acid when continuous is always evidence of a serious disturbance of nutrition. The gravity of the condition will depend upon the degree of this excess and upon its duration.

The treatment of lithuria is the treatment of the condition upon which it depends. The essential pathological condition is not so much excessive elimination as excessive production.

Urine containing Crystals of Uric Acid in the Form of Brick-Dust Deposit.—This condition is not to be confounded with the one just described. As already stated, such precipitation is not to be taken as evidence of an excess of uric acid, and, in fact, in most of these cases there

* See Haig on Uric Acid in Health and Disease.

is no excess. The condition is rather one in which the solvent power of the urine for uric acid is much reduced. Such urine, as a rule, is high-coloured, strongly acid, and may have a high specific gravity.

This condition also is dependent upon a disturbance of nutrition, and one which is most frequently associated with a gouty diathesis. It is not very common in children except in those of gouty antecedents. In such patients it is only occasionally present, and is usually associated with some other disturbance of nutrition, often of digestion. It is frequently the cause of local irritation of the urinary passages, which is usually slight, but which may be severe.

In my experience these cases are most improved by cutting off sugar from the diet almost entirely, by greatly reducing the amount of starchy food and substituting a diet rich in nitrogen and fat, viz., meat, milk, and cream, together with plenty of outdoor exercise. The continued use of alkaline waters is also of decided advantage in most cases.

INDICANURIA.

Indicanuria is a condition characterized by the presence of indican in the urine. To Herter is due the credit of bringing this subject prominently to the minds of the profession in this country. Indican (indoxyl-potassium sulphate) is derived from indol, which is formed in the intestine by the agency of bacteria from the excessive putrefaction of the proteids. It may also be produced in other parts of the body where putrefactive processes are going on, as in extensive suppuration without drainage, in pulmonary cavities, empyema, etc. Indican is only one of the ethereal sulphates produced in the manner above indicated, and when other conditions like those mentioned are excluded it may be taken as an index of the amount of putrefaction going on in the intestine.

The presence of indican in the urine is demonstrated by adding certain oxidizing agents, which produce an indigo-blue colour.* The existence

* The commonly employed test for indican is that known as Jaffé's test. It is described by Herter as follows: Pour into a test-tube equal quantities of urine and strong hydrochloric acid so as to fill the tube to within half an inch of the top, and shake. If there is much indican, a dark blue or purple colour will be produced. Then add sufficient chloroform to completely fill the tube and shake thoroughly. It is important that the chloroform should completely fill the tube so that no air bubbles get in by the agitation. If, after standing, the chloroform assumes a deep-blue or violet colour, there is certainly an excess of indican. The reaction may not appear at first, but may come out after standing several hours, or if slight at first it may increase in intensity. Sometimes, when no reaction is obtained, it may be produced by adding one drop of a saturated solution of chloride of lime or of peroxide of hydrogen. No more than one drop should be added at a time, or the blue colour may be bleached. In alkaline urine the indican is usually destroyed, so that the test may be negative.

of indicanuria in children was formerly believed to be pathognomonic of tuberculosis. Later investigations have shown that this is not the case; for in cases of tuberculosis indican is almost as frequently absent as present.

Herter gives the following as the conditions under which indicanuria is likely to be present: It is found in chronic intestinal indigestion; in very many cases of chronic constipation; in many cases of epilepsy, just about the time of the seizures; in some cases of masturbation; frequently in children who are the subjects of night terrors, and in whom there are usually disturbances of digestion. According to other observers, it is found with great constancy in acute putrefactive diarrhœas. With the exceptions above noted, the source of the indican is always the same, viz., the excessive putrefaction of the proteid substances in the intestine.

Indicanuria is most frequently a symptom either of acute or chronic intestinal disease. It is important as being a guide by which we may estimate the other symptoms in these conditions, and the effects of treatment. While a trace of indican is frequently present in health, a strong indican reaction is always to be considered abnormal in a child. The indications for treatment are to diminish intestinal putrefaction. This is mainly dietetic, and is to be accomplished by means referred to in the treatment of chronic intestinal indigestion (page 368).

ACETONURIA—DIACETONURIA.

Acetone exists in small quantities in the urine of healthy children. According to Baginsky and Schrach, it is found in large quantities in many febrile diseases. It increases with the height of the fever and subsides with it. Acetone is probably formed from the destruction of the nitrogenous material of the body, as it is increased by a nitrogenous diet, and may disappear by a diet of carbohydrates. Baginsky found it also in children with epilepsy, sometimes during the attacks. It is not, however, believed to be the cause of the convulsive seizures, as it is absent in convulsions occurring under other conditions. It has no relation to rickets. According to Schrach, there is no connection between acetonuria and the nervous symptoms accompanying fever. Von Jaksch found acetone in a case of diabetic coma.

Binet found *diacetic acid* in sixty-nine out of one hundred and fifty examinations in febrile diseases, chiefly in scarlet fever, measles, and pneumonia. In diabetes this condition often precedes the development of coma, otherwise it is of no prognostic significance. Schrach found diacetonuria exceedingly common in all cases of continuous high fever. It is more frequently present than acetonuria, and ceases with the fever.*

* For literature, see Baginsky, Archiv für Kinderheilkunde, Bd. xi, p. 1.

ANURIA.

By this term is meant an arrest of the urinary secretion. To that form which occurs in the course of renal disease the term "suppression" is generally applied. Anuria is to be carefully distinguished from retention, from the scanty secretion which occurs whenever food is refused or withheld on account of illness, and also from that which accompanies acute diarrhœa with large, watery discharges. Anuria is sometimes seen in the newly born, where it depends upon some malformation of the genital tract; or it may depend upon uric-acid infarctions in the kidneys. The first urine passed after such an attack is very often highly acid, and may contain an abundance of uric-acid crystals and larger masses visible to the naked eye. Other cases admit of no such explanation, and the condition must be regarded as of nervous origin. For the time, the secretion appears to be completely arrested, as the bladder, both by palpation and catheterization, is found to be empty. This condition is not a very uncommon one in infancy, and it may continue for from twelve to thirty-six hours. So long as infants appear to be perfectly normal in every other respect, the suspension of the urinary secretion even for twenty-four hours need excite no anxiety.

The treatment is very simple and effectual, and consists in the administration of sweet spirits of nitre, either alone or in combination with the acetate or citrate of potash, and plenty of water. To an infant of three months one minim of the nitre and one grain of the citrate of potash may be given every hour in half an ounce of water until the urinary secretion is established, which will usually be in six or eight hours. If the urine is very highly acid, and stains the napkins, the potash should be continued for several days. Hot fomentations over the kidneys may be used with advantage.

DIABETES INSIPIDUS (POLYURIA).

This is a chronic disease characterized by the excretion of a very large amount of pale urine of low specific gravity. It is invariably accompanied by polydipsia. The disease is an exceedingly rare one in children.

The exact pathology of diabetes insipidus is not known; but from the conditions under which it occurs it is believed to be a neurosis. The irritation which gives rise to it may be in or near the floor of the fourth ventricle, or it may affect the renal nerves.

Etiology.—Of eighty-five cases collected by Strauss, twenty-one were under ten years of age and nine under five years. In Roberts' collection of seventy cases, the disease began in twenty-two before ten years, and in seven during infancy. In some cases it begins soon after birth. Males are more frequently affected than females, and in certain cases heredity is an important factor. Weil has published a remarkable example of the

disease existing in many members of a single family. Falls or blows upon the head, concussion of the brain, tumours of the brain, especially of the occipital region, tuberculous or cerebro-spinal meningitis or chronic hydrocephalus, all have been found associated with diabetes insipidus. It sometimes has followed the acute infectious diseases; but in many cases no cause whatever can be found.

Symptoms.—The quantity of urine is enormous, usually exceeding even that in diabetes mellitus. From five to twenty pints daily may be passed. The urine is pale, the specific gravity from 1·001 to 1·006, and it contains neither albumin nor grape sugar. In a few cases the presence of inosite (muscle sugar) has been found. Restricting the amount of fluid taken causes a very marked diminution in the amount of urine. The intense thirst leads patients to drink enormously of water and other fluids. Various contradictory statements are made by different writers regarding the quantity of uric acid and urea eliminated in these cases. The following are the results obtained in a case recently under observation in the Babies' Hospital.* The child was three years old, quite anæmic, and losing in weight. On January 20th the fluids were unrestricted, on the other days they were restricted:

Date.	Daily quantity of urine.		Specific gravity.	Total urea.	Total uric acid.	Indican reaction.	Inosite.
	Grammes.	Ounces.		Grammes.	Grammes.		
January 20	3,300	101½	1·006	22·276	0·173	None.	None.
" 25	750	25	1·010	9·049	0·072	Strong.	None.
" 26	775	25½	1·010	6·478	.....	.....	None.
February 8	1,320	49	1·007	12·113	0·110	None.	None.

The elimination of urea in this case is excessive, but the uric acid is not far from the normal.

Nervous symptoms are usually present. There may be disturbed sleep from the frequent micturition, palpitation, flushing of the face and other vaso-motor disturbances, headache, restlessness, and neuralgia. There may be incontinence of urine. The skin is pale and dry, and perspiration is scanty. The general health may not be disturbed. In most cases, however, it is somewhat affected, and there may be the usual symptoms of malnutrition, and even neurasthenia. If it affects young children, their growth may be considerably retarded. The appetite usually remains quite good. The temperature is at times slightly subnormal. The course of the disease is indefinite. It is very chronic, and may last for many years, death taking place only from intercurrent affections.

Prognosis.—A few of the cases recover spontaneously. Those of short duration are often cured by treatment. Of the chronic cases in which

* The analyses were made by Dr. C. A. Herter.

the disease is well established very few are controlled. The prognosis is worse if there are marked disturbances of the digestive tract or organic brain disease.

Diagnosis.—This is easily made from the two marked symptoms, excessive thirst and the polyuria. From diabetes mellitus it is easily distinguished by the low specific gravity and the absence of sugar from the urine. In older children, chronic nephritis with contracted kidney may be confounded with it.

Treatment.—Fluids should be moderately restricted. It is a serious mistake to reduce the quantity of fluids too much, since the drinking is not the cause of the diuresis. The diet should be simple and nutritious, consisting largely of meat, with a moderate amount of carbohydrates. The general treatment should be directed to the condition of malnutrition. The clothing should be warm, and a moderate amount of exercise should be allowed. Drugs are of little use; those which have sometimes been beneficial are arsenic, belladonna, ergotine, the bromides, and antipyrine. Treatment must be continued for many months to be of any value.

CHAPTER II.

DISEASES OF THE KIDNEYS.

MALFORMATIONS AND MALPOSITIONS.

Malformations of the kidney are not infrequent. In seven hundred and twenty-six consecutive autopsies at the New York Infant Asylum malformations of the kidney or ureters were met with in seventeen cases. This does not represent the actual frequency with which they occur, for in about half the number of autopsies in two other institutions only a single example was seen. Adding to the cases mentioned two others seen elsewhere, there are twenty cases of renal malformation of which I have notes, classed as follows:

Fusion of the kidneys, or horseshoe kidney	4 cases.
Supernumerary ureters	4 "
Hydronephrosis (alone)	8 "
Cystic degeneration of the kidney (alone)	2 "
Hydronephrosis and cystic kidney	1 case.
Single kidney	1 "

In all malformations the left kidney is much more frequently affected than the right, the proportion being nearly two to one. Malformations are more often seen in males than in females.

Fusion of the Kidneys.—In one case, in a child who died of pneumonia at the age of three years, the kidneys were fused into one irregular ovoid mass, lying upon the lumbar vertebræ; in another case the mass lay upon the promontory of the sacrum; in both there were two renal arteries and two ureters. In the two other cases the organs were united at their lower extremities, and in both of these there were two ureters passing in front of the kidney. In one there was also hydronephrosis and chronic diffuse nephritis. The children died at the ages of four and five months respectively.

Cystic Degeneration of the Kidneys.—In two of these three cases the right kidney was affected, and in one the left. The ages at which the children died were from seven to ten months. No renal symptoms were present. In all the cases the cystic kidney was very small, about an inch and a half in length and one inch in width. The organ was entirely made up of smaller and larger cysts containing a clear fluid, held together by loose connective tissue. The ureter was small and rarely pervious throughout. In one case there was hydronephrosis of the opposite side; in the others the opposite kidney was considerably enlarged, being about one half larger than normal. In addition to these small cystic kidneys there has been described a cystic degeneration in which very large cysts have formed even *in utero*, sometimes filling the abdominal cavity of the child and seriously interfering with delivery.

Single Kidney, the other being rudimentary or absent.—Of this I have seen but one example, which was found in a young man twenty-two years of age, who died of typhus fever in Bellevue Hospital. The right kidney weighed seven and a half ounces; the left was represented by a nodular mass about the size of an ovary, showing no trace of renal tissue. The ureter was pervious to within four inches of the kidney; the suprarenal capsule was normal. Macdonald has reported a case in which there was no trace whatever of the right kidney; the left was greatly enlarged, and weighed nine ounces. There were two suprarenal capsules but only one ureter. Schaeffer has reported absence of both kidneys in a seven-months' fœtus, associated with many other malformations.

Hydronephrosis.—Of the ten cases of which I have notes, this existed as the principal deformity in eight. In two cases it was associated respectively with cystic degeneration of the opposite kidney and horseshoe kidney. In seven cases only the left side was affected; in three there was double hydronephrosis. Seven patients were males and three females. Six died before they were six months old, and only two lived to be two years old. This condition is undoubtedly the result of some obstruction to the outflow of urine in the ureter, bladder, urethra, or prepuce, but in only three of my cases could there be found an obstruction sufficient to explain the deformity. In two there was marked hypertrophy of the bladder. In no case was a calculus found as the cause of the obstruction. In most of the cases the ureter was dilated to a diameter of from one

fourth to one half of an inch, and in two it was so large as to be easily mistaken for the small intestine. Usually the ureters appeared much elongated and sacculated; the pelvis of the kidney was dilated to the capacity of half an ounce or more, the calices forming pockets about half an inch in diameter. Less frequently the greater part of the kidney was destroyed, leaving only a series of communicating pockets surrounded by a thin cortex of renal tissue from one fourth to one eighth of an inch in thickness. In five cases there was chronic diffuse nephritis of the affected side, and sometimes both kidneys were involved, even though the hydronephrosis was unilateral. The nephritis was usually of a very advanced type. In two cases, typical examples of the atrophic form (contracted kidney) were seen, one of these children dying at the age of one month.* The organs are shown in Fig. 105. In two of the cases the bladder was the seat of very marked hypertrophy.

Urinary symptoms were noted in but one case, and in that they were due to pyelo-nephritis dependent upon the presence of calculi in the kidney not the seat of hydronephrosis. In no other case was the malformation suspected during life. Four patients died of marasmus, two of acute bronchopneumonia, and one of ileo-colitis. In only one was there any malformation outside the urinary tract, this being a case of congenital heart disease.

Double hydronephrosis is generally associated with, or results in, such changes in the kidneys that the patients die during infancy, commonly in the first year. At this age it rarely gives rise to a tumour, and is recognised only by the changes in the urine or by the other symptoms of nephritis. There may be the general and local symptoms of chronic diffuse nephritis, or, when infection of the genital tract occurs, there are added the symptoms of pyelitis. In the great majority of cases the condition is unrecognised, the patient dying of some disease not perhaps in itself fatal, but rendered so by the condition of the kidneys.

If hydronephrosis is unilateral there may be no symptoms until the

* This was in every way a remarkable case. The child died apparently of marasmus. There was double hydronephrosis, the ureters being three fourths of an inch in diameter. The right kidney was nodular upon the surface, and had a very adherent capsule. Just beneath the capsule there were small cysts containing pus. The left kidney was the seat of hydronephrosis, only its cortex remaining, this being about one sixth of an inch in thickness. Microscopical examination showed great thickening of the capsule of the right kidney, and several small abscesses situated in the cortex just beneath the capsule. The rest of the kidney was converted into a mass of dense fibrous tissue in which were scattered many uriniferous tubules, the epithelium of which was clear, nucleated, and of the embryonic type. The left kidney was the seat of chronic diffuse nephritis of the atrophic variety, with well-marked changes in the medullary portions. The cortex showed much exudation and less atrophy, being nearly normal in thickness. The small size of the organ was due chiefly to atrophy of the pyramids. The walls of the bladder were greatly hypertrophied, being in places one fourth of an inch thick. The urethra and prepuce were normal.

dilatation of the pelvis of the kidney has reached a sufficient size to form an abdominal tumour. In most of the cases in children this condition has been noted between the third and the eleventh years. This tumour may be situated in the lumbar region, or it may fill the abdomen. It is cystic, and may be confounded with a dermoid cyst of the ovary. On

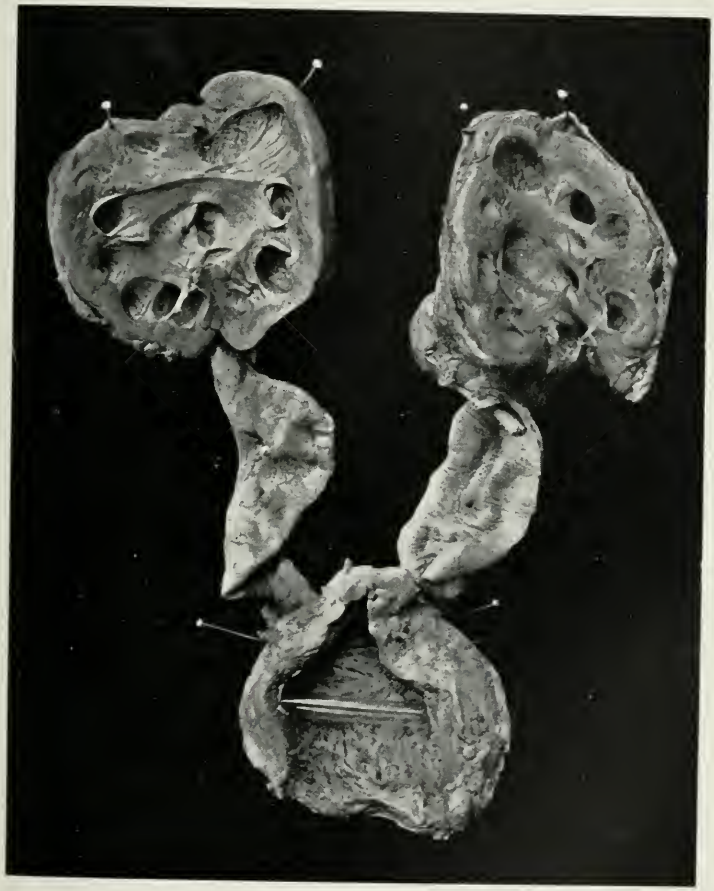


Fig. 105.—Congenital hydronephrosis, dilated ureters, and hypertrophied bladder. (From a child one month old.)

aspiration a fluid is withdrawn which may be clear, or of a brownish colour, and recognised as urine by the fact that it contains urates and urea. After aspiration the urine passed *per urethram* may be bloody. Aspiration affords only temporary relief, as the tumour quickly refills. If an incision is made and the kidney drained, a cure may result with the formation of a fistula. This may continue indefinitely, or infection of the fistulous tract may occur and suppurative nephritis be set up, which

speedily carries off the patient. A better operation is nephrectomy, which may result in a permanent cure if the opposite kidney is healthy, which is usually the case if the child is over three years of age for the reason above stated, viz., that a child with malformation of both kidneys usually dies in infancy. Whether the other kidney is the seat of serious disease or not, will depend much upon how far advanced the changes are upon the side of the hydronephrosis. In most cases the sooner this condition is removed the better will be the outlook for the patient; hence the question of operation should always be carefully considered.

Supernumerary Ureters.—These were noted in four cases, more frequently on the left side. The usual deformity was for two ureters to be given off, one from the upper and one from the lower part of the kidney, each ureter having a separate pelvis. The ureters either joined just above the bladder, or entered this organ by separate openings. This condition is of no practical importance, and was not found associated with other renal changes.

Malposition of the Kidney.—This was noted in my series of autopsies only once, in a case of fusion of the kidneys already mentioned. Of twenty-one cases collected by Roberts, the displacement was always of one kidney only; the left being displaced fifteen times, the right six times. Northrup has reported two cases, both displacements of the left kidney; in one, the organ lay in the hollow of the sacrum; in the other, in the median line, partly above and partly below the promontory of the sacrum. Malpositions of the kidney are compatible with perfect health and development. In most of the cases there is no other deformity present.

Movable or Floating Kidney.—This is one of the rarest of the abnormal conditions seen in this organ in early life. Cases have, however, been reported by Phillips, Korsakow, and others, with symptoms similar to those seen in adult life.

URIC-ACID INFARCTIONS.

These consist in a deposit in the straight tubes of the kidneys of uric acid or of amorphous or crystalline urates; usually both kidneys are affected, and all the pyramids of each kidney. The infarctions appear to the naked eye as fine, brownish, fan-shaped striæ. Associated with them there may be granular deposits of uric-acid salts in the pelvis of the kidney, and sometimes evidences of catarrhal inflammation of the pelvis, including even the presence of blood. This condition probably occurs, to some degree at least, in nearly all infants during the first ten days of life. It was formerly supposed that the discovery of these appearances was proof that an infant had breathed, and a certain medico-legal importance was therefore attached to them. This is now known not to be the case, as they are sometimes found in still-born infants.

The cause of this condition is the excretion of uric acid before there is

sufficient water to dissolve it, so that the crystals are deposited in the tubes. Uric-acid infarctions are found chiefly in children dying before the end of the second week, although it is not uncommon to see them as late as the third or fourth or even the sixth month. In most of the cases, as the urinary secretion becomes more abundant, the deposits are washed out in the urine and appear as brownish-red stains upon the napkins. Infarctions may give rise to a slight inflammation of the renal tubules, but very rarely to any serious lesion; sometimes they remain as deposits in the calices or the pelvis of the kidney or in the bladder, forming the nucleus of a calculus. The symptoms to which they give rise are mainly scanty urination during the first week of life, and occasionally anuria for the first day or two. Sometimes there is evidence of pain on micturition, and there is the stain upon the napkin already referred to. The treatment is to give water freely and some alkaline diuretic such as citrate of potash. One grain should be given every two hours until the secretion is fully established; this in most cases will be within twenty-four hours.

ACUTE CONGESTION OF THE KIDNEY.

In acute congestion of the kidney all its blood-vessels contain much more blood than normal, and from them there may be an escape of serum and even of the red blood-cells by diapedesis. This congestion may result from traumatism, the ingestion of certain poisons, from any of the infectious diseases, or from cold.

The urine is usually scanty, of high specific gravity, and contains albumin and red blood-cells, sometimes blood casts. This may be only a temporary condition passing off in a few days without further symptoms, or it may exist as the first stage of acute nephritis. It is most serious when it occurs in kidneys already the seat of serious disease. There are sometimes no symptoms except those of the urine; or there may be headache, pain in the back, and some general indisposition.

The treatment consists in free catharsis, the use of hot vapour baths, and counter-irritation over the kidneys by means of hot poultices or dry cups.

CHRONIC CONGESTION OF THE KIDNEY.

This results from interference with the return circulation of the kidney, and may be caused by congenital malformation or valvular disease of the heart, chronic broncho-pneumonia or chronic pleurisy; also by the pressure of any abdominal tumour upon the inferior vena cava or the renal veins.

The kidneys are generally enlarged, firmer than normal, and dark-coloured. All the capillary vessels are swollen and distended with blood, and their walls are thickened. In addition to the symptoms of the pri-

mary disease, the amount of urine passed is usually scanty and of high specific gravity. Albumin and casts are generally present, but are not constant. The treatment should be directed toward the primary condition, and, in addition, an effort should be made to increase the urine by alkaline diuretics, caffein, digitalis, and the sweet spirits of nitre.

ACUTE DEGENERATION OF THE KIDNEYS.

In the succeeding pages devoted to diseases of the kidney I shall follow the classification of Delafield, which seems to me the simplest and most exact that has yet been proposed. For the description of the lesions I am indebted largely to his Lectures.

In acute degeneration of the kidney the principal or only change is in the epithelium of the tubules. It is exceedingly common both in infancy and in childhood, being found to a greater or less degree in all autopsies upon patients dying of acute infectious diseases, but it is most marked in cases of scarlet fever, diphtheria, and acute pleuro-pneumonia. It may be found in any disease characterized by prolonged high temperature; and it is the explanation of the cases of so-called febrile albuminuria. The cause is in all probability direct irritation of the epithelium of the tubules by the toxines eliminated by the kidneys. It may also be induced by irritating drugs, such as cantharides or turpentine. By some writers these cases have been classed as examples of acute nephritis; hence the great discrepancy which exists in statements made as to the frequency of nephritis in the different infectious diseases.

The kidneys are usually slightly enlarged, and paler than normal. On section the cortex may be somewhat thickened, and the straight tubules marked by yellowish-gray lines. It is the appearance commonly spoken of as "cloudy swelling." The organs are seldom much congested. The microscope shows a granular degeneration and death of the epithelium of the tubules, and when severe this may be accompanied by congestion and the exudation of serum.

Acute degeneration of the kidneys gives rise to no symptoms in addition to those of the original disease, except the appearance of a moderate amount of albumin in the urine, and sometimes a few hyaline or granular casts. It can not be said that such a condition adds much to the danger of the original disease. In cases that recover, the condition of the kidney entirely clears up. The development of the symptoms of degeneration of the kidneys in infectious diseases calls for no special treatment beyond a continuance of the fluid diet.

ACUTE EXUDATIVE NEPHRITIS.

Synonyms: Acute parenchymatous nephritis, acute desquamative nephritis, acute septic interstitial nephritis.

Etiology.—This variety of nephritis occurs apparently as a primary disease both in infants and in older children. Most such cases are undoubtedly of infectious origin, although the point of entrance of the infection it may be difficult or impossible to determine. This form of inflammation is much more frequently secondary to the acute infectious diseases, especially to scarlet fever and diphtheria. It occasionally follows measles, varicella, empyema, typhoid fever, acute diarrhœal diseases, pneumonia, meningitis, influenza, and, in rare instances, eczema. This is the characteristic variety of secondary nephritis occurring in septic conditions. The exciting cause of the inflammation is in some cases the irritation from toxines; in others there is in addition the entrance of pyogenic germs, carried by the circulation.

Lesions.—This inflammation is characterized by congestion and exudation of the blood plasma with leucocytes and red blood-cells, also by changes in the renal epithelium and the glomeruli. In infants and young children the predominant feature of the lesion is usually the exudation of leucocytes. In severe cases the kidneys are enlarged, and usually soft and œdematous. The cortex, which is the seat of the most marked changes, is thickened and of a uniform yellowish-white colour, or it may be mottled with red, owing to small hæmorrhages. Sometimes there is congestion of the entire organ. At other times, both on the surface and on section, the kidney presents a mottled yellow appearance, these yellow spots being aggregations of pus cells; they are scattered through the organ, and vary in size from a pin's head to a pea. Minute abscesses may even be found. The microscope shows the renal epithelium of the tubules to be swollen, loosened, and degenerated. The tubules may be dilated, and contain red and white blood-cells and degenerated epithelium. The glomerular changes are often marked. There are swelling and proliferation of the cells covering the capillary tufts, and similar changes in the capillaries themselves. There may be red or white blood-cells in the cavities of the capsules, and cocci may be found in the small blood-vessels. There are accumulations of leucocytes in the tubes, in the stroma, and in the venous capillaries. These cells are usually in irregular patches. The excessive emigration of leucocytes may not be accompanied by blood serum, and hence there may be no albumin in the urine.

I have made autopsies upon six cases of nephritis of this variety in young infants, which were apparently primary. In all these cases the excessive exudation of leucocytes was the striking feature of the disease.

Under the microscope they were in places so dense as to obscure all the renal elements.

Symptoms.—1. *Primary form in infants.*—These cases are not common, and the symptoms are so obscure that they are usually overlooked. In 1887 * I published five cases of my own, and collected from literature fourteen others of primary nephritis under two years of age. Since that time four additional cases have come under my observation.

A study of these cases yields the following facts: The onset in nearly every instance was abrupt, usually with high fever and vomiting, the temperature being in several cases over 104° F. Dropsy was very exceptional, being noted in but six cases; in most of these it was slight, and seen only toward the close of the disease. Fever was present in all cases. In those observed by myself it was high and irregular in type, ranging from 101° to 105° F. The duration of the disease was from eight days to four weeks, the average being about two and a half weeks. Vomiting and diarrhœa were noted in half the cases, but were rarely prominent, and marked either the onset of the attack, or were traceable to indigestion accompanying the fever; very rarely did they exist as symptoms of uræmia. Anæmia was a prominent symptom in nearly every case, and it was this which enabled me in several instances to make a correct diagnosis. Nervous symptoms were usually prominent. In several patients there was dyspnœa without pulmonary disease, partly due, no doubt, to the anæmia. In nearly all cases there was marked restlessness or muscular twitchings, and in three there were convulsions. Dulness and apathy were present in the majority of the fatal cases, but deep coma was never seen. Several patients presented the typical symptoms of the typhoid condition. The urine was rarely scanty until near the close of the disease, and sometimes not even then. Suppression of urine occurred in but a few cases. Albumin was frequently absent early in the attack, but was invariably present at a late period, although rarely in large amount. Casts were found in all cases that were carefully examined microscopically. They were not usually numerous, and were chiefly of the hyaline, granular, and epithelial varieties. No blood casts were seen. There were usually many pus cells and renal epithelial cells, together with red blood-cells in moderate numbers.

Of the twenty-three cases, fifteen died and eight recovered. Of my own nine cases, eight were fatal, the diagnosis being confirmed by autopsy in every case but one. Whether these figures represent the actual mortality of the disease it is difficult to say. No doubt there are many mild cases which escape notice altogether. The severe ones, however, are quite uniformly fatal, chiefly on account of the tender age of the patients.

2. *Primary form in older children.*—This also is a rare form of renal

* Archives of Pædiatrics, vol. iv, pp. 1, 103; and ix, p. 263.

disease. As compared with the same condition in infants, the onset is usually less abrupt, the febrile symptoms are less marked, and the termination is less frequently fatal. There is little dropsy, often none at all. The urine is only slightly diminished in quantity; the amount of albumin is small; casts are not numerous, and usually hyaline, epithelial, or granular; very rarely is there much blood present. Uræmia is very infrequent, and the prognosis is much more favourable than in infancy.

3. *Secondary form.*—This is the most common variety of secondary nephritis of infectious diseases. It usually occurs at the height of the febrile process, and its severity is generally proportionate to the intensity of the infection. The constitutional symptoms are often not marked, and dropsy is rare. Unless the urine is examined the condition may be overlooked. The urinary changes are essentially the same as those already mentioned in the primary cases. While the involvement of the kidneys adds to the danger of the primary disease, it is rare that the nephritis is itself the cause of death. Suppression of urine and the development of the symptoms of acute uræmia are infrequent.

ACUTE DIFFUSE NEPHRITIS.

Synonyms: Acute Bright's disease, glomerulo-nephritis.

This is a more severe form of inflammation than is exudative nephritis, and is much more likely to be followed by permanent damage to the kidney.

Etiology.—Acute diffuse nephritis occasionally occurs in children apparently as a primary disease, its origin being then obscure. It is usually attributed to cold and exposure, and certainly this is sometimes the case. It is the secondary form which is especially important in early life, and in the great majority of cases this follows scarlet fever. It is the characteristic post-scarlatinal nephritis. Occasionally, however, it follows diphtheria, and may indeed occur after any severe form of infectious disease. The cause in the scarlet-fever cases is now generally admitted to be the poison of the primary disease—probably the result of direct irritation from toxines. While it may sometimes follow a definite exposure, as when patients have been allowed to get up or go out too soon, it occurs also in those who have been kept in bed throughout the attack; sometimes even in mild cases. But there is little doubt that exposure may precipitate an attack in a patient who might otherwise have escaped. An important etiological factor is the too early use of solid food. The frequency of nephritis as a sequel of scarlet fever varies much in different epidemics; in some it is rarely seen, while in others it may occur in nearly half the cases; the average is probably from six to ten per cent. While it most frequently follows a severe form of scarlet fever, it may occur after an attack which has been so mild as to escape notice until the appearance of

desquamation. Season appears to have but little influence upon its frequency.

Lesions.— In this form of inflammation most of the changes of acute exudative nephritis are present, but in addition there are marked alterations in the stroma of the kidney and the Malpighian bodies. The kidneys are enlarged, often considerably so, and appear rather soft and flabby. In the early stage they are sometimes much congested; later, they are of a yellowish-white colour with a fine red mottling. The cortex usually appears much thickened and yellow, while the pyramids are red. The characteristic lesions of this form of nephritis are a production of connective-tissue cells in the stroma, and proliferation of the cells forming the capsules of the Malpighian bodies. These changes usually occur in patches. In recent cases there are found only the new connective-tissue cells; in older ones the connective tissue is more dense and even fibrous in character. The changes in the glomeruli may be permanent, the tufts being compressed by the growth of the endothelial cells lining the capsules, which may ultimately form new fibrous tissue.

Symptoms.—When the disease is primary, it may begin abruptly with febrile symptoms, dropsy, headache, lumbar pains, scanty urine, and often with vomiting; or it may come on somewhat insidiously with few constitutional symptoms, but with dropsy and changes in the urine. When it follows scarlet fever it most frequently develops during the third or fourth week of the disease. The onset is usually gradual, with dropsy, scanty urine, and moderate fever. The subsequent course may be the same in both the primary and secondary cases, whatever the mode of onset.

There is in most cases some fever; usually the temperature ranges from 100° to 101·5° F., but in very severe attacks it may be 104° or 105° F. Dropsy is almost invariably present, and is generally marked. It is first seen in the face, next in the feet, legs, and scrotum, and there may be general anasarca, with dropsy of the serous cavities of the body; this is usually of the pleura or the peritonæum, rarely of the pericardium. As the disease progresses there is always a very marked degree of anæmia.

The urine is, as a rule, greatly diminished in quantity, and may be suppressed. Albumin is invariably present, and usually in large amount, often enough to render the urine solid upon boiling. The urine is of a dark, reddish-brown or smoky colour, owing to the presence of red blood-globules or hæmoglobin. The amount of urea eliminated is far below the normal. The specific gravity may be low, even though the quantity is very small. Casts are present in great numbers—chiefly hyaline, granular, and epithelial casts from the straight tubes; not infrequently there are blood casts. Occasionally twisted or cork-screw casts are seen. These come from the convoluted tubes, and are regarded by Ripley (New York) as of grave significance, indicating that all parts of the kidney are

involved. Red blood-cells are present in great numbers; also many leucocytes, and always a large amount of renal epithelium.

The duration of the active symptoms in cases terminating in recovery is from one to three weeks. The temperature and dropsy gradually subside. Improvement in the urine is shown by an increase in quantity, by increased elimination of urea, and by a diminution in the amount of blood, albumin, and the number of casts. A few casts may persist for several weeks, and a small amount of albumin for two or three months.

In the graver cases, where the onset is accompanied by high temperature, pain in the back and loins, and a rapid, full pulse of high tension, the urine is very scanty and is often suppressed. Then follow the symptoms of uræmia. In children this is usually manifested by vomiting, great restlessness or apathy, and often by diarrhœa. Less frequently there are headache, dimness of vision, stupor developing into coma, or convulsions. If the secretion of urine is re-established, the nervous symptoms abate and the patient may recover. This has been known to occur after complete suppression has lasted thirty-six hours. Care should be taken not to mistake retention for suppression. If doubt exists, percussion of the bladder and the use of the catheter will quickly settle the question.

There are several complications for which the physician must constantly be on the lookout during attacks of acute nephritis; the most frequent are pneumonia, pleurisy, pericarditis, and endocarditis; more rarely there may be meningitis and œdema of the glottis. It is from complications or acute uræmia that death usually occurs.

Prognosis.—This is to be considered from two points of view: first, the danger to life during the acute stage of the disease, and, secondly, the danger of the development of chronic nephritis. The great majority of patients survive the acute stage, and not infrequently even those recover who have presented grave symptoms of uræmic poisoning. The quantity and specific gravity of the urine, and the number and variety of the casts, are a much better guide in prognosis than the amount of albumin. The existence of severe nervous symptoms, such as stupor, intense headache, dimness of vision, and persistent vomiting, add much to the gravity of the case, as does also the presence of any serious complication. In general it may be said that if there is no suppression of urine, or if there are no symptoms of uræmia and no complications, recovery is almost certain if the child is over three years old; in younger children the outlook is less favourable. The general opinion prevails that acute diffuse nephritis in childhood, whether it is primary or occurs as a complication of scarlet fever, is rarely followed by the chronic form of the disease; and such was the view I formerly held. Larger experience, however, has convinced me that this sequel is not very uncommon. The interval of apparent health may sometimes cover a period of several years, and the later nephritis may be attributed to other causes; but all cases of severe scarlatinal ne-

phritis should be carefully watched for a long time, and after a severe attack a guarded prognosis should always be given as regards the ultimate result.*

Treatment of Acute Nephritis.—Prophylaxis is important, and relates principally to the secondary form which occurs in the course of infectious diseases, especially post-scarlatinal nephritis; † but the measures here outlined apply equally to all varieties. The inflammation of the kidney being in most of these cases the result of direct irritation by the toxines which are eliminated by them, it follows that elimination through the skin and intestines should be increased, and that the urine should be rendered as little irritating as possible by largely increasing its quantity. The first indication is met by frequent sponging, warm baths, and keeping the bowels freely opened by saline cathartics, sufficient being given to produce one or two loose movements daily. To meet the second indication, the patient should be kept upon a fluid diet, preferably milk, at least for the three weeks of the disease, and, if possible, for a full month. At the same time he should drink very freely of alkaline mineral waters, or of plain water to which a small dose (two or three grains) of some alkaline diuretic like the citrate of potassium has been added. If milk is not well borne, kumyss, whey, buttermilk, or junket may be used, or thin gruels mixed with milk. When the first trace of albumin appears in the urine this plan of treatment should invariably be followed. In addition to these measures, after an attack of scarlet fever the patient should be kept in bed for at least a week after the temperature has become normal.

The mild cases of acute nephritis tend to spontaneous recovery under the hygienic and dietetic treatment mentioned—i. e., rest in bed, fluid diet, the drinking of large quantities of water, and attention to the action of the skin and bowels. These measures should be continued so long as the urine contains any considerable amount of albumin, or so long as the patient's general condition will permit. Should he become very anæmic, or lose much in weight, it may be necessary to enlarge the diet by the addition of solid food. This should at first be of the carbohydrates only, usually in the form of some farinaceous food. An increase in the diet and exercise should be made very gradually, and the effect upon the urine carefully watched.

---

* The following case may be cited as an illustration of this point: A girl at the age of seven years had scarlet fever, followed by nephritis; the dropsy having lasted, it was reported, for three months. She was believed to have recovered perfectly, and remained in apparent health until she was sixteen, when, as a supposed result of a severe chilling, she developed dropsy and all the symptoms of acute nephritis. From that time, although she lived for three years, and was often for months at a time seemingly in the best of health, her urine was never free from casts and albumin, and she finally died in uræmic convulsions.

† See W. H. Flint, New York Medical Journal, January 6, 1894.

The severe cases, with scanty urine, fever, and marked dropsy, require more active treatment. Free diaphoresis should be maintained by the hot pack or vapour bath (page 54), and in bad cases even pilocarpine may be used hypodermically, a dose of gr. $\frac{1}{60}$ being given to a child of three or four years. To counteract the depressing effects of this drug, stimulants should be given at the same time. Active counter-irritation should be maintained over the kidneys by dry cups followed by poultices, or the mustard paste. Two or three loose movements from the bowels should be secured by the administration of calomel, or, better, by Rochelle, or Epsom salts. Harm is sometimes done by carrying this depletion too far, and its effect upon the patient's general condition must be closely watched. If suppression of urine occurs with the development of uræmic symptoms—delirium, high temperature, flushed face, vomiting, and a pulse of high tension—nitroglycerin may be given; a child of five years may take gr. $\frac{1}{300}$ every hour for three or four doses, or until an effect is produced. Uræmic convulsions may often be averted by the use of morphine hypodermically; but if the symptoms are very urgent, nothing is so rapid or so certain to give relief as venesection. This has lately been revived in the practice of New York physicians, and has now the endorsement of the best practitioners in the city. From a child of five years from two to six ounces of blood may be taken, according to the general condition and the urgency of the symptoms. Even though the improvement which follows bleeding under the conditions mentioned is very certain, it is often only temporary; but it gives time for the use of other measures, such as catharsis and diaphoresis. The depressing effects may be largely overcome by following the venesection by an intravenous injection of a saline solution (gr. iv to water ℥ j). The amount introduced should be nearly twice that of the blood taken.

One should always be on the lookout for complications, especially dropsy of the serous cavities, pericarditis or endocarditis, and œdema of the lungs. Convalescence is nearly always slow, and a patient who has suffered from nephritis needs careful attention for a long time. Anæmia is always present, and iron is required. The diet must consist largely of fluids for several months. If the disease tends to pass into a subacute form, the child should, if possible, be sent to a warm climate, and kept there during the succeeding winter, and every means taken to build up the general nutrition. Flannels should be worn next to the skin, and every precaution taken against any exposure which might cause an exacerbation of the disease.

CHRONIC NEPHRITIS.

Chronic inflammation of the kidney is an infrequent condition in childhood. In infancy it is almost unknown, except in connection with congenital hydronephrosis or other malformations of the kidney. Two

pathological varieties are met with: (1) Chronic diffuse nephritis with exudation, known also as the large white kidney, chronic parenchymatous nephritis, and waxy kidney. (2) Chronic diffuse nephritis without exudation, known also as interstitial nephritis, granular kidney, and contracted kidney.

Etiology.—Chronic nephritis is most frequently seen as a sequel of the acute nephritis of scarlet fever. It also occurs with the prolonged suppuration of chronic bone or joint disease, where it may be chronic from the beginning. The only other important causes in early life are hereditary syphilis, alcoholism, chronic tuberculosis, and valvular disease of the heart. Nearly all the cases occur in children over seven years of age.

Lesions.—The lesions of chronic nephritis in childhood do not differ essentially from those seen in later life. In *chronic diffuse nephritis with exudation*, the kidneys are usually enlarged, the surface is smooth or slightly nodular, and yellowish-white on section. The microscope shows that the renal epithelium is swollen, granular, fatty, and degenerated. The tubes contain cast-matter and the detritus of broken-down epithelial cells. In some places they are dilated, in others atrophied. In the glomeruli there is a growth of capsule cells, compression and atrophy of the tufts, with the formation of new connective tissue. When there is waxy degeneration, the kidneys are usually considerably enlarged, and of a glistening gray colour. Amyloid degeneration is seen especially in the small arteries of the kidney and the capillary vessels of the tufts. With iodine the mahogany-brown reaction is obtained. Amyloid changes in the kidney are nearly always associated with similar lesions in the liver and spleen, and sometimes also in the intestinal villi.

In the *chronic diffuse nephritis without exudation* (granular kidney) the organs are smaller than normal, with a nodular surface and adherent capsule. The cortex is thinned, and the colour is gray or red. In addition to the lesions found in the preceding variety, there is an extensive production of new connective tissue, which is irregularly distributed throughout the kidneys. The tubules in some places are dilated to form cysts of considerable size, while in others they have completely disappeared. The glomeruli may be atrophied to little fibrous balls, but if chronic congestion has preceded the inflammation, they may be large and the capillaries dilated.

Symptoms.—1. *Chronic nephritis with exudation.*—This form of disease is not usually chronic from the outset, but follows an acute attack from which the patient is often supposed to have recovered completely. The symptoms sometimes immediately follow the acute attack; at others there is an interval of apparent recovery, extending over a few months or even years. Very rarely no such history of an antecedent acute attack can be obtained, and the symptoms come on gradually and insidiously. Such

cases occur chiefly in older children, and their clinical features do not differ essentially from those of adult life.

As a rule dropsy is present, although it is variable in amount, and fluctuates considerably from time to time. There may be not only œdema of the cellular tissue, but effusion into the pleura, peritonæum, and even the pericardium. As the case progresses, anæmia is always a marked symptom. There are various disturbances of digestion—loss of appetite, occasional vomiting, and attacks of diarrhœa. From time to time nervous symptoms may be quite prominent, such as headaches, sleeplessness, neuralgia, fatigue upon slight exertion, and dyspnœa. Attacks of epistaxis are not infrequent.

The urine contains albumin and casts nearly all the time. They vary much in amount at different periods in the disease, according to the rapidity of its progress. During periods of exacerbation, both albumin and casts are very abundant, while in the intervals the amount of albumin is small and the casts few. The casts are hyaline, granular, epithelial, and fatty. The daily quantity of urine is much reduced during the periods of exacerbation, while at other times it may be nearly normal. The specific gravity is usually low.

If waxy degeneration is present, there are generally associated with the renal symptoms, others dependent upon waxy changes in other organs. The spleen and liver are enlarged; there may be ascites and diarrhœa, and there is usually present the peculiar "alabaster cachexia."

The duration of this form of chronic nephritis depends much upon the surroundings of the patient and the treatment. It is rarely shorter than two years, and it may last for many years. The progress is always irregular, and marked by periods of exacerbation and remission. The patients die from acute uræmia, or from complicating pneumonia, pleurisy, pericarditis, endocarditis, or from pulmonary œdema.

2. *Chronic nephritis without exudation.*—This is a very rare disease in early life, being much less frequent even than the preceding variety of nephritis. In some cases there is a history of hereditary syphilis; in others, of chronic alcoholism. The early symptoms are few, and the disease usually develops insidiously. The urine is pale, excessive in amount, and of low specific gravity—1·001 to 1·008. Albumin is more often absent than present, and, when found, the quantity is small. Dropsy likewise is rare, and never marked. Nervous symptoms are often prominent, such as headaches, attacks of spasmodic dyspnœa resembling asthma, neuralgias, and disturbances of vision. High arterial tension and hypertrophy of the left ventricle are regular symptoms; and even atheromatous degeneration of the arteries may be present. Dickinson reports an instance of this in a patient only six years of age. Late in the disease, hæmorrhages may occur, and these may be the cause of death. Filatoff has reported a cerebral hæmorrhage in a child of eleven.

Acute uræmia is, however, the usual termination of this form of nephritis. The course is slow, and the disease may be overlooked until the final uræmic symptoms occur.

Prognosis.—The prognosis of chronic nephritis as to complete recovery, is always unfavourable; and although cases are seen in which symptoms are absent for several years, they almost invariably return. Cases have been reported of recovery from waxy degeneration of the kidney after removal of the bone disease upon which the condition depended. Although symptoms may be absent for a long time, complete recovery is very doubtful. An extended period of observation is necessary before the patient can be pronounced cured. As to the duration of the disease, no exact prognosis can be given because, from the symptoms, it is difficult or impossible to determine exactly the extent of the disease in the kidney and the rapidity of its progress. According to Delafield, the continued passage of a large amount of urine of low specific gravity is invariably to be interpreted as evidence of fibroid changes in the Malpighian tufts, and is a bad symptom. A large amount of dropsy, the coexistence of valvular disease of the heart, and marked renal insufficiency, as shown by a quantitative examination of the urine, are all very unfavourable symptoms.

Diagnosis.—Chronic nephritis like the acute forms is likely to be overlooked because of the failure to examine the urine in children. Regular and frequent examinations should be made in all cases of convulsions, of persistent or frequent headaches, severe anæmia, hypertrophy of the heart, high arterial tension and of general malnutrition, as well as when the more obvious symptoms of renal disease, such as dropsy and scanty urine, are present. Nor should one be too ready to make the diagnosis of functional albuminuria because he finds albumin only occasionally and in small quantity. All such cases demand most careful observation and the closest attention for a long period before excluding organic renal disease.

Treatment.—Children with chronic nephritis are to be treated on the same general plan as adults. The purpose of treatment is to retard as much as possible the progress of the disease and to relieve the symptoms as they arise. It is of the greatest importance to remove the patient from conditions in which exacerbations are liable to occur. If it is possible, he should be sent to a warm, dry climate in winter, and all exposure to cold avoided; an out-door life is desirable. Most patients require a general tonic treatment with very moderate but regular exercise, never carried to the point of fatigue, as much rest as possible in a recumbent position, a fluid diet, consisting largely of milk as long as this can be borne, and the administration of iron, particularly the tincture of the chloride. Excessive dropsy calls for diuretics, saline cathartics, and heart stimulants. If uræmia develops, with high arterial tension and stupor, headache, and convulsions, venesection should be resorted to, or nitro-

glycerin used. Morphine may be given hypodermically if the pupils are dilated and nervous symptoms are very marked.

TUBERCULOSIS OF THE KIDNEY.

In general tuberculosis, miliary tubercles are frequently seen both upon the surface of the kidney and in its substance. These give rise to no symptoms and are of no clinical importance. Larger tuberculous deposits are extremely rare in early life. They usually occur in patients who are the subjects of general tuberculosis, and are associated with tuberculosis of other parts of the genito-urinary tract; or they may exist as the primary, and even the only, tuberculous lesion in the body. At least two such cases are on record in children, one reported by West and the other by Rilliet and Barthez. Infection of the kidney generally takes place through the circulation, and not from the bladder. Aldibert's figures show that in children the bladder usually escapes even when the kidneys are tuberculous, for of thirteen cases of renal tuberculosis the bladder was involved in but two. The ages of twelve of these patients were as follows: from two to four years, four cases; from seven to eleven, five cases; from eleven to fourteen, three cases. The disease probably begins in the mucous membrane of the pelvis and the calices of the kidney, and extends to the pyramids, finally involving the cortex. As a rule, but one kidney is affected. The process may be confined to the pyramids, where are found cheesy nodules which may be single or multiple. These ultimately break down and form abscesses. The process may result in almost complete destruction of the pyramids, and even of portions of the cortex, so that the kidney may consist of a mere shell of renal tissue. Suppuration in the neighbourhood of the kidney (perinephritic abscess) often coexists.

The symptoms are quite indefinite. There may be localized pain and tenderness in the region of the kidney, and a tumour if there is perinephritis. The symptoms of irritability of the bladder may be almost as severe as in cases of calculus. Pus appears in the urine usually as a constant symptom; but the only thing that is diagnostic is the discovery of tubercle bacilli in the urine.

The treatment of renal tuberculosis is purely surgical. Of the thirteen cases collected by Aldibert in which nephrectomy was done for this condition, there were nine recoveries and four deaths; two of the deaths, however, not being traceable to the operation or to the original disease. No recurrence had taken place in one case at the end of eight years, and none in another after three years.

MALIGNANT TUMOURS OF THE KIDNEY.

In the great majority of cases tumours of the kidney are malignant. Of fifty-one cases collected by Aldibert which were operated upon, forty-eight were malignant and three benign.

Malignant growths are almost invariably primary. In children under five years, although not common, they are yet more frequent than any other variety of malignant tumour of the abdomen. The earlier cases reported were classed as carcinoma. It is now well established that carcinoma is very infrequent, and that nearly all the cases are varieties of sarcoma. Fischer reports nineteen of sarcoma and two of carcinoma; Aldibert, thirty-eight of sarcoma and five of carcinoma. The sarcoma may be round- or spindle-celled, or myo-sarcoma. In some of the cases there are both sarcomatous and carcinomatous features, so that they might be classed as sarcomatous carcinoma. The tumour grows from the cortex of the kidney, or from the pelvis, sometimes from the adrenals. It may infiltrate the whole kidney, so that there is no trace of renal structure remaining, or it may form an immense tumour on one side of the kidney, which is only partially invaded. These tumours are very rarely cystic, but they are quite soft, and hæmorrhages often occur into their substance. Secondary growths may occur in the liver, the lungs, the retro-peritoneal glands, in the opposite kidney, in the intestines, or in the pancreas. Pressure of the tumour upon the ureter may lead to hydronephrosis; and upon the inferior vena cava, to thrombosis of that vessel. As it grows, the tumour sometimes becomes adherent to nearly all the abdominal organs by localized peritonitis. It may lead to ascites, but it very rarely causes general peritonitis. The growth may reach a great size, usually from five to fifteen pounds, but in one case reported by Jacobi it weighed thirty-six pounds. In Seibert's collection of 48 cases the right kidney was involved in 24, the left in 22, and both kidneys in 2 cases.

Etiology.—These tumours of the kidney may be congenital. This was true of 5 cases in a series of 55 collected by Jacobi. The majority occur in early childhood. In the collection of 130 cases by Longstreet Taylor in which the ages are given, 106 were in the first five years, and 57 of these in the first two years of life. The sexes were about equally affected. In a small number of cases the history of a fall was given.

Symptoms.—The principal symptoms are tumour, hæmaturia, and cachexia. The tumour is usually first noticed. It is in most cases discovered in the loin, but grows forward toward the median line. Its surface may be lobulated and irregular or quite smooth; and although solid, it is sometimes so soft as to give an obscure sensation of fluctuation. It may grow to an enormous size, causing displacement of the liver, spleen, intestines, and lungs. The progress of the growth is usually rapid, so that from the size of a fist, the tumour may grow in the course of three or four months so as to fill the abdomen. By careful palpation it will be found—certainly when the tumour is small—that although it may be quite freely movable, its attachment is near the lum-

bar spine. Aspiration may show blood, but more frequently the result is negative.

Hæmaturia was observed before the tumour in 19 of 50 cases (Seibert), it being then the first symptom noticed. The amount of blood passed is sometimes quite large, but is usually small, and may be discovered only by the microscope. Pain is rare, and is due to localized peritonitis. Constitutional symptoms are absent until the tumour has attained a large size, when a cachexia develops and the patient wastes steadily while the tumour continues to grow. The pressure effects are dyspnœa, from compression of the lungs; œdema of the lower extremities, from pressure upon or thrombosis of the vena cava; vomiting and indigestion, from pressure upon the stomach and intestines. Secondary deposits very rarely cause any symptoms except in the lungs, where they may give rise to cough, and even to hæmoptysis.

The course of the disease is steadily from bad to worse. The usual duration of life in patients not operated upon, is from three to ten months after the tumour is discovered; very rarely do they live a year, death usually occurring from exhaustion.

Diagnosis.—The diagnosis of sarcoma of the kidney is usually quite easily made from the position and attachment of the tumour, its rapid growth and solid character, the existence of hæmaturia, and the age of the patient (under five years). It may be confounded with hydronephrosis, dermoid cyst of the ovary, enlargement of the spleen, retro-peritoneal sarcoma, tumours of the liver, or even of the abdominal wall.

Treatment.—Nothing is to be said regarding the medical treatment of these cases. Unless operated upon, I believe they invariably terminate fatally. The results of operation during recent years have been so encouraging that no case should be abandoned, no matter how young the patient. Aldibert has collected the results of forty-five cases operated upon: twenty deaths occurred soon after the operation, two thirds of them from shock; in eleven cases recurrence of the growth occurred within nine months, and caused death. This raises the total mortality to 78 per cent. Recently, in the Babies' Hospital, two cases have been successfully operated upon by my colleague, Dr. Robert Abbe; one, a nursing child, thirteen months old, where the tumour weighed seven pounds, and the child after the operation only fifteen pounds. This case made an uninterrupted recovery, and three years after the operation was in perfect health. The accompanying illustrations (Figs. 106 and 107) are from photographs of this patient. The second case was in a child two years old, and the tumour weighed two and a quarter pounds. The child made an excellent recovery, and was in perfect health three years and nine months after the operation. These results certainly are encouraging, and show conclusively that infancy is no contraindication to the operation.

Fig. 106.—Sarcoma of the kidney, child thirteen months old.

Fig. 107.—The same child one year after operation.

For a discussion of the surgical aspects of this question, and details of the operation, see the papers of Abbe * and Aldibert.†

Benign Tumours.—These are distinguished by their slow growth, and by the fact that the constitutional symptoms are mild or wanting. Of the three cases collected by Aldibert, one was adenoma, one fibroma, and one was fibro-cystic. Two cases recovered, and one died of septic peritonitis. The duration of the disease was from twenty months to six years.

PYELITIS.

Pyelitis is an inflammation of the mucous membrane lining the pelvis of the kidney. It may exist alone, or with an inflammation of a portion of the ureter, or of the kidney itself (pyelo-nephritis); and it may be acute or chronic. It may result in an accumulation of pus in considerable quantity in the pelvis of the kidney (pyonephrosis).

Etiology.—Of local causes, the most frequent is irritation from renal calculi. It is also associated with congenital malformations of the kidneys or ureters, with renal tuberculosis and renal tumours. It may result from an extension of inflammation from the tissues surrounding the kidney (perinephritis), or from an abscess opening into the pelvis of the kidney. The secondary pyelitis, which so often follows cystitis in adults, is an extremely rare occurrence in childhood. In addition to the forms mentioned, there is seen an infectious form of acute pyelitis, which usually occurs as a complication of scarlet or typhoid fever, diphtheria, malaria, or pyæmia; but it is also seen apart from these diseases, when it occurs apparently as a primary affection. I have seen in infants three cases of this description. In this group of cases the infection is probably through the circulation, but in the cases which occur independently of the acute infectious diseases it may be impossible to determine the point of entrance of the infection. In most, if not all these cases there is also present a certain amount of nephritis.

Lesions.—When pyelitis develops from a local cause it is usually unilateral. In the infectious form both kidneys are involved. In the acute cases there are the usual appearances of an acute catarrhal inflammation of the mucous membrane, with congestion, swelling, and sometimes minute hæmorrhages. In chronic cases there is thickening and sometimes a granular condition of the lining membrane. There may be an accumulation of pus of considerable size, distending the pelvis and calices (pyonephrosis). If the condition is one depending upon a calculus or congenital deformity, and in all protracted and severe cases, the kidney itself is involved to a greater or less degree; the extent of the nephritis will depend upon the nature of the exciting cause and the duration of the process.

* Annals of Surgery, January, 1894.

† Revue Mensuelle des Maladies de l'Enfance, November, 1893.

Symptoms.—The history of the following case illustrates the main clinical features of acute infectious pyelitis, in this instance occurring apparently as a primary disease:

A previously healthy female infant of eight months was taken suddenly with a chill, followed by a very high fever. The child was ill for ten days before the nature of the disease was suspected. During this time the temperature ranged between 101° and 106° F., touching 105° nearly every day; but the chill was not repeated. The other constitutional symptoms were not severe. At the first examination of the urine there was found a large amount of pus, which on standing was equal to one twelfth of the volume of the urine passed; the reaction was strongly acid. There were no signs of vaginitis or vulvitis, no *ardor urinæ*, no evidence of local pain either in the bladder or kidney, no abnormal frequency of micturition, no localized tenderness, and no vomiting. At later examinations there were found in moderate numbers epithelial cells from the bladder, and the tubules and pelvis of the kidney, also a few hyaline casts, but not more albumin than would be explained by the amount of pus. Under no treatment except alkaline diuretics, the temperature gradually fell to normal and the pus steadily diminished in quantity, and at the end of five weeks had practically disappeared from the urine. A report sixteen months later stated that the child had remained well and entirely free from urinary symptoms.

In some cases there are recurring chills, with wide fluctuations in temperature; in others there may be only pyuria, with moderate fever and few other constitutional symptoms. If the disease complicates one of the acute infectious diseases, pyuria may be the only symptom. The urine in acute pyelitis is turbid from the presence of pus, the amount of which may be from one to fifty per cent of the volume of the urine. The quantity of urine is generally somewhat diminished, and it may be quite scanty. The reaction is usually acid, even though the amount of pus is large. Albumin is present in proportion to the amount of pus or the degree of nephritis. Red blood-cells are found under the microscope in most of the very acute cases, and may be in sufficient numbers to colour the urine. The pus cells in recent cases are usually well preserved, but in old cases they may be degenerated. There are many epithelial cells—conical, fusiform, and irregular cells with long tails. There may be renal epithelium and hyaline, granular or epithelial casts, varying in number with the severity of the nephritis. Bacteria also are found in great numbers.

In chronic pyelitis only pyuria may be present, or there may be a tumour owing to the pyonephrosis. From time to time in the chronic form there may be intermittent attacks of acute pyelitis resembling those above described. In pyelitis depending upon congenital malformations, pyuria is usually the only symptom, unless pyonephrosis is present. With

calculi we may have acute or chronic pyelitis; there may be localized pain, tenderness, sometimes a tumour, occasionally hæmaturia, and perhaps a history of renal colic or the passage of gravel. With tuberculosis we have chronic pyuria and the presence of tubercle bacilli in the urine. There are commonly associated the symptoms of general tuberculosis. If associated with perinephritis, the inflammation is usually acute, and there are present the local symptoms of the original disease. If an abscess opens into the pelvis of the kidney we may have a sudden discharge of pus in large quantity with a subsidence of previous local symptoms, including the tumour. With neoplasms we have congestion and hæmorrhage more frequently than pus, but both may be present.

Diagnosis.—The characteristic symptoms of acute pyelitis are a chill, which may be repeated, high and fluctuating temperature, scanty urine, frequently pain and tenderness over the kidneys, and pyuria. The diagnosis of pyelitis is made only by an examination of the urine, which should never be omitted in cases of obscure high temperature, even in infancy, particularly if chills are associated. Given the existence of a large amount of pus in the urine, it may be difficult to decide whether this comes from the bladder or the kidney. Pus from the bladder is exceedingly rare in children even when a vesical calculus is present. If the pus comes from the opening of an abscess into the bladder, ureter, or pelvis of the kidney, the local signs of such abscess will usually be present. The existence in an acid urine of a large amount of pus, many epithelial cells like those described, with high fever and chills, are generally sufficient to establish the diagnosis of pyelitis.

Prognosis.—In cases apparently primary, and in those complicating infectious diseases, the prognosis is good. The danger is chiefly from the nephritis which follows or complicates the process. In cases depending upon local conditions, the prognosis will depend upon the nature of the exciting cause. Here, also, the principal danger is from nephritis. If calculi are present and if pyonephrosis develops, the patient may die from exhaustion before a serious degree of nephritis has developed.

Treatment.—In all cases the diet should be fluid. Water should be given freely, and alkalies up to the point of neutralizing the excessive acidity of the urine. In infants, from twelve to twenty-four grains of the citrate of potash are required daily for this purpose. If the urine is alkaline, benzoic acid may be used in the same doses. In acute cases, counter-irritation over the kidney by means of poultices or dry cups may be employed. If calculi are present the same treatment is indicated. Surgical interference is called for if pyonephrosis develops, or if the disease is evidently unilateral and the kidney is becoming disabled. The advisability of surgical interference will depend upon the clearness and severity of the symptoms.

RENAL CALCULI.

Small renal calculi are very common in infancy. In the autopsy-room of the Babies' Hospital we frequently see, on opening the kidneys of young infants, fine brown granules in the pelvis and calices, and occasionally a calculus as large as a small pea is found. They are usually composed of uric acid. Only once in over one thousand autopsies of which I have records, was a stone of any considerable size seen in an infant. In this case it was an inch in length and half an inch wide. It is surprising that these are so rare, when we consider how very frequently the minute calculi are met with. The probable explanation is, that the majority of them have been dissolved or washed down into the bladder and passed *per urethram* because of the fluid diet of the first two years. The granular deposits are usually lodged in the pelvis of the kidney, and are generally seen upon both sides. With the larger collections there is often a slight catarrhal pyelitis.

Symptoms.—The small deposits give no symptoms, and even quite large calculi may be found at autopsy where no indication of their presence had existed during life, as in the case above mentioned. At other times symptoms are produced which resemble those of renal calculi in the adult.

There may be tenderness with pressure, pain localized over the affected kidney, or radiating to the bladder, the perinæum, and even the opposite kidney, and there may be irritation and retraction of the testicle. The urine may show, especially after exercise, a trace of blood; there may be the added symptoms of pyelitis, with some fever, localized tenderness, and the appearance in the urine of pus and epithelial cells from the pelvis of the kidney.

Renal colic is produced when a stone of any considerable size passes from the kidney to the bladder. It is characterized by symptoms similar to those seen in the adult. There are sudden attacks of severe sickening pain in the loins, shooting down the thigh or to the testicle. There may be vomiting and even collapse. The urine is passed frequently, in small quantities, and contains blood. The symptoms quickly subside when the stone reaches the bladder. The calculus may sometimes become impacted in the ureter and give rise to hydronephrosis or pyonephrosis, which soon becomes pyelo-nephritis.

Treatment—The treatment of renal calculi in children is to be conducted upon the same general principles as in adults. Small calculi may be suspected, but a positive diagnosis is impossible except by the passage of gravel in the urine. When these conditions exist the diet should be largely fluid, and alkaline waters freely given. When the calculi are large enough to give positive symptoms, which continue to increase in severity, a surgical operation should be considered, and it should be urged in propor-

tion to the severity of the symptoms and the clearness of the diagnosis. If calculous pyelitis exists, it is certain sooner or later to lead to serious nephritis, and it is only a question of time when the kidney will be disabled. The same is true of hydronephrosis from the impaction of a calculus in the ureter. Aldibert has collected four cases of nephrectomy in children for renal calculi in which the kidney was healthy, with three recoveries and one death from shock. In nine cases of operation for calculous pyonephrosis, there were six recoveries and three deaths. This is certainly an encouraging showing, and should lead one to consider operation seriously in many cases for which formerly nothing was done. The earlier the operation the greater the chances of success, because of the better condition of the other kidney. Although the continued use of water and the so-called solvents may relieve some of the symptoms, it is very questionable whether they do more.

TRAUMATIC HYDRONEPHROSIS.

In addition to the hydronephrosis which results from congenital malformations and from the impaction of calculi, a form is occasionally seen following severe injury to the kidney. The pathology of hydronephrosis in these cases is not well understood. After the early symptoms of traumatism have subsided, there develops in from two weeks to two months a tumour in the region of the kidney, which may reach a considerable size and present all the ordinary characteristics of hydronephrosis arising from other causes. This tumour may disappear spontaneously, or it may increase in size and demand surgical intervention for its cure. In seventeen cases which Aldibert has collected there was only one of spontaneous recovery; aspiration was done in seven cases, with six cures and one death; incision with or without nephrectomy was practised in nine cases, with seven recoveries and two deaths.

PERINEPHRITIS.

This consists in an inflammation in the cellular tissue surrounding the kidney, which may terminate in resolution or in suppuration. It is not of very uncommon occurrence, and is of importance chiefly from the frequency with which it is confounded with disease of the hip or spine. Perinephritis may be secondary to suppurative processes in the kidney itself, whether from calculi or tuberculous deposits, or it may be primary. In children the latter is the common form. Primary perinephritis is attributed to traumatism, cold, or exposure, or it may develop without assignable cause. It usually runs an acute or subacute course; very rarely it may be chronic.

For the clinical picture of this disease I am chiefly indebted to a paper by Gibney, who published in 1880 a report of twenty-eight cases of

primary perinephritis in children. I was at that time an interne in the Hospital for the Ruptured and Crippled, New York, where these cases were under observation, and had an opportunity to see many of those reported in Dr. Gibney's paper.*

The ages of these patients were between one and a half and fifteen years, the majority being between three and six years. The two sides and the two sexes were about equally affected. About one third of the cases were clearly traceable to traumatism; in the others no adequate exciting cause could be discovered. The majority of the cases were referred to the hospital with the diagnosis of hip-joint disease or caries of the spine. Resolution followed in twelve of these cases, and sixteen terminated in suppuration.

When abscess forms, it usually burrows between the lumbar muscles and comes to the surface posteriorly near the middle of the ilio-costal space; it may burrow forward between the abdominal muscles and point just above Poupart's ligament; very rarely it may follow the psoas muscle and appear at the upper and inner aspect of the thigh, like an ordinary psoas abscess; or it may open into the peritoneal cavity.

Symptoms.—The onset of acute perinephritis may be quite abrupt, with chill, fever, and localized pain; or it may be gradual, with stiffness of the spine, lameness referred to the hip, and deformity due to contraction of the flexors of the thigh. The pain is usually felt in the loin, but may be referred to the groin, to the inner side of the thigh, or to the knee. It is often severe, and increased by using the limb. It is in most cases accompanied by localized tenderness in the neighbourhood of the kidney. There is lameness upon the affected side which may come on gradually, being sometimes referred to the hip and sometimes to the spine. These symptoms often develop slowly in the course of two or three weeks. They are usually accompanied by a slight elevation of temperature. In the most acute cases the temperature is high (102° to 104° F.), and prostration severe.

As the disease progresses fever is a constant symptom, the temperature usually varying between 101° and 103° F. There is in most cases increasing deformity, and finally the patient may be unable to walk at all. On examination at the height of the disease there is found in a typical case a deviation of the spine with the concavity toward the affected side; the thigh may be held flexed to a right angle; passive extension is resisted and causes pain, although all the other movements at the hip joint are normal. In the lumbar region there is tenderness, and there may be an area of infiltration filling the ilio-costal space. At first this is only appreciable by percussion, but later a distinct tumour is present. In

* Chicago Medical Journal and Examiner, 1880, where will be found a very full bibliography.

addition to the tumour in the usual region, there is sometimes one at the upper and inner aspect of the thigh, owing to a burrowing of pus, and the sacs may communicate.

Lameness, pain, deformity, and fever sometimes exist for two or three weeks before any tumour can be made out. The constitutional symptoms are often severe, and symptoms of the typhoid condition may even be present. The bowels are usually constipated. The size of the abscess is sometimes very great. In one case I have seen it extend from the spine to the median line in front, and from the crest of the ilium nearly to the free border of the ribs. The amount of pus varies from a few ounces to two or three pints. Urinary symptoms are sometimes wanting; at other times there is increased frequency of micturition, accompanied by pain from an irritation referred to the bladder. The urine may contain pus from a complicating pyelitis. In only one of Gibney's cases was this present. It developed in the fourth week, and the case recovered.

The duration of the disease in the acute cases varies from three to eight weeks; in the subacute it may be five or six months. When suppuration occurs the symptoms subside quite rapidly after the pus has been evacuated, and recovery is complete. Where resolution takes place, there is a gradual subsidence of the symptoms, and often some stiffness of the thigh, with slight lameness for several months. In the series of cases above referred to, 65 per cent recovered completely in three months.

Diagnosis.—In many cases a diagnosis of hip-joint disease is made, and they are reported as "hip-joint disease cured without deformity," etc. The points of differential diagnosis are quite distinct, and if a careful examination is made there is no excuse for confounding the two conditions. Hip-joint disease develops more insidiously, is very much more chronic, and rarely produces so great deformity in a year as is often seen in perinephritis in two or three weeks; abscess is infrequent during the first year of the disease; on examination, there is found limitation of all the movements of the joint, and not of extension alone; atrophy of the thigh and joint tenderness are present. In perinephritis, on the other hand, we have a tolerably acute onset, sometimes with chill, fever, marked lameness, and deformity, developing in two or three weeks; abscess often forms in a month, and complete and permanent recovery usually follows after a few months at most; the deformity is due solely to flexion of the thigh; all other movements at the hip may be free, and joint tenderness is absent. Psoas abscess from Pott's disease may cause deformity, tumour, and lameness similar to that seen in perinephritis, but on examination there is found the angular prominence and other signs of disease of the lumbar vertebræ.

Prognosis.—Primary perinephritis in children almost invariably terminates in complete recovery. Of the twenty-eight cases referred to, and eight subsequently observed by Gibney, all recovered perfectly. The only

condition liable to prove fatal is rupture of the abscess into the peritoneal cavity.

Treatment.—The patient should be put to bed and kept as quiet as possible throughout the attack. In the early stage, a blister, hot fomentations, or an icebag, should be applied over the affected side; heat is generally to be preferred. When suppuration is inevitable and pain severe, a poultice may be used. Abscesses should be opened early, to prevent burrowing, and danger of a possible rupture into the peritoneal cavity.

GENERAL ŒDEMA NOT DEPENDENT ON RENAL DISEASE.

This is of not very infrequent occurrence in infants and young children. In the Babies' Hospital, during the last seven years, over fifty cases have been observed. Nearly all were in infants under six months of age, and the majority have been under three months. This general dropsy was invariably associated with extreme malnutrition and anæmia. It comes on gradually in the course of four or five days, often the first thing noticed being that a wasting child has unexpectedly increased half a pound or a pound in weight. On closer inspection there will be found œdema of the feet, ankles, thighs, face, hands, and sometimes of the abdominal walls, and the back. This may be quite marked, so that it may be almost impossible to open the eyes, and the extremities may be nearly double their normal size. I have occasionally seen dropsy in the serous cavities. No explanation of this œdema is found in the urine. It is not albuminous; it is frequently very scanty, but is sometimes apparently normal in amount. Opportunities for the examination of the kidneys have been afforded in several instances, and these organs have been in all cases normal, even upon microscopical examination.

The cause of this œdema was ascribed by Tarnier, who had observed it in connection with premature infants fed by gavage, to the giving of too much fluid food. He states that it disappeared when the amount of food was reduced. This has not been my experience. Many children who were fed by gavage showed no signs of it, and others who took a comparatively small quantity of food became œdematous. The best explanation seems to me to be that it depends upon a condition of hydræmia, associated with feeble resistance in the walls of the small blood-vessels, through which a transudation of serum readily takes place. The degree of anæmia noted in these patients is sometimes extreme.

The prognosis in this condition is extremely bad, as it rarely occurs except in hopeless cases of marasmus. This is not, however, invariably the case. The dropsy may disappear to return again, or it may disappear permanently and the case go on to recovery.

If the urine is scanty, such diuretics as the citrate of potash and the sweet spirits of nitre often cause a diminution and sometimes even a disappearance of the dropsy in a short time. The best of all remedies,

however, is digitalis. To an infant of two months, ♏ $\frac{1}{10}$ of the fluid extract may be given every two hours for two or three days; and for a short period somewhat larger doses may be employed.

CHAPTER III.

DISEASES OF THE GENITAL ORGANS.

MALFORMATIONS.

Adherent Prepuce.—This condition is sometimes called false phimosis. It is so constantly present that it can hardly be regarded as a malformation. It is, however, a condition needing attention in every male infant. The prepuce should be forcibly retracted so as to expose the glans completely. The smegma should then be washed away, the glans covered with a drop of oil, and the skin drawn forward. This should be repeated daily until there is no disposition to a recurrence of the adhesions.

Phimosis.—This is such a narrowing of the prepuce that it can not be retracted over the glans. The degree of phimosis varies greatly. In very rare cases there is no preputial opening. In other cases the orifice is so small that no part of the glans can be exposed, and there is obstruction to the outflow of urine; but usually a small part of the glans can be seen. Phimosis may be complicated by an elongated prepuce (hypertrophic phimosis), and the elongation may exist without any narrowing of the orifice, although this is usually present to some degree.

The presence of phimosis makes cleanliness impossible in many cases, and want of cleanliness leads to infection and to balanitis. This is quite frequent even in infants. It may be complicated by urethritis, and even by cystitis. Another consequence of the straining induced by phimosis is hernia, which may be either inguinal or umbilical. To cure the hernia is often impossible, unless the phimosis is relieved. Straining also leads to prolapsus ani, and, from pressure on the spermatic vessels, to hydrocele. More important even than these mechanical results of phimosis are the reflex conditions resulting from the irritation. Such symptoms may come from preputial adhesions as well as from phimosis. The hyperæsthetic condition and the resulting pruritus cause frequent priapism, and are among the most common causes of masturbation. It may produce other nervous symptoms, such as insomnia, night terrors, etc. Phimosis often causes frequent micturition, dysuria, and, in fact, most of the symptoms of stone in the bladder. It sometimes leads to vesical spasm and retention of urine, but more frequently to nocturnal incontinence.

The list of reflex phenomena which have been attributed to phimosis is a long one, and includes most of the functional nervous diseases of childhood. There is abundant evidence that phimosis may be a cause, although a rare one, of chorea, convulsions, epilepsy, hysterical manifestations, pseudo-paralysis, spasm of the muscles about the hip causing symptoms resembling the early stage of hip-joint disease, strabismus, amaurosis, diarrhœa, and many other nervous conditions. There is, however, no evidence that cases of spastic diplegia or paraplegia are ever caused by phimosis or improved by circumcision. There has been in the past a disposition on the part of some writers to attribute nearly all the nervous disturbances of boyhood to phimosis, and an exaggerated importance has certainly been attached to this condition. Still, in a delicate, anæmic child with unstable nervous centres, phimosis is capable of giving rise to nervous symptoms of a most serious and alarming character. It is an important etiological factor in many neuroses, and one which should not be overlooked. On the other hand, a very marked degree of phimosis often exists in robust children without producing any symptoms whatever.

Treatment.—Every case of phimosis should receive attention in infancy. Often very little treatment is needed; but trouble is likely to come sooner or later if it is neglected. When there is a very long prepuce with phimosis, the operation of circumcision should invariably be done, even when the degree of phimosis is slight. Many cases of phimosis in which the prepuce is not long can be relieved by stretching. If no part of the glans can be exposed, the simplest plan is to slit up the dorsum of the prepuce with a pair of scissors and forcibly break up the adhesions. The corners of the flaps thus made can then be snipped off and one stitch inserted on either side. This is very easily done, and gives most excellent results. In the case of obscure nervous symptoms in older boys, the condition of the prepuce should be examined and the same rules of treatment applied. In all cases of hernia, hydrocele, or prolapsus ani, when phimosis is present it should be relieved as the first step in the treatment.

Hypospadias.—In this condition the urethra is not continued to the tip of the penis, but opens on the inferior surface some distance back, being represented in front of this only by a shallow furrow. In more severe cases there is a deep fissure which divides the scrotum, and sometimes even the perinæum. Into this fissure the urethra opens. This is a condition likely to be mistaken for that of hermaphrodism, especially as the testicles are frequently in the abdominal cavity. It may be impossible to decide the sex of the child until puberty. Surgical operations for the relief of these deformities are not very successful.

Epispadias.—This is a condition in which the urethra opens on the dorsal surface of the penis. It is much less frequent than hypospadias.

There may be simply a division of the glans, or the fissure may extend the whole length of the organ and be complicated by—

Exstrophy of the Bladder.—This deformity is met with in all degrees of severity. In the complete form there is a median fissure from the umbilicus to the tip of the penis. It includes the anterior abdominal wall, the pelvic bones, and the urethra. The bones are entirely separated at the symphysis, or connected behind the bladder by a fibrous band. The hypogastric region is occupied by a red, mucous surface, slightly corrugated, which is all there is of the bladder. This is generally surrounded by a narrow rim of integument. In the lower lateral portions of the red mucous membrane two slightly rounded elevations are seen, from which urine oozes. These are the openings of the ureters. The penis is short, and presents a shallow furrow on its dorsal surface. With this deformity, also, the testes are often in the abdominal cavity.

An analogous deformity is sometimes seen in girls. There is a division of the clitoris and the labia minora and majora. The fissure may be so deep as to reach nearly to the anus. The vagina is usually absent. The rectum may open into the prolapsed bladder.

All these deformities are compatible with long life. In most of them the individual is incapable of procreation. In exstrophy of the bladder, whether complete or partial, patients are a nuisance to themselves and to all about them. It is almost impossible to prevent the clothing from being soaked with urine, which gives everything connected with the patient a strong ammoniacal odour. The skin is often excoriated. Operation for the relief of these cases should, I think, always be undertaken. Brilliant results have been obtained even in some of the most severe cases.

Undescended Testicle—Cryptorchidism.—In fœtal life the testes are situated in the abdominal cavity below the kidneys. They usually descend into the scrotum during the ninth month, but in children born at full term the testicle may be in the inguinal canal, or even in the abdomen. The former condition is quite a frequent one, being present according to good authorities, in fully ten per cent of all children. In the great majority of these the descent takes place without difficulty during the first weeks of life, and causes no symptoms. In others the condition persists. The testicle may be found in the abdominal cavity or at any point in the canal. If the latter, it may be felt as a small, hard tumour, slightly painful upon pressure. Even in some of these cases a natural descent takes place about puberty, usually without symptoms. The testicle occasionally makes for itself a false passage, and is found in the perinæum. When in the inguinal canal, descent of the testicle into the scrotum may sometimes be facilitated by manipulation. In other situations it had best be left alone, unless it gives rise to much pain or tenderness, as may happen when a false passage has been made. It should then be removed.

With the exceptions already mentioned, deformities of the female genitals belong rather to gynæcology than to pædiatrics, since they are chiefly of the internal organs, and do not usually give symptoms before puberty.

DISEASES OF THE MALE GENITALS.

Balanitis.—Balanitis, or inflammation of the prepuce, is one of the results of phimosis. It may follow decomposition of the smegma, infection of the mucous membrane, injury, or masturbation. The parts are swollen, œdematous, red, painful, and sometimes bathed in pus. Retraction of the prepuce is impossible. Under proper treatment the inflammation usually subsides in two or three days, but there may be some discharge for a considerable time. Abscess may follow, and even gangrene of the prepuce. The most severe cases are likely to be complicated with anterior urethritis.

The object of treatment is to remove the irritating and infectious material lodged beneath the foreskin. This may be quite difficult. It is best accomplished by syringing with a 1-to-5,000 bichloride solution. This should be repeated several times a day, the prepuce being held in contact with the syringe, so that it is distended by the injection. Where it is impossible to do this, an antiseptic lotion may be used and ice applied until the œdema has subsided. It is sometimes necessary to slit up the prepuce before the parts can be thoroughly cleansed, and in severe cases this is often the quickest method of cure. Circumcision should not be done during an attack.

Urethritis.—This, like the same disease in females, may be simple or specific. Both forms are less frequent in little boys than in the other sex. In simple urethritis the inflammation usually affects only the anterior part of the canal, the fossa navicularis. There is a slight discharge of pus, and sometimes pain on micturition. The most frequent cause is want of cleanliness.

Gonorrhœal inflammation is more common. This occurs even in boys as young as eighteen months, but most of the cases are in those over seven years old. The usual cause is direct contagion. The symptoms are more severe than in the simple form, and resemble the same disease in the adult, with the exception that constitutional symptoms are usually absent. A microscopical examination of the discharge (page 642) is the only positive means of diagnosis between the two varieties. In these cases it reveals the gonococcus in great numbers. Conjunctivitis and arthritis are seen as complications, just as in the female. Orchitis is very rare, but balanitis and bubo are not infrequent. Poynter has reported a case in a boy of three years, who, when five years old, required treatment for a urethral stricture. He was infected by a nurse.

The first thing in the treatment is always to keep the parts covered, otherwise the infection is almost certain to be carried by the hands to

other mucous membranes, usually the conjunctiva. In other respects the treatment is the same as in the adult.

Hydrocele.—Hydrocele consists in an accumulation of serum in some part of the serous pouch brought down by the testicle in its descent. In infants it is usually due to the imperfect closure of this pouch at some point, where a fluid accumulation occurs. Four varieties of hydrocele are met with in young children:

1. *Congenital hydrocele.*—In this the condition is a congenital one, although the tumour is not necessarily present at birth. The tunica vaginalis communicates with the general peritoneal cavity. There is present an elongated tumour, extending from the bottom of the scrotum throughout the whole length of the cord. The tumour is reducible, sometimes spontaneously by position, sometimes, when the opening is smaller, only by pressure. It reduces slowly, without gurgling, never going back *en masse* like a hernia. The tumour is translucent, and is flat on percussion. The testicle is above and posterior, and usually indistinctly felt. Congenital hydrocele may be complicated by hernia.

2. *Hydrocele of the tunica vaginalis with the canal closed.*—In this form the accumulation of fluid is in the scrotum, communication with the peritoneal cavity having been entirely cut off by the complete obliteration of this pouch in the canal in the normal way. This is one of the most frequent forms. It gives rise to an oval or pear-shaped tumour, quite tense and firm, usually about two inches in length. The cord is distinctly felt above it, the testicle is behind and somewhat above it, and not always felt very distinctly. This variety gives translucency and the usual elastic feeling of a hydrocele.

3. *Hydrocele of the cord.*—This is one of the rare forms. The serous pouch which accompanies the spermatic cord is open above, and communicates with the peritoneal cavity; but below it is closed. The scrotum is normal, and the testicle is in its usual position. The tumour is small, elongated, and reducible, and entirely above the scrotum. Usually it stops at some point in the inguinal canal. This hydrocele also may be complicated by hernia. The diagnostic points are the same as in the form first mentioned.

4. *Encysted hydrocele of the cord.*—The peritoneal pouch of the cord in this variety is closed for some distance above, and again below, but somewhere in its course it is open, and here the fluid accumulates in the form of a cyst. When small it resembles an undescended testicle; but on examination this organ is found below and in its normal position. When in the canal, it is often mistaken for a lymph gland, sometimes for a small hernia. The tumour is usually about the size of an almond. It is elastic and irreducible, and gives translucency like the other varieties. In cases of doubt it may be punctured by a hypodermic needle.

Treatment of Hydrocele.—In the congenital form the first point is to cause obliteration of the canal, so as to shut off the hydrocele sac from the general peritoneal cavity. This is usually done by the use of a truss, and, if applied early, it may be accomplished in the course of a few months. It is subsequently managed like an ordinary hydrocele of the tunica vaginalis. In infants and young children it is rare that active operative measures are called for in any variety of hydrocele, as these tend, in a great majority of cases at least, to disappear spontaneously in the course of a few months. Absorption is often facilitated by the application of collodion. In many cases the internal administration of iodide of potassium, twelve grains a day, causes a rapid disappearance of the effusion. Iodine may be applied locally over a hydrocele of the cord, but should not be applied to the scrotum. In some cases which do not disappear promptly, simple puncture with the needle, allowing the fluid to drain off into the cellular tissue of the scrotum from which it is absorbed, is an excellent means of treatment. Others are cured by a single aspiration with hypodermic syringe. I have treated in the neighbourhood of one hundred of these hydroceles in infants and young children, and have never yet seen one in which it was necessary to resort to the injection of irritants like iodine or carbolic acid.

DISEASES OF THE FEMALE GENITALS.

VULVO-VAGINITIS.

This is a catarrhal inflammation, usually affecting the mucous membrane of the vulva, vagina, urethra, and often that of the cervix uteri. It may be simple or specific (gonorrhœal). Neither form is very rare.

Simple Vulvo-vaginal Catarrh.—This may be seen at any age, even in infancy. It is, however, most frequent after the second year. It more often occurs in girls who are anæmic, or suffering from malnutrition, than in those whose general health is good, being especially common in those who live in unhygienic surroundings or where personal cleanliness is neglected. It may follow any of the infectious diseases, particularly measles. There seems to be little doubt that even this form may be spread by contagion. It is common in children in institutions, where small epidemics are sometimes seen. It may be communicated by direct contact, or by handling the parts, or through clothing, diapers, sponges, towels, etc. The disease may be traumatic, as from attempted rape,* or the introduction of foreign bodies. It may be secondary to the presence

* See "Twenty-one Cases of Rape in Young Girls," by Walker, Archives of Pædiatrics, vol. iii, 1886, where the medico-legal points with reference to this condition are fully discussed.

of pinworms, or to scabies, and it is sometimes the cause, sometimes the result, of masturbation.

Symptoms.—The disease generally begins as a subacute catarrhal inflammation, the discharge being the first thing noticed. In the milder cases this is thin and yellowish-white, with some pain on locomotion, itching, and burning on micturition. In the more severe form it is abundant and of a yellowish-green colour, causing the labia to adhere, and the secretion, drying, forms crusts. The odour is sometimes extremely fetid, and the skin of the thighs may be excoriated. The local examination shows the mucous membrane to be red, swollen, œdematous, and bathed in pus. All the visible parts—urethra, hymen, vagina, etc.—are involved. By using an ordinary urethral speculum in the vagina, pus may be seen in most of the severe cases to come from the cervix uteri (Koplik). There are no constitutional symptoms. There may be swelling, and even suppuration, of the inguinal glands. The disease has no definite course, but usually with proper treatment lasts from one to three weeks, when there may be complete recovery, or there may persist for a long time a leucorrhœal discharge. In children who are in poor general condition, and where proper means of treatment are neglected, vulvo-vaginitis may last for months.

Gonorrhœal Vulvo-vaginitis (Uro-genital Blennorrhœa).—Recent studies of the micro-organisms in the discharge have shown cases of true gonorrhœa in young girls to be very much more numerous than was formerly suspected.* While indirect infection is no doubt possible, and in certain cases proved, nearly all writers agree that this is very exceptional, and that the most common origin of the disease is direct contact, either intentional or accidental, with another case of gonorrhœa, sometimes sexual and sometimes by the hands. In this way the disease may be conveyed from one child to another, or from adults to children, very often from parents who occupy the same bed with the child. Pott states that, in 90 per cent of his forty-four cases, the mothers were found to be suffering from leucorrhœa. The mode of contagion may be difficult to trace, but this fact should cast no doubt upon the diagnosis in the case. The disease occurs in girls of all ages, but chiefly between three and eight years. Epstein has reported cases in the newly-born. The incubation in three cases in which it could be definitely traced, was exactly three days (Cahen-Brach).

Symptoms.—The disease is believed to begin usually in the urethra, although this is in most cases difficult to establish, as there are generally found on the first examination evidences of inflammation of all the mucous

* For an excellent *résumé* of this subject, with references to recent literature, see Koplik, Journal of Cutaneous and Genito-Urinary Diseases, June, 1893; also Heiman, New York Medical Record, June 22, 1895.

membranes of this region. There is a copious secretion of thick, yellow pus. There may be erosions of the vaginal mucous membrane, so that the parts bleed readily. Crusts form on the labia. When a view of the cervix can be obtained by means of a small speculum, this is almost invariably seen to be involved. For the first day or two, in the most severe cases, there may be slight fever and general indisposition, but more frequently—and this is one of the most striking points of difference from the disease as seen in adults—constitutional symptoms are wanting altogether. Micturition is painful, and sometimes frequent, there are also excoriations of the skin, and difficulty in walking, all these symptoms being usually more severe than in simple catarrh. The duration of these cases is indefinite, being from one to six months. Under the most favourable conditions it is several weeks, largely owing to the great difficulties in the way of a thorough application of local treatment. It is always more obstinate than is a simple catarrh.

A positive diagnosis between the simple and gonorrhœal catarrh can be made with certainty only by a microscopical examination of the discharge. The pus for examination should be taken from as high a point in the tract as possible, preferably the orifice of the urethra, in order to avoid contamination. In a simple catarrh the discharge is made up of epithelial and pus cells, with quite a variety of bacterial forms—bacilli, cocci, and diplococci. These bacteria are found in the epithelial cells and in the pus cells, but they are generally associated, and the diplococci are few in number. In cases of gonorrhœal inflammation there are found in the pus cells large masses of diplococci, these being usually the only bacteria present. It should then be emphasized that the mere presence of a few diplococci, even though found in the pus cells, is not enough to establish the diagnosis of gonorrhœa, since there are varieties of diplococci found in the simple catarrh, and even in the normal vaginal secretion, which morphologically closely resemble the gonococcus of Neisser. It is the presence of these in large masses in the pus cells which is the characteristic feature (Koplik). According to the very careful observations of Heiman, the two varieties of diplococci may be positively differentiated by staining by Gram's method. The gonococcus is decolourized, while the other form is not.

Nearly all the complications of gonorrhœa which are seen in the adult have been observed in young children, but the majority of them are rare. The most frequent one is conjunctivitis, infection being carried by the hands from the vaginal discharge to the eyes. Gonorrhœal arthritis is not common, but may affect the knee, ankle, wrist, or elbow. The symptoms of arthritis resemble those of ordinary rheumatism. Cystitis is extremely rare. Bubo is occasionally seen, and may be simple or suppurative. As already stated, the disease in many, probably in nearly all the severe cases, affects the lining of the uterus. Infection may extend from the

uterus to the tubes and cause pyosalpinx, or even peritonitis. Sänger reports a case of pyosalpinx from gonorrhœal infection in a little girl of three years, and Huber a fatal case of peritonitis of similar origin in one of seven. I have myself seen one of severe pelvic peritonitis in a girl of seven. In all these cases the diagnosis of the gonorrhœal origin of the disease must rest upon the presence of gonococci in the vaginal discharge.

Treatment of Vulvo-Vaginitis.—The first thing is proper isolation, and care to prevent the spread of infection by means of clothing, linen, etc. In institutions, and in families where there are many children, the greatest care is necessary even in catarrhal cases.

Simple vaginal catarrh requires cleanliness, which is best secured by irrigating twice daily with a warm saturated solution of boric acid, or 1 to 10,000 bichloride. A pad of sterilized absorbent cotton, the meshes of which are filled with boric acid and starch, or iodoform, may be placed between the labia in the most severe cases, the patients being kept in bed. The skin should be protected by ointments. In obstinate cases, irrigation with astringent solutions, such as sulphate of zinc or tannic acid, may be used. More radical means are rarely required. Attention to the general condition of the patient must not be overlooked, and the health should be built up by iron, cod-liver oil, and other tonics. Every young child should wear a napkin, to prevent carrying the disease to the eyes by the hands.

In the *gonorrhœal cases* nothing is so efficient as the irrigation with the solutions above referred to. They should, however, be employed more frequently; in the early stage, where the secretion is abundant, as often as three or four times a day. In cases passing to the chronic stage, a solution of nitrate of silver, ten grains to the ounce, may be applied to the vagina through a speculum. This should be repeated every second or third day. In all circumstances these cases are tedious, and require the closest attention to detail to insure the best results. Relapses are not uncommon in cases which had apparently recovered.

HERPES OF THE VULVA.

This may occur on the cutaneous surface only, or there may be a herpetic condition of the mucous membrane. The skin of the perinæum may be involved, and the disease may extend quite to the anus. On the skin, the eruption runs the ordinary course of herpes elsewhere. Vesicles form and rupture or dry, forming crusts or leaving small ulcers, which heal in a week or ten days if the parts are simply protected. On the mucous membrane the vesicles are succeeded by small ulcers, which may coalesce and form larger ones, the appearance resembling the same condition in the mouth. The symptoms are itching, burning pain, and a slight discharge. The herpetic ulcer may be confounded with a mucous patch. These cases usually recover promptly if dusted with some absorb-

ent powder like boric acid and oxide of zinc, or talcum. In addition, cleanliness should be secured. It is important that this condition should be attended to, as it is sometimes followed by more serious disease.

GANGRENOUS VULVITIS (NOMA).

This is the same process as that seen in the mouth and known as cancrum oris. It usually follows one of the infectious diseases, most frequently measles, occurring in patients whose general vitality has been greatly reduced. The condition may succeed a simple catarrh or a herpetic vaginitis. There is first noticed a tense, brawny induration, the skin being shiny and swollen over a circumscribed area. In the centre of this there soon appears, usually upon one of the labia majora, a dark, circumscribed spot. Day by day the gangrenous area advances, preceded by the induration. It may involve the whole labium, extending even to the mons veneris and the perinæum. These cases are generally fatal. If recovery takes place, it is with considerable deformity of the parts in consequence of the extensive sloughing and cicatrization. As sequelæ, there may be fistulæ, stenosis, or atresia of the vagina. The prognosis is very bad. The only radical treatment is early excision of the gangrenous part, and the application of the actual cautery or nitric acid.

CHAPTER IV.

ENURESIS.

Synonyms: Incontinence of urine; bed-wetting.

Enuresis may be due to some malformation of the genital tract, such as an abnormal opening of the bladder into the vagina, to extroversion of the bladder, or to the persistence of the urachus; in the latter case the urine is discharged from the umbilicus. It also occurs in organic diseases of the central nervous system, such as idiocy, cerebral palsy, acute meningitis, tumours of the brain, certain forms of myelitis, and in injuries of the cord. In many of these conditions there is associated incontinence of fæces. Both of the groups of cases mentioned are quite distinct from the ordinary form of incontinence of urine which is seen in childhood. The latter is to be regarded as a neurosis, and is the only variety which will be considered here.

In early infancy, evacuation of the bladder is purely a reflex act. An impulse is sent from the nerves of the bladder to the spinal centre, and a reflex impulse from this centre produces simultaneously a contraction of the detrusor urinæ and a suspension of the contraction of the vesical

sphincter. It is often possible to teach infants to control the evacuation of the bladder before the end of the first year; usually, however, control is not acquired even during waking hours until some time during the second year, and in some healthy infants not before the end of the second year. The time depends very much upon the training. If a child during its third year can not control the evacuation of the bladder during its waking hours, incontinence may be said to exist.

Etiology.—Incontinence of urine may be due to a continuance of the infantile condition, to anything which increases the irritability of the spinal centre, or which interferes with the cerebral control over this centre, or to anything which increases the irritability of the terminal filaments of the vesical nerves or of those in the neighbourhood, in consequence of which too many or too strong impulses are sent to the spinal centre. The causes of incontinence thus may be in the central nervous system, in the urine, in the bladder, or in any of the adjacent organs.

The causes relating to the central nervous system are in the main those of the other neuroses of childhood; these are anæmia, malnutrition, an inherited nervous constitution, or a condition of extreme nervousness or neurasthenia, the result of the child's surroundings. In such cases incontinence is often associated with chorea, epilepsy, hysteria, headaches, neuralgia, and other nervous symptoms. In these conditions there may be not only an increased irritability of the nerve centres, but also of the peripheral nerves, accompanied by loss of tone of the vesical sphincter. A similar condition may exist with almost any form of acute illness, usually, however, being only temporary.

The causes referable to the urine are chiefly a highly-acid urine, generally associated with lithuria. In such cases the incontinence is very often due more to the constitutional than the local condition.

In the bladder itself, cystitis and vesical calculus, although infrequent, should not be overlooked as possible causes. In a few cases, where incontinence has existed a long time, the bladder becomes so contracted that it will hold only an ounce or two of urine. This condition, although not the primary cause of enuresis, may be enough to continue it.

Local irritation in the neighbouring organs may be due to adherent prepuce, balanitis, phimosis, or to a narrow meatus. All of these conditions are frequently associated with incontinence. Rectal irritation may be caused by pinworms, anal fissure, or rectal polypus; and vaginal irritation by vulvo-vaginitis or adherent clitoris, both, however, being extremely rare. Often we have incontinence as the result of a combination of several causes, no one of which alone would have been sufficient to produce it. Thus, in a healthy child phimosis may give rise to no symptoms, while in one who is anæmic or neurasthenic it may produce enough local irritation to cause incontinence. In many cases heredity seems to be a factor of some importance, parents often having suffered in their child-

hood from the same disease; quite frequently there are seen two and sometimes even three children in the same family affected. In many cases the condition seems to be mainly the result of habit, and in all cases habit is a potent factor in continuing the incontinence, sometimes after the original exciting cause has been removed. Frequently no adequate cause can be found. Both sexes are about equally liable to enuresis, and it may be seen in all ages up to puberty.

Symptoms.—Enuresis may be nocturnal or diurnal, or both. Of 194 cases, 73 were nocturnal, 9 diurnal, and 102 were both nocturnal and diurnal. Cases differ greatly in severity. Incontinence may be habitual, occurring every night, often several times during the night, and frequently during the day; or it may be only occasional under the influence of some special exciting cause, where it continues a few days or weeks until the cause is removed. In a considerable number of cases, the condition lasts from infancy until the sixth or seventh year. It may even continue until puberty; but it generally ceases at that period, unless its cause is mechanical, or depends upon some organic disease of the brain or cord. In ordinary enuresis there is never dribbling of the urine, but usually a contraction of the walls of the bladder follows almost immediately upon the desire, before the patient can make his wants known or reach a convenient place for micturition. At night the same thing may occur without wakening the child, the contraction being of purely reflex origin.

Prognosis.—The condition is usually hopeless when it depends upon organic disease of the brain and cord; also in cases due to malformation, unless these are amenable to surgical treatment. In the ordinary cases seen, the prognosis depends upon the age of the child, the duration of the symptom, and the nature of the exciting cause. As a rule, it is better in children only four or five years old than in those of eight or nine, for the obvious reason that a case which has lasted to the latter age is usually an intractable one. If a cause can be discovered and if this is one that can be removed, the prognosis is much better than if no cause can be found. In the great majority of the cases a cure is possible, provided the patient can be held long enough to a regular plan of treatment. The treatment must in most cases be continued from three months to a year, and always for several months after the incontinence has ceased, on account of the strong tendency to relapses.

Treatment.—The first indication is to remove the cause, where one can be found. If there are preputial adhesions, they should be broken up and irritating smegma removed. If phimosis is present, it should be relieved by stretching or circumcision. A narrow meatus should be cut to proper dimensions. If stone in the bladder is suspected, as it should be when the incontinence is worse by day and accompanied by straining and painful spasm of the bladder, the patient should be sounded for stone. Pinworms in the rectum should receive the appropriate treatment by

injections. A urine of high acidity, with deposits of uric acid, calls for alkalies and the free use of fluids, especially water. While the local conditions mentioned should always be attended to, the fact remains that few cases are cured simply by relieving them, except those due to vesical calculi. The explanation of this is that habit is so important a factor in keeping up incontinence where it has existed a long time. In most cases, therefore, we must depend upon general measures and drugs directed toward the relief of the symptom, either in conjunction with local treatment or alone.

Care should be taken to secure for the child a simple, natural life, preferably in the country. There should be no overtaxing of the nervous system at home or in school. Every cause of unnatural excitement should be avoided. Early hours and plenty of sleep must be insisted upon. Certain articles of diet are to be avoided, and coffee, tea, and beer should be absolutely prohibited. Sweets and all highly seasoned food should be very sparingly allowed, or not at all. Although it is believed by many that a diet into which meat enters largely is injurious, from personal experience I have not found the exclusion of meat to be of any advantage; nor is anything to be gained by limiting the amount of water which the child takes, except possibly in cases of nocturnal incontinence, where it is well to restrict the quantity taken late in the afternoon. When incontinence is associated with highly-acid urine, it is often aggravated by cutting down the fluids. The diet which succeeds best is a simple one composed of milk, vegetables, fruits, meats, and cereals. Punishments, whether corporal or otherwise, do no good, and are in most cases absolutely harmful. They should never be allowed. Rewards are much more effectual. The moral treatment of a case is important; it is well to work upon a child's pride, and use every means to strengthen his will. Where the incontinence is solely or chiefly at night, the child should be taught to hold his water as long as possible during the day, in order to accustom the bladder to full distention.

Measures which are directed toward the patient's general condition are quite as important as those employed for the control of the incontinence. Anæmia, chlorosis, malnutrition, indigestion, and constipation should each receive careful attention. Any local condition, such as adenoid growths of the pharynx, which might serve to increase the general nervous irritability, should be removed.

Of the drugs used for the purpose of affecting the incontinence, belladonna stands at the head of the list; but it must be given in full doses, usually sufficient to produce the physiological effects, and continued for a long time, in most cases for many months. Either the fluid extract or the alkaloid, atropine, should be employed. My preference is for the latter, because of its more uniform strength. A convenient method of administration is to use a solution of atropine, one grain to two ounces of

water, of which one drop ($\frac{1}{1000}$ of a grain) may be given for each year of the child's age. For nocturnal incontinence this dose should at first be given at 4 and 10 P. M.; after a few days, at 4, 7, and 10 P. M. Usually this may be gradually increased until double the quantity is given. A child of five years would then be taking ten drops ($\frac{1}{100}$ of a grain) at each of the hours mentioned. I have rarely found it advisable to go above these doses. As the larger doses are reached the increase should be more gradual. When the condition is under control, or when the full physiological effects of the drug are produced, the same dose should be continued for some time and then reduced, the atropine being given for at least two months in gradually diminishing doses after the incontinence has ceased. This is very important if the cure is to be permanent, as there is so strong a tendency in these cases to relapse.*

Strychnine may be added in cases not yielding to the atropine alone. It is particularly advantageous when there is diurnal as well as nocturnal incontinence, for under these conditions there is usually a lack of tone in the sphincter, as well as increased irritability in the mucous membrane of the bladder. The initial dose for a child of five years should be $\frac{1}{100}$ of a grain twice daily; this may be gradually increased to $\frac{1}{50}$ of a grain three times a day; but there is rarely any advantage in pushing it further. Ergot is sometimes useful, but rarely gives relief when both strychnine and atropine have failed. The indications for its administration are the same as for strychnine, but it is objectionable for prolonged use on account of the disturbance of the stomach. Rhus aromatica, although inferior to the drugs already mentioned, possesses a certain amount of value, and may be tried in case the others fail. From three to twenty drops of the fluid extract should be given three times a day. Like strychnine, it is indicated in atonic cases. Of the other measures recommended, raising the foot of the bed at night to keep the urine away from the neck of the bladder, may give temporary relief, as may also some of the various contrivances for preventing the child from sleeping upon the back; but

* As an illustration of the success which may be obtained by this plan of treatment when faithfully carried out, our experience in the New York Infant Asylum may be cited. Twelve obstinate cases, in none of which could any local cause be found, were selected and treated by Dr. Kerley, then resident physician, in the manner indicated. After five months' treatment, seven of the cases were so much improved that incontinence rarely occurred. The atropine was, however, continued in smaller doses for four months longer, at the end of which time the cases were well. In the remaining five cases but little improvement was seen after five months' treatment, and not until the end of ten months could it be said that much improvement had occurred. In these cases the drug was continued for two months longer and all treatment discontinued, as the cases were cured. None of these had relapsed six months afterward. It was here of great advantage that the children were under close observation in an institution where the treatment could be continued. In dispensary and private practice the want of early success would no doubt have deterred mothers from continuing the medicine.

none of these are in any sense curative. Some obstinate cases have been relieved by galvanism, the positive pole being placed over the lumbar spine and the negative pole over the bladder. If there is reason to suspect a contracted bladder, as when the incontinence has lasted for years and the bladder will never hold more than an ounce or two of urine, cure is sometimes accomplished by daily distending the organ up to its normal capacity with warm water.

VESICAL SPASM.

This is quite a common condition, and often passes under the name of *genital irritation*. It is characterized by frequent, sometimes by difficult and painful, micturition. It occurs in children of all ages, even in infants, but is especially frequent between the ages of two and five years. This symptom has already been referred to in connection with uric-acid infarctions in very young infants.

The usual cause is the irritation of the bladder by a concentrated, highly-acid urine. It often results from cold; it may accompany acute febrile processes, and is sometimes merely a symptom of nervous irritability. The cause may thus be in the bladder or in the urine. It may be accompanied by enuresis, but usually occurs without it. It is sometimes symptomatic of disease in adjacent parts, as in the rectum or the pelvic peritonæum, or it may be associated with inflammation of the vulva or urethra. It is also one of the symptoms of vesical calculus.

The *symptoms* of vesical spasm are local only. The child passes water very frequently, often several times an hour. The accompanying pain may be intense, not infrequently sufficient to cause the child to cry out. Often there are pain and severe vesical tenesmus with the passage of only a few drops of urine at a time, but blood is not present. If the condition depends upon the character of the urine, or is only an expression of an extreme vesical irritability, the symptoms are generally of short duration, possibly a day or two. If it depends upon vesical calculus, it may be intermittent. If it is associated with disease of the adjacent pelvic viscera, it is inconstant, and may continue for a considerable period, depending upon the nature of the cause.

The *treatment*, in the ordinary cases, consists in the administration of an abundance of water, with alkaline diuretics, and either belladonna or hyoscyamus. The following formula is one that I have usually found efficient:

℞ Tincturæ hyoscyami..... ℥ ss.
Potassii citratis..... ʒ j
Aquæ destillat..... ℥ ij
M. Sig.: Half a teaspoonful in water every hour to a child of two years.

If the cause is outside the bladder, it should receive appropriate treatment.

VESICAL CALCULI.

The nucleus of a vesical calculus is usually a renal calculus which has passed the ureter, but has been prevented by its size from going farther. Stone in the bladder is extremely rare in infancy, probably owing to the fluid diet, but it is not infrequent in children from two to ten years of age. The most common variety of calculus at this time is the uric acid. The other forms, although occasionally seen, are all quite rare.

The symptoms in children are somewhat different from those in adults, and the condition is often overlooked. There is frequently pain upon micturition, especially at the end of the act, which may be felt at the end of the penis or in the perinæum. There may be a sudden stoppage in the flow of urine. The straining often leads to rectal tenesmus and even to prolapse. This complication is so frequent that, in a case of persistent prolapse, stone should always be suspected. Incontinence of urine is a prominent, and often the principal, symptom ; in many cases it is noticed only during the day. The urinary changes are not generally marked ; hæmaturia is rare, and mucus and pus are infrequent and in small quantity. The genital irritation may lead to the habit of masturbation. A stone of any considerable size may often be felt by a bimanual examination, one finger being placed in the rectum and the other hand above the pubes. This is easier in males than in females, but it is not very trustworthy, and not conclusive when it gives a negative result. A positive diagnosis is made only by exploring the bladder with a sound.

The treatment of calculus is purely surgical. In young children the suprapubic is now generally preferred by surgeons to the perineal operation, if the calculus is too small to be easily removed by crushing.

SECTION VII.

DISEASES OF THE NERVOUS SYSTEM.

CHAPTER I.

INTRODUCTORY.

The Weight of the Brain.—From ninety-eight observations made in the post-mortem room of the New York Infant Asylum, the following were the average weights noted:

At three months..... 21 oz. (602 grammes).
At six months..... 25½ " (712 ").
At twelve months..... 32½ " (916 ").
At two years..... 35 " (990 ").

The following are the figures given by Boyd and Schäfer:*

Age.	Males.		Females.	
	Ounces.	Grammes.	Ounces.	Grammes.
At birth (full term).....	11½	330	10	283
Under three months.....	17½	493	16	451
From three to six months.....	21	602	20	560
From six to twelve months.....	27	776	26	727
From one to two years.....	33	941	30	843
From two to four years.....	39	1,095	35	990
From four to seven years.....	40	1,138	40	1,135
From seven to fourteen years.....	46	1,301	40⅔	1,154
From fourteen to twenty years.....	48½	1,374	44	1,244

At birth the weight of the brain to that of the body is nearly 1 : 8. During infancy and childhood the following is the ratio, according to Bischoff: during the first year, 1 : 6; the second year, 1 : 14; the third year, 1 : 18; at the fourteenth year, 1 : 15 to 1 : 25; in adults, 1 : 43.

The Spinal Cord.—The weight of the cord to the weight of the body at birth is 1 : 500; in adult life it is 1 : 1500. According to Kölliker, the spinal cord and the vertebral column are the same length until the end of the third month of fœtal life, there being at this time no cauda equina. At the ninth month the lower end of the cord is opposite the third lumbar vertebra; in the adult it is opposite the first.

* Quoted by Sachs.

Some Peculiarities in the Diseases of the Nervous System in Infancy and Childhood.*—The relatively large size, the rapid growth, and the immaturity of the brain and cord during early life, explain much that is peculiar in the nervous diseases of this period.

At this time, apparently trivial causes are enough to produce quite profound nervous impressions, because of the instability of the nervous centres and the greater irritability of the motor, sensory, and vaso-motor nerves. These are conditions which are very much increased by all disturbances of nutrition. These disturbances may be manifold in character, but they lie at the root of very many of the neuroses of early life,—e. g., extreme nervousness, disorders of sleep, stuttering, chorea, incontinence of urine, tetany, and convulsions. The great liability to convulsions depends not only upon the greater irritability of the peripheral nerves, but on the instability of the nervous centres and the lack of inhibition over the motor ganglion cells of the spinal cord. The nervous centres are more easily exhausted than later in life. Prolonged or continuous overstrain from any cause whatsoever, frequently leads to headache and chorea, and sometimes even to epilepsy and insanity.

Another peculiarity is the serious consequences which often follow reflex irritation, although this is rarely the only factor in the case. Conditions which in adult life produce almost no effect may in infancy be the cause of most alarming symptoms. As a few examples may be cited, reflex symptoms due to phimosis, to intestinal worms, convulsions from disturbances of digestion, nervous symptoms due to eye-strain, or to adenoid growths of the pharynx. In the production of some of these, especially attacks of convulsions, there are several factors, such as the great irritability of the peripheral nerves, the instability of the nervous centres—often a result of disturbed nutrition, as in rickets—and the lack of inhibitory action of the cortex of the brain.

As a third point of importance may be mentioned the grave permanent results which often follow relatively small organic lesions. A good illustration is seen in the lesions which produce cerebral birth-palsy. Here the damage is only in small part the immediate effect of the hæmorrhage, for this often is not great, but it is the interference with the development of certain parts of the cortex that makes this condition so serious.

From what has been said, it follows that the hygiene of the nervous system is of the utmost importance in infancy and childhood. It is essential for the healthy development of the nervous system that all stimulants should be avoided,—not only tea, coffee, and alcohol, but undue and unnatural excitement, the effect of which in infancy is almost as serious. A normal development can take place only in the midst of

* See Rachford; Some Physiological Factors in the Neuroses of Childhood. Cincinnati, 1895.

quiet and peaceful surroundings, with plenty of time for rest and sleep. The conditions of modern life, especially in cities, are such that these laws are almost invariably violated, and the consequences of this are seen in the marked and steady increase in nervous diseases among children.

CHAPTER II.

GENERAL AND FUNCTIONAL NERVOUS DISEASES.

CONVULSIONS—ECLAMPSIA.

Under this head are included attacks of acute transient nervous disturbance, characterized by involuntary rhythmical spasm of the muscles, either of the face, trunk, or extremities, or all of them, usually accompanied by loss of consciousness. They may be regarded as "motor discharges" from the cortex of the brain.

Etiology.—The principal predisposing causes are infancy, conditions affecting the nutrition of the brain, and heredity influences. Of all these factors, the most important one is the instability of the nerve centres which is characteristic of infancy and is associated with the non-development of the voluntary centres of the cortex. The brain grows more during the first year than in all later life, and this rapidity of growth is in itself an important predisposing cause of functional derangement. After infancy, attacks of convulsions are much less frequent, and after seven years they are relatively rare. While convulsions occasionally occur in children previously healthy, the majority of attacks are in those in whom there is at least some disturbance of the nutrition of the brain,—the cerebral instability of infancy being greatly exaggerated by such nutritive disorders. The most frequent one is rickets, which may be regarded as altogether the most important predisposing cause of infantile convulsions. They are often one of the earliest symptoms of that disease, and where convulsions occur in infancy without evident cause, rickets should always be looked for. Any disturbance of nutrition may predispose to convulsions, such as exhaustion, anæmia, malnutrition, syphilis, and debility resulting from any acute disease, especially those of the digestive tract. Children who inherit from their parents a peculiarly nervous temperament are more liable to convulsions than are others. This predisposition is often seen in several members of the same family. Females are rather more frequently affected than males.

The exciting causes include a wide variety of pathological conditions, among which disturbances of digestion take the first place. Where the susceptibility is very great, the exciting cause may be a trivial one. These

causes may be grouped under three general heads: (1) direct irritation of the cortex of the brain; (2) reflex irritation; (3) toxic influences.

Under the head of direct irritation may be included all convulsions occurring with the various forms of cerebral disease; the most frequent are meningitis, meningeal or cerebral hæmorrhage, tumour, abscess, hydrocephalus, embolism, and thrombosis. As examples of reflex irritation may be classed the convulsions following severe injuries, like compound fractures or burns, renal or intestinal colic, retention of urine, phimosis, a foreign body in the ear, or intestinal strangulation. A case has been related to me in which the application of cold to the skin repeatedly induced convulsions. Other conditions classed under this head are dentition and worms, but both must be regarded as exceedingly rare causes of convulsions. The exciting cause is very frequently the presence in the stomach or intestines of undigested food; such attacks are sometimes ascribed to reflex irritation, but the majority are better regarded as toxic. Acute and chronic indigestion are to be ranked among the most frequent causes of convulsions, both in infants and older children. In either there may be but one attack, or attacks may recur at intervals of a few months with a repetition of the cause. Of toxic origin may be considered not only the convulsions resulting from conditions like uræmia and asphyxia, but also those which occur at the onset or in the course of various infectious diseases, sometimes classed as febrile convulsions. They are very frequent at the onset of certain diseases, particularly pneumonia, scarlet fever, malaria, acute indigestion, and gastro-enteric infection; less frequently of measles, typhoid fever, ileo-colitis, and diphtheria. In these cases the convulsions seem due partly to the intensity of the poison and partly to the suddenness with which it affects the nervous system. Convulsions occurring late in the course of many diseases may be due to toxic influences, especially when associated with exhaustion of the nerve centres, from the prolonged disturbances of nutrition accompanying the febrile condition.

In pertussis—which, of all infectious diseases, is the one in which convulsions are most frequent—several factors may be present: asphyxia due to a severe paroxysm, cerebral congestion or hæmorrhage resulting from such a paroxysm, or simply from the peculiar susceptibility of the patient brought about by the disease itself.

Convulsions may be associated with enlargement of the thymus gland. I have notes of three cases of fatal convulsions where there was found at autopsy great enlargement of this body, which weighed from one to one and a half ounces. Two of these infants were previously healthy; one was rachitic. The similarity of all these cases convinced me that the convulsions were in some way due to the enlarged thymus, probably from pressure either upon the bronchi and lungs, or upon the pneumogastric (page 43).

There are some cases of convulsions for which no cause can be discovered even at autopsy, and for the present we must be content to class them as idiopathic. One attack of convulsions renders the patient more liable to a second, and where there have been several, they occur from causes which are less and less marked.

Pathology.—The "nervous discharge" which occurs in an attack of convulsions differs in no essential particulars from that of ordinary epilepsy. In the latter disease there is seen a tendency to recurrence with greater or less frequency, until the discharge may take place from very slight causes.

The part of the brain most intimately concerned in the production of convulsions is the cortex. Such attacks may be regarded as involuntary discharges of nerve force from the cortical motor centres, which result from direct irritation of these parts by disease; or from an irritation arising in some other part of the brain, as from the vaso-motor centres of the medulla; or from a reflex irritation in a distant part of the body. Convulsions may depend upon the fact that while nerve cells may be able to generate nerve force they can not control its discharge, as in the convulsions of rickets. An important element in the convulsions of infancy, according to Hughlings Jackson, is the lack of development of the higher cerebral functions, in consequence of which they do not exert the controlling influence over the discharge of nerve force which they do in later life.

The condition of the brain in the beginning of an attack of convulsions is one of anæmia; this is shortly followed by venous hyperæmia which may be very intense. In infants who die during convulsions the brain and its meninges are usually found intensely congested. They may be the seat of punctate hæmorrhages, and sometimes of more extensive ones. The lungs are also deeply congested, and the right heart is generally distended with dark clots. The other lesions found are accidental.

Symptoms.—In some cases prodromal symptoms are present, such as extreme restlessness, irritability, slight twitchings of the muscles of the face, hands, feet, or eyelids. More frequently, however, the attack comes quite suddenly with but momentary warning. Usually the first thing noticed is that the face is pale, the eyes fixed, sometimes rolled up in their orbits; in a moment or two convulsive twitchings begin in the muscles of the eye or face, or in one of the extremities, which usually rapidly extend until all parts of the body participate. In most cases the convulsions become general, but they may, however, remain unilateral even when not due to a local cause,—a point which is often forgotten. The contraction of the facial muscles causes a succession of grimaces; the neck is thrown back; the hands are clenched; the thumbs buried in the palms; and a quick spasmodic contraction of the extremities occurs. There may be some frothing at the mouth, and in all true convulsions there is loss of consciousness. Respiration is feeble, shallow, and may be

spasmodic. The pulse is weak; it may be slow or rapid; often it is irregular. The forehead is covered with cold perspiration. The face is first pale, then becomes slightly blue, especially about the lips. Unnatural rattling sounds may be produced in the larynx. The bladder and rectum may be evacuated. The convulsive movements consist in an alternation of flexion and extension occurring rhythmically. All varieties of tonic and clonic spasm may be seen, and in all degrees of severity. The contractions of the two sides of the body are usually synchronous. After a variable time, from a few moments to half an hour, the convulsive movements are gradually less frequent, and finally cease altogether, usually leaving the patient in a condition of stupor. They may recur after a short time or there may be but one attack. A period of general relaxation usually follows the convulsive seizures, frequently accompanied by marked evidences of prostration. Transient paralysis, apparently due to exhaustion of the nerve centres, is not an uncommon sequel.

Death may take place from a single attack; this, however, is rare except in very young infants, especially those who are rachitic. There may be no sequel to the convulsions if the cause is a temporary one, or they may produce some serious brain lesion, particularly meningeal hæmorrhage. Death from convulsions is generally due to asphyxia, or to exhaustion from the rapidly recurring attacks. Many cases recover in which the children for several minutes had the appearance of being moribund.

One attack of convulsions is very apt to be followed by others; for the occurrence of the first one usually reveals a peculiar susceptibility of the nervous system, and each succeeding attack comes from a less powerful exciting cause than the previous one. The longer the interval which has passed, the less likely is there to be a repetition, especially if the child has passed its third year. The number of attacks may be very great. In a case recently under the care of Dr. A. M. Thomas and myself, an infant during the latter part of its second year had during six months over thirty-five hundred distinct attacks of convulsions. For a considerable period they reached the almost incredible number of eighty a day, and yet the mental condition of the child in the interval was apparently normal.*

Diagnosis.—There can rarely be any difficulty in recognizing an attack of convulsions. The difficulty consists in determining with which of the many possible exciting causes we have to do in the case before us. Is it epilepsy? Does it depend upon cerebral disease? Does it mark the onset of some other acute disease? Is it reflex, and if so to what is it

* The post-mortem examination of this case has not yet been completed, but thus far there have been found only degenerative changes in the nerve cells of the cortex in the motor area and an increase in the neuroglia. These changes existed over quite an extensive area, and were more marked upon one side.

due? To answer these questions a careful history must be obtained, and all the circumstances surrounding the patient, the character of the convulsions, and all the other symptoms present must be taken into consideration.

In infancy, epilepsy is certainly the least probable diagnosis. In older children the most important points indicating that disease are: the presence of some of the stigmata of degeneration (page 757), a history of previous attacks, a distinct aura preceding the seizure, or a sudden onset with a cry or fall, biting of the tongue, a tonic spasm preceding the clonic, and, finally, perfect recovery in the course of a few hours after the attack. Convulsions which come on with high fever, even though a patient may have repeated attacks, are seldom epileptic. However, in some cases only prolonged observation can enable one to decide positively whether or not epilepsy is present.

Convulsions occurring in brain disease, except acute meningitis, are not as a rule accompanied by any marked rise in temperature. Focal symptoms are often present, such as localized paralysis or rigidity, changes in the pupils, and strabismus. The convulsive movements are frequently limited to one side of the body. It should, however, be borne in mind that unilateral convulsions, even when repeated, do not always mean a local lesion, as I have seen proved by autopsy more than once. In hæmorrhage or meningitis, convulsions are likely soon to recur. In tumour they may recur after a longer interval.

Convulsions may be thought to indicate the onset of some acute disease when they occur in a child over two years old, and when they come on suddenly or with only slight premonition in a child previously well; but the most important point is that they are accompanied by a high temperature,—104° to 106° F. Acute meningitis is the only other condition likely to produce these symptoms. Whether the convulsions mark the onset of lobar pneumonia, scarlet fever, malaria, or some other disease, can be determined only by carefully watching the patient's symptoms for twenty-four or thirty-six hours.

In convulsions depending upon some disorder of the alimentary tract, one may get a history of chronic constipation, of improper feeding, and in nursing infants sometimes of passion, or even intoxication, in the wet-nurse. Convulsions are so frequently due to digestive derangements that the condition of these organs should be one of the first things to be looked into.

Examination of the urine should never be omitted in any case of convulsions of doubtful origin, even where no dropsy is present. This, both in infants and older children, is too often overlooked. Asphyxia may be suspected in the case of convulsions occurring in the newly born, late in pneumonia, in some cases of pertussis, in spasmodic or membranous laryngitis, or in laryngismus stridulus. Dentition and worms should be considered among the least probable, never as the most probable, causes of

reflex irritation, and should not be so accepted without positive evidence. Worms are so rare in infancy that at this period they may be practically ignored. Dentition seldom, if ever, causes convulsions except in patients who are markedly rachitic. In all cases of convulsions of doubtful or obscure origin occurring in infants, rickets should be suspected as the underlying cause, and the child carefully examined for other evidences of that disease.

Prognosis.—This depends upon the age of the patient and the cause of the convulsions. Idiopathic or reflex convulsions are rarely dangerous to life except in very young or in rachitic infants. In such patients death from convulsions is not at all uncommon. Convulsions occurring at the onset of acute febrile diseases are seldom fatal, and not often serious; they may not even indicate an unusually severe type of the disease. Especially fatal are the convulsions of pertussis and of asphyxia when they occur late in any form of laryngeal or pulmonary disease. In nephritis, while always serious, convulsions are by no means invariably fatal. The conditions during an attack which should lead one to make a bad prognosis are when the convulsions are prolonged or recur frequently; also the presence of very great prostration, a feeble pulse with cyanosis, or deep stupor.

In the prognosis one must take into account not only the immediate result of the attack, but its possible outcome. Except where convulsions mark the beginning of epilepsy, they are much less serious than they are generally supposed by the laity. In a highly nervous or susceptible child a convulsion may often mean no more than an attack of severe migraine in an older person. Such are undoubtedly most of the attacks seen in practice. Permanent injury to the brain, simply as a result of an attack, although possible, is still rare. But when convulsions are repeated the development of epilepsy is to be feared. There is little doubt that some cases of epilepsy have their origin in attacks of convulsions, which in the beginning were the result simply of digestive derangements; by a constant repetition of the exciting cause the convulsive habit finally becomes established. This possibility is therefore to be borne in mind in all cases where children have had several convulsions, although it is unusual that this result is seen. The farther apart the attacks are and the more definite the exciting cause, the less likely is this to be the case.

Treatment.—Summoned to a child in convulsions, it is a physician's duty to go at once and remain with the patient until the attack has subsided. He should take with him chloroform, a hypodermic syringe with morphine, and a solution of chloral. In order to treat convulsions intelligently one must have in mind the prominent pathological conditions. These are acute cerebral hyperæmia, a more or less severe asphyxia with pulmonary congestion, an overtaxed right heart, and in fact a tendency to congestion of all the internal organs. The nervous centres are in a condition of such unnatural excitability that the slightest irritation may bring

on convulsive movements when they have temporarily subsided. The patient should therefore be kept perfectly quiet, and every unnecessary disturbance avoided. Cold should be applied to the head—best by means of an ice cap or cold cloths—and dry heat and counter-irritation to the surface of the body and extremities. The time-honoured mustard bath causes so much disturbance of the patient that it may well be dispensed with and the mustard pack (page 52) substituted. The feet may be placed in mustard water while the child lies in its crib. The mustard pack and footbath should be continued until the skin is well reddened. The degree to which counter-irritation of the skin should be carried will depend upon the condition of the pulse and the cyanosis.

In controlling convulsions the three remedies which may be depended upon are the inhalation of chloroform, morphine hypodermically, and chloral by the rectum. Chloroform is undoubtedly the most reliable remedy for an immediate effect, and should be used even in the youngest infant. At the same time that it is being administered, chloral should be given *per rectum*. The initial dose should be, at six months, four grains; at one year, six grains; at two years, eight grains, dissolved in one ounce of warm milk. It should be injected high into the bowel through a catheter, and prevented from escaping by pressing the buttocks together. It may be repeated in an hour if necessary. The effect of the drug is generally obtained in twenty minutes. If, in spite of the chloral, the convulsions show a marked tendency to continue as soon as the chloroform is withdrawn, or if the enema of chloral has been expelled, morphine should be given hypodermically. Where the heart's action is weak, this is probably the best of all remedies. Objections are urged against it only by those who have had no experience with its use. To a well-grown child two years old, $\frac{1}{16}$ of a grain may be given; one year old, $\frac{1}{24}$ of a grain; six months old, $\frac{1}{48}$ of a grain. This dose may be repeated in half an hour if no effect is seen. The tolerance of opium in cases of convulsions is very marked, and sometimes double the doses mentioned may be required. The only other agent of much value is oxygen. I have seen convulsions which continued in spite of all other means, yield immediately to oxygen. This is most likely to be valuable in cases of convulsions due to asphyxia.

When once under control, the recurrence of the convulsions may be prevented by keeping the patient for two or three days under the influence of chloral with bromide of sodium, the amount of chloral being gradually reduced. If it is badly borne by the stomach and not easily retained by the rectum, either antipyrine or phenacetine may be used with the bromide. Where there is a strong tendency to recurrence of the convulsions, urethan is sometimes even more efficient than chloral. It may be given in the same or in slightly larger doses.

As soon as the convulsions have ceased, the cause should be sought

and treated. In infancy it is wise in every case to irrigate the colon thoroughly with warm water, to remove any possible source of irritation. If there is reason to suspect the presence of indigestible food in the stomach, this may be washed out. Much more frequently it is in the intestines, and free purgation by calomel is advisable. If there is high temperature, this should be reduced by the cold bath or pack. Secondary attacks are to be prevented by careful feeding, by improving the general nutrition by means of fresh air, iron, cod-liver oil, and phosphorus. The last two are especially valuable in cases due to rickets.

EPILEPSY.

Epilepsy may be defined as a disease in which there is an established disposition to convulsions of a certain type, with loss of consciousness, which have recurred until a habit of convulsions has become fixed.

A distinction must be made between cases of so-called "idiopathic" epilepsy and those which are secondary to a definite lesion of the brain, such as tumour, sclerosis, or abscess. Convulsions of the latter character are designated as "symptomatic" epilepsy, and are discussed in connection with the various diseases in which they occur. The nature of the attack may, however, be identical in both varieties, and may not differ from an ordinary attack of convulsions or eclampsia.

The proportion of idiopathic cases in children is not so large as was formerly supposed; for many of these have been shown to depend upon lesions once overlooked, particularly infantile cerebral paralyses of a mild type.

Etiology.—From a consideration of 1,450 cases of epilepsy, Gowers states that 12 per cent begin in the first three years of life, and 46 per cent between ten and twenty years. The greatest tendency to the development of the disease is shown about the time of puberty. Females are rather more liable to be affected than males, although the difference in sex is slight. Heredity plays an important rôle in the production of the disease. In one third of the cases, according to Gowers, there is a family history either of epilepsy or insanity. Not infrequently more than one child in the family is affected. All hereditary nervous diseases predispose to epilepsy, but it is a question whether other hereditary diseases have any special influence.

Not very infrequently epilepsy may be traced to convulsions occurring during infancy. In what proportion of the cases this is true it is impossible to state with accuracy. Infantile convulsions are very common, and usually the cause which produces them is a transient one. The proportion of such cases which develop epilepsy later in life is certainly small. In the second and third years, however, the occurrence of convulsions not infrequently marks the beginning of true epilepsy. Given a strong predisposition to epilepsy, it is easy to see how early infantile convulsions so often

associated with rickets may have been the first of the epileptic series. The first seizure is sometimes traceable to fright, great excitement, heat-stroke, or blows or falls upon the head even without any gross lesion. It may follow any of the acute diseases of childhood, particularly scarlet fever, rarely measles or typhoid. In none of these, however, is it often seen. As reflex causes may be mentioned intestinal worms, phimosis, adenoid vegetations of the pharynx, delayed or difficult menstruation, and masturbation. Most of these are rare causes, but they may be sufficient to produce the disease where a strong predisposition exists. Syphilis may be the cause of epilepsy even when there is no local disease of the brain.

Among the most important factors in producing a paroxysm, is intestinal putrefaction associated with chronic constipation and chronic intestinal indigestion. This subject has been lately investigated with great care by Herter and Smith,* who studied 238 specimens of urine from 31 epileptics. In 72 per cent of their observations there was unmistakable evidence of excessive intestinal putrefaction, as shown by the presence of ethereal sulphates in the urine in large amount, just before the occurrence of the paroxysm. The inference seems warranted that this intestinal condition was closely connected with the epileptic seizures. The statement of Haig, that there is an excessive elimination of uric acid preceding the paroxysm, was not borne out by the observations of Herter and Smith. The association of intestinal putrefaction with seizures of epilepsy is very important as furnishing a clew to the management of many of these cases. I believe it to be one of the most important etiological factors in cases occurring in children, particularly as an exciting cause of the first attacks.

Pathology.—It is not within the scope of this work to discuss the various theories which have been advanced. The following are the conclusions reached by Gowers: †

"The muscular spasm is to be regarded as the result of the sudden overaction (discharge) of nerve cells, the violent liberation of nerve force, and the sensations which the patient experiences before losing consciousness must be due directly or indirectly to the same cause. The disease which excites convulsions is most frequently at the cortex, and when organic disease causes convulsions that begin locally, the disease is almost invariably at the cortex. In idiopathic epilepsy the convulsions sometimes begin in this way, and this suggests very strongly that in such cases the change occurs in the cortex. Epilepsy must then be regarded as a disease of the gray matter, most frequently of the gray matter of the cortex."

---

* New York Medical Journal, August and September, 1892.

† Diseases of the Nervous System, American ed. 1888, p. 1098.

While there is pretty general agreement that the seat of the morbid changes in true epilepsy are in the cortex, but little is yet definitely known as to the nature of these changes. Van Gieson has published * some very careful observations made upon portions of the cortex removed at a surgical operation from two epileptic patients. In one of these the disease was primarily due to a foreign body; in the other, to an old cicatrix. The conditions found represent the earlier changes of the disease, and were essentially the same in both cases. There were degenerative changes in certain of the ganglion cells, which in places had resulted in almost complete dissolution of these cells. In addition there was a distinct hyperplasia of the neuroglia tissue. Diffuse neuroglia sclerosis starting from the focus of disease has been reported by certain French writers—Marie, Féré, and Chaslin.

Symptoms.—Two distinct types of epileptic seizures are met with: the major attacks, or *grand mal*, in which there are severe convulsions lasting from two to ten minutes, with loss of consciousness, etc.; and minor attacks, or *petit mal*, in which the convulsive movements are slight and may be absent, and in which the loss of consciousness is often but momentary. Between these two extremes all gradations are seen.

Grand mal.—The onset may be sudden, without premonition, or it may be preceded by certain prodromal symptoms known as the aura. The aura may be motor, such as a local spasm of the hand, face, or leg; or sensory, such as numbness and tingling in any part of the body, or some abnormal sensation rising gradually to the head, at which time loss of consciousness occurs. The variety of sensations described by patients as indicating an attack is endless. There may be a sensation in one finger, in the face, tongue, eye, or in any part of the body; or the warning may be of a general character, like a tremor or a shivering sensation, or a feeling of faintness. There has also been described a visceral or pneumogastric aura, in which there is epigastric pain, sometimes nausea, and a sensation of a ball in the throat; or there may be palpitation, or cardiac distress. There may be general giddiness or vertigo, or a sensation of fulness in the head; or feelings of strangeness, or a dreamy, dazed condition; and, finally, the aura may have reference to any of the special senses, most frequently to sight. Sparks may appear before the eyes, or flashes of light or colour, or strange objects may be seen; or there may be a momentary loss of hearing; or strange sounds may be heard. In most cases the aura is peculiar to the individual, whose attacks are likely to be preceded by the same symptoms.

At the beginning of the seizure the face becomes pale, the pupils widely dilated, the eyes rolled up in their orbits and fixed. Speedily there is loss of consciousness. Simultaneously with these symptoms, or imme-

* New York Medical Record, April 24, 1893.

diately following them, there occurs a violent tonic spasm to which are due the characteristic symptoms of the early part of the seizure—viz., the fall, cry, biting of the tongue, cyanosis, and evacuation of the bladder or rectum. The fall is forcible, violent; in fact, the patient is precipitated usually forward, and frequently suffers injury, never sinking down as in a faint. The head is often strongly rotated to one side. The position of the hands is that assumed in tetany. The cry is a hoarse, inarticulate sound, not very loud, and is due to forcible expiration, owing to spasm of the muscles of respiration with the glottis partly closed. The cyanosis is the result of tonic spasm of the muscles of respiration; it may be quite intense, so that the face is livid, bloated, and the features distorted. The spasm of the muscles of mastication causes the biting of the tongue. Evacuation of the bladder and rectum may result from contraction of their walls, or from spasm of the abdominal muscles. The violence of the muscular spasm in this stage may be very great; it has caused fracture of bones, rupture of muscles, and even dislocation of joints.

The stage of tonic spasm may be only momentary, the patient passing almost at once into the stage of clonic convulsions. The usual duration is from ten seconds to half a minute. In the stage of clonic spasm which follows, the symptoms are those of an ordinary attack of convulsions. The muscular contractions are violent, and there is often frothing at the mouth. Gradually the muscles of respiration relax, air enters the chest, and the cyanosis passes off. After the clonic spasm has continued for a variable time—from two or three minutes to half an hour—the muscular contractions become less and less frequent, and finally cease altogether. In a few minutes the patient may regain consciousness, look vacantly around, and in a dazed way perhaps ask what has happened, he being completely oblivious to all that has occurred. More frequently, however, he passes at once into a deep sleep, which continues for an hour or more, but from which he can be aroused. From this he usually wakens with a severe headache, which may continue for several hours. After this he often feels better than for several days preceding the attack. During the seizure the temperature may be elevated one or two degrees, but rarely more. The attack may be followed by a slight temporary paresis, or aphasia, hysterical phenomena, vomiting, and intense hunger. In very rare cases the urine may contain a trace of sugar.

Petit mal.—The minor attacks of epilepsy may present a very great variety of symptoms, and at times it is almost impossible to decide that these are epileptic, except from their periodical occurrence. They pass under the names of "spells," "attacks of dizziness," "fainting turns," etc. The most striking thing which stamps them as epileptic is the loss of consciousness, and this may be of short duration, sometimes only momentary, and so pass unnoticed. In some cases it is absent altogether. There is no fall, but there may be a slight dropping of the head, a fixed stare for a

moment or two, and that is all. This may or may not be preceded by an aura. After such a mild attack the patient's mind may be somewhat confused, and he may do or say strange things. All sorts of curious acts have been performed in an automatic way by patients in the condition which follows an attack of epilepsy, which may perhaps be regarded as part of the attack. In rare instances even acts of violence may be done.

The mental condition of epileptics.—In this connection a careful distinction must be made between cases in which epilepsy is secondary to some organic brain disease, such as infantile cerebral palsy, which may itself be a cause of mental impairment, and the mental disturbances seen in cases of idiopathic epilepsy. The children who are the subjects of the latter disease, and who are perfectly normal mentally, are certainly few. All degrees of disturbance may be seen, from those who are simply dull, apathetic, backward in development, and uncontrollable in temper, to those who are melancholic, idiotic, and even maniacal. The earlier in childhood epilepsy develops, the greater is usually the mental disturbance seen, because of the effect of the seizures upon the brain during its period of active growth. Speech and all mental development may be greatly retarded. The more frequent and more severe are the attacks, the more marked are the mental symptoms present.

Symptomatic epilepsy.—This occurs most frequently in children as a sequel of cerebral palsy, usually with hemiplegia, and it may follow either the congenital or acquired form. Epilepsy may come on at any time after the onset of the paralysis—from a few months to five or six years. At first the attacks may be separated by long intervals, but they gradually become more frequent as time passes. The convulsions in post-hemiplegic epilepsy begin, as a rule, on the paralyzed side, and for a long time they may be confined to that side; but later they may become general, in which cases they are indistinguishable from attacks of idiopathic epilepsy. Severe seizures are more likely to be seen than are the mild ones.

Course of the disease.—This is extremely irregular. In most cases seizures at first occur at long intervals, of perhaps a year, but later they become more and more frequent. Either the mild or the severe attacks may be first seen, and may remain throughout as the only type present, or they may be associated in the same case. There are most frequently seen, occasional major attacks with a large number of minor ones. The interval between the epileptic seizures in most cases is from two to four weeks, although they may be of daily occurrence. Sometimes three or four seizures will follow one another closely, and then there will occur a long interval of immunity. The seizures may come on either during sleep or in the waking hours, and in some cases for a long time they may occur only in sleep. Such cases present peculiar difficulties in diagnosis, and are often long unrecognised as epileptic. The general health of patients may be quite normal.

Death rarely, if ever, results from epilepsy, except from some accident at the time of the seizures, or from the condition known as the *status epilepticus*; in this the attacks come on with great frequency and severity, the patient at times passing rapidly from one convulsion into another, the temperature rising to 105° or 106° F., and death occurring either from exhaustion, owing to the severity of the convulsions, or from coma.

Diagnosis.—In most cases there is little difficulty in recognising the major attacks when they occur by day. Nocturnal attacks may be diagnosticated by the cry, the biting of the tongue, blood upon the pillow, sub-conjunctival extravasation, evacuation of the bladder or rectum, and the severe headache. Minor attacks present the greatest difficulties, and a positive diagnosis is often impossible until the patient has been watched for a long time. The most important points to be noted are sudden pallor, dilatation of the pupils, temporary loss of consciousness, or simply mental confusion, and sometimes the evacuation of the bladder. The duration of the attack is shorter than is usual in an ordinary faint. The difficulty of distinguishing epilepsy from hysteria rarely occurs in childhood.

It is not always possible to distinguish between secondary or symptomatic epilepsy and the idiopathic or hereditary form, particularly if the case comes under observation late in the course of the disease. The points which go to establish the first form are: that the convulsive movements are partial, or limited to one side; that when they are general, they always begin in the same part of the body; or that there is a history of partial or unilateral attacks for some time before the occurrence of any general convulsions. It is important in all cases to examine the patient carefully for signs of an old hemiplegia, the symptoms of which may be so slight as to be readily overlooked. A marked increase in the reflexes of one side is, according to Sachs, quite as conclusive evidence as a distinct weakness of the arm or leg. In idiopathic epilepsy some of the stigmata of degeneration (page 758) are usually present. The sudden development of epileptic seizures in a child previously healthy, and in whom there is no hereditary history of the disease, should always arouse the suspicion of organic brain disease, especially tumour; and if there are besides, severe headache, vomiting, and optic neuritis, the existence of tumour is reasonably certain.

Prognosis.—The danger to life in epilepsy is very slight. Death is generally due to some accident, particularly drowning, at the time of a seizure. The tendency to spontaneous cessation of the attacks is small, while the tendency to recurrence is very great.

The prognosis in any given case depends upon the cause of the disease and the duration of the symptoms. Where the cause can be removed, and where the symptoms have lasted less than a year, the prospects of per-

manent cure are fairly good. This is particularly true of cases in which the epilepsy clearly depends upon gross errors in diet, with chronic intestinal indigestion. In such cases, if the patient can be placed under proper control and dietetic measures well carried out, the development of chronic epilepsy can be arrested in a considerable number of cases. If, on the contrary, the hereditary tendency to the disease is marked, if the epileptic seizures have developed apart from any adequate exciting cause, and if they have continued untreated or in spite of treatment for two or three years, the symptoms may perhaps be relieved, but there is no prospect whatever of permanent cure. In the cases also which are due to local irritation, like that resulting from an old meningeal hæmorrhage, the prognosis is invariably bad, and only temporary relief is to be expected. A few cases of traumatic epilepsy have been cured and many have been greatly improved by a surgical operation.

Treatment.—The first indication is to remove the cause where one can be found. If in the male phimosis exists, or other evidence of genital irritation, circumcision should be done, or the prepuce retracted and adhesions broken up. Adenoid growths of the pharynx should be removed, and likewise every other cause of reflex irritation. Particular attention should be given to the digestive organs. The most hopeful cases are those associated with acute and chronic disturbances of digestion, especially chronic intestinal indigestion with constipation. These cases are to be managed like others of the same sort in which epileptic attacks are not present (page 368). Meat should be allowed once a day and in moderate quantity. Milk should be given, diluted if necessary, also kumyss and matzoon. Green vegetables, except peas and beans, may be given freely; also all fresh fruits. Tea, coffee, and alcohol in every form must be absolutely prohibited; also potatoes and oatmeal. The most careful attention should be given to the bowels. Under no circumstances should a condition of chronic constipation be neglected. A dose of calomel once a week and intestinal irrigation two or three times a week are of great value in many cases. Where the symptoms of intestinal putrefaction are marked, borax is at times of decided value—two grains three times a day to a child of five years—or salicylate of sodium, salol, or the benzoate of sodium may be given; the dose of each being from two to ten grains, according to the age of the child, after each meal. The general hygiene of the patient must receive careful attention. He must lead a simple, regular life, as much as possible out of doors, away from the excitements of a large city, or from association with many children, and in short the nervous system should be kept as quiet as possible.

All the foregoing means of treatment are of equal importance with the use of specific drugs. The most common mistake is to rely only upon drugs, ignoring the other measures mentioned. It not infrequently happens that drugs are without any avail when they are the only means of

treatment employed, whereas in conjunction with other measures marked improvement is seen.

The bromides are unquestionably the best means of combating the epileptic habit. Either the sodium salt alone or a combination of the sodium and ammonium bromides is to be preferred. The purpose should be to give the smallest doses which will control the seizures. Children require proportionately larger doses than adults, and in most cases a child of five years will need from twenty-five to fifty grains a day. Seguin's* method of administering the bromides is largely followed in New York, and is of great value. It is to give the larger part of the quantity for twenty-four hours, shortly before the time when the seizures have usually occurred; in the interval to give much smaller doses, and in all cases to give the dose largely diluted,—in from six to eight ounces of water. He gives a full dose early in the morning, and, where the seizures are apt to come at night, one at bedtime.

Cases of *petit mal* are especially difficult to control. For such there is often an advantage in combining belladonna with the bromides. In all cases the treatment must be continued for a long time if anything is accomplished. The bromide should be gradually reduced after the attacks are controlled, but must be given in moderately large doses for at least two years after the seizures have ceased. The addition of borax seems occasionally better than the bromides alone in cases where there is excessive intestinal putrefaction. Sometimes the combination of chloral or antipyrine with bromides is advantageous, particularly if the latter are badly borne or cause an annoying amount of acne. Seguin states that he has been able to control the acne in many cases by giving at the same time moderate doses of arsenic. Other drugs occasionally useful as adjuvants to the bromides are strychnine and digitalis.

The surgical treatment of epilepsy has of late attracted much attention. An operation is to be considered in cases in which the paroxysms are very frequent and severe, and when there is present a definite local cause, such as an old fracture of the skull, or where epilepsy has followed an injury to the head even without fracture. Sachs sums up the present status of this question as follows: "In a case due to a traumatic or organic lesion an early operation may prevent the development of cerebral sclerosis. If early operation is not done, the occurrence of epilepsy is a warning that secondary sclerosis has been established and an operation may prevent it from increasing. Operation must include the removal of the diseased area; here, if all other parts are normal, a cure may result. Under favourable conditions a few cases of epilepsy may be cured by surgery and many more improved."

The education of epileptic children is a subject of great difficulty and is often neglected. There are many reasons why it is impracticable to

* New York Medical Journal, March 29, 1890.

send them to ordinary schools, and it is very desirable that special schools for them should be established.

The management of the attack.—Abortive measures are sometimes successful in cases with a distinct aura, the most reliable being the inhalation of nitrite of amyl. While the seizure lasts, the patient should be prevented from injuring himself. The clothing should be loosened, a spool or cork should be placed between his teeth to protect the tongue, but no effort made to restrain his movements unless he is liable to do violence to himself. An epileptic child should never be without some companion.

TETANY.

This is a condition characterized by tonic muscular spasm, which may be intermittent or continuous. It usually affects the muscles of the extremities, especially the hands and feet, more rarely the neck, face, and trunk. When limited to the hands and feet it is known as carpo-pedal spasm or arthrogryposis; and although sometimes classed separately, this seems to be really only one manifestation of the same general condition. In infants, tetany is very frequently associated with laryngismus stridulus, this being present in fully two thirds of the cases; but in older children this association is quite rare. General convulsions occur in from twenty to thirty per cent of the cases. Tetany is rare in this country, as shown by the fact that Griffith * in 1895 could find reported only fifty cases, of which thirty-eight were in children.

Etiology.—While tetany may occur at any age, it is most frequent in infancy. Of eighty-seven cases reported by Barthez and Sanné, fifty per cent were observed in the first two years, twenty per cent from three to six years, and twenty-five per cent from twelve to fifteen years. Of the cases in children collected by Griffith, sixty-six per cent were under two years of age. In infancy males are much more frequently affected; but when the disease occurs in older children, females seem much more liable to it. Tetany rarely occurs as a primary disease. It is most frequently associated with rickets; in fact, rickets is almost invariably found in the infantile cases. It sometimes occurs with chronic diarrhœa and with marasmus. It has been known to follow broncho-pneumonia, pertussis, typhoid fever, rheumatism, and measles. Of the exciting causes, the most frequent one is some irritation in the gastro-enteric tract. This may be the products of chronic indigestion, or of acute diarrhœa, worms, and sometimes even intussusception. Attacks in older children are frequently ascribed to cold. In girls, tetany may occur at the time of puberty, especially where menstruation is delayed; it has followed removal of the thyroid gland; and it has been known to occur epidemically in much the same way as chorea.

* American Journal of the Medical Sciences. February, 1895.

Pathology.—Up to the present time no constant anatomical lesions have been demonstrated in tetany. The circumstances in which it occurs, its symptoms and course, all indicate that it is a neurosis probably dependent upon disturbances of nutrition in the nerve cells of the spinal cord and medulla.

Symptoms.—The spasm may occur quite suddenly, or it may be preceded by various sensory disturbances, such as pain, numbness, or tingling. The upper extremities are usually first affected, the spasm gradually becoming more severe and finally involving the lower extremities. Both sides of the body are equally affected. The position assumed by the hands is very characteristic: The fingers are flexed at the metacarpophalangeal joint and the phalanges extended; the thumbs are adducted almost to the little finger; the wrist is flexed at an acute angle, and the whole hand drawn somewhat to the ulnar side (Fig. 108). No motion is allowed at the wrist, but movements at the elbow and shoulder are usually normal. The feet are strongly extended, sometimes in the position of typical equino-varus. The first phalanges of the toes are flexed, and the second and third rows extended; the plantar surface is strongly arched, and the dorsum of the foot is very prominent, standing out like a cushion. The typical position of the feet is well shown in the accompanying illustration. There are rigidity of the muscles of the calf and tension of the plantar fascia. The tendo-Achillis stands out prominently. Motion at the hip and knee is generally free. The spasm in many cases is limited to the hands and feet; more rarely the muscles of the thigh, usually the adductors, may be involved. I have seen three or four cases in which the spasm affected only the cervical muscles, producing marked opisthotonus. This form is generally mild, and may be associated with marasmus. In very rare cases the muscles of the trunk, the face, or the eye may be involved.

Where the spasm is intermittent, and in some cases where it has subsided, it may be excited by making pressure upon the large nerve trunks and arteries of the parts affected. This is known as "Trousseau's symptom," and is characteristic of the disease.

Pain owing to the spasm is frequently present. It is usually sharp and lancinating, and may be so severe as to cause children to cry out. Pain is induced by any attempt to overcome the spasm, and sometimes it is constant. Other disturbances of sensibility are even more common than pain. There is no loss of consciousness and no fever. The spasm is generally continuous, although there may be periods of remission or even of intermission. When associated with laryngismus stridulus, the spasm is much increased during these attacks. The electrical reactions are as a rule increased, and the knee-jerk and cutaneous reflexes are exaggerated.

The duration of the disease is from a few days to several weeks. The mild form, which is usually seen in infants, in most cases passes away spontaneously in one or two weeks, although there may be relapses and

second attacks at variable intervals. The most important complication is general convulsions. These may come on at any time in the course of

Fig. 108.—Tetany, showing the characteristic position of the hands and feet, in a child two years old.

the disease. Spasm of the glottis may either precede or follow tetany. When associated they generally cease at the same time. Slight paralysis may follow or alternate with the spasm.

Diagnosis.—The diagnostic features of the disease are bilateral spasm—in infants usually limited to the hands and feet—without loss of consciousness, the spasm being increased or excited by pressure upon the nerves, exaggerated reflexes, and the presence of some previous disease, especially

rickets or some disorder of the intestines. The severe form may be mistaken for tetanus; but this is very rare except in the newly born; and trismus is the rule, and generally it is the first symptom. Trismus is extremely rare in tetany. From meningitis, tetany is distinguished by the absence of cerebral symptoms; from cerebral tumour, by the bilateral character of the spasm, the absence of headache and focal brain symptoms; from hæmorrhage, by the absence of cerebral symptoms; from malarial spasm, by the fact that it is constant, not intermittent.

Prognosis.—Tetany *per se* is not fatal, but death may result from the development of general convulsions or from the original disease which tetany complicates. Recovery is usually perfect, although Gowers states that in rare cases it has been followed by muscular atrophy.

Treatment.—The first indication is to remove the cause, and this in most cases is found in the digestive tract. If rickets is present it should receive the usual treatment, both dietetic and medicinal. If worms are suspected a vermifuge should be given. For the relief of the spasm; the hot bath is a most valuable remedy; friction may also be employed. Drugs which have the power of allaying spasm should be given,—chloral, bromides, and antipyrine. In the event of failure by these methods galvanism may be tried. After the attack the child's general nutrition should receive careful attention, to prevent relapses.

LARYNGISMUS STRIDULUS—SPASM OF THE GLOTTIS.

Idiopathic spasm of the glottis, or laryngismus stridulus, is a rather rare disease, and belongs especially to infancy. It is a pure neurosis, not often seen except in children who are rachitic. It is frequently associated with carpo-pedal spasm and with general convulsions. The disease is not to be confounded with ordinary spasmodic croup or catarrhal spasm of the larynx, which is of very frequent occurrence.

Spasm of the larynx may be seen in several conditions quite different from laryngismus stridulus. It forms one of the essential features of pertussis. It occurs both in infants and in older children from pressure upon, or irritation of, the pneumogastric or recurrent laryngeal nerve by a tumour in the mediastinum,—usually a tuberculous lymph node, or retro-œsophageal abscess. Reflex spasm of the larynx is also associated with enlarged tonsils, adenoid growths of the pharynx, and elongated uvula. There is a form of reflex spasm which occurs in the newly-born accompanied by crowing inspiration; this is not frequent, and is rarely serious.

Idiopathic spasm of the larynx is quite different from any of these. It is peculiar to infancy, the great proportion of cases occurring between the sixth and eighteenth months. Males appear to be more susceptible than females. The constitutional condition with which it is usually associated is rickets. In a large number of cases, but not in all, there is cranio-tabes. Many writers believe that laryngismus is invariably of rachitic origin. Of

fifty cases observed by Gee, there were found in all but two unmistakable evidences of rickets. The disease occurs in delicate infants who have been closely confined in warm rooms, and it is probably on this account that it is more often seen in the winter and spring than at other seasons. The exciting causes of this spasm may be a breath of cold air, or any form of nervous excitement, such as fright or crying. Sometimes it is induced by swallowing, and it may be traced to indigestion or constipation.

Pathology.—There are no anatomical changes in this disease. It is a pure neurosis, and it is generally believed to be of central origin, depending essentially upon imperfect nutrition of the motor centres of the spinal cord and medulla.

Symptoms.—The disease is often unnoticed by the parents until the attacks have become quite frequent, the first ones being mild, and the later ones more and more severe. Occasionally the very first paroxysms may be severe. The attack comes on suddenly. The child throws back its head, the face becomes pale, then livid, and for the time there is complete arrest of respiration. This continues for a few moments, during which the cyanosis deepens, and the child seems in great distress, making violent efforts to breathe. If the paroxysm is a severe one, the asphyxia may be so great as to lead to loss of consciousness, and it may even be fatal, or the attack may terminate in general convulsions. In milder attacks, after fifteen or twenty seconds the muscular spasm relaxes, the glottis opens, and a long, deep inspiration occurs, with the production of a crowing sound. Such attacks may occur as frequently as every fifteen or twenty minutes, or there may be only six or eight during the day. Between them the condition of the child may be normal, or carpo-pedal spasm may be present. It is important to note that in this disease there is not a stridor due to narrowing of the glottis, as in ordinary croup, but a condition of apnœa from its complete closure. Not all the paroxysms in the same case are equally severe. A child may have in the course of a day a great many mild attacks, but only a few severe ones. General convulsions are seen in over one third of the cases, and carpo-pedal spasm or tetany complicates a still larger proportion. While this is present in the interval, it is always increased during the attacks.

The duration of the disease varies from a few days to several weeks, or even months. In cases which terminate in recovery there is a gradual diminution in the frequency and severity of the paroxysms, until they finally cease altogether.

Prognosis.—This is good, except when there are general convulsions. The cases in which fatal asphyxia occurs are very rare. Usually with proper treatment marked improvement begins in the course of a few days.

Diagnosis.—This is to be made from catarrhal spasm of the larynx. The differential points have been mentioned under the latter disease

(page 440). Owing to the occurrence of paroxysms and the crowing sounds, the disease may be mistaken for whooping-cough, and in fact this diagnosis is not infrequently made by parents. A careful examination of the patient during the attacks, the absence of cough, and the frequent association of tetany, are sufficient to differentiate this from pertussis.

Treatment.—During the attack the object is to break the spasm. In mild cases this may be done by sprinkling water in the face. In severe cases inhalations of chloroform may be required, and even intubation. Between the attacks the patient should be given either bromide and chloral, or antipyrine. Sodium bromide, gr. v, and chloral, gr. ij, may be given every three or four hours to a child a year old until the frequency and severity of the attacks are controlled; afterward three times a day. My recent experience with antipyrine in this disease leads me to the belief that it is more effective than bromide and chloral. When the symptoms are severe, two grains of antipyrine may be given every four hours to a child a year old, the dose being gradually diminished as the symptoms improve.

The general treatment of the child is quite as important as drugs directed toward relieving the spasm. Cold sponging should be used in every case unless it occasions so much fright as to increase the number of paroxysms. Careful attention should be given to the diet. Children should be kept in the open air as much as possible, and those who are rachitic should receive phosphorus. Cod-liver oil is needed in most cases. Any source of local irritation, such as enlarged tonsils, elongated uvula, or adenoid growths, should be removed; for, if not the actual cause of the attack, they may be the means of aggravating the symptoms. In all cases the treatment should be continued for several weeks after the paroxysms have subsided.

CHOREA—SAINT VITUS'S DANCE.

Chorea is a functional nervous disease characterized by aimless, irregular movements of any or all the voluntary muscles. Choreic movements are of a somewhat spasmodic character, often accompanied by an apparent or real loss of power in the groups of muscles affected, and by a mental condition of extreme irritability.

Etiology.—Chorea is most frequently seen between the ages of seven and fourteen years. Of 146 cases, 6 were under five years, 72 between five and nine years, and 68 between ten and fourteen years. The youngest case of which I have record was that of a child four years old. It is extremely rare before the third year, although it may occur even in infancy, and in a few recorded cases it was undoubtedly congenital. My own observations coincide with those of nearly all writers, that the disease is more than twice as frequent in females as in males. While chorea may be seen

at all seasons, it is much more frequent in the spring months. Of 717 attacks studied by Lewis (Philadelphia), the largest number began in March, and the next largest number in May; in my own cases May stood first.

The relation of chorea to rheumatism is of much importance, and has during late years attracted a great deal of attention. Thus far the investigations of different writers have given results which are somewhat contradictory. Some have found evidences of rheumatism in but a small proportion of the cases—in not more than 5 or 10 per cent—while the statistics of others have placed the percentage of rheumatism as high as 50 or even 60 per cent. It is rather striking that the statistics of neurologists, almost without exception, have given a very much smaller percentage of rheumatism in choreic cases than those taken from children's clinics and hospitals. The question hinges largely upon what is to be admitted as evidence of rheumatism in a child; if cases of acute articular inflammation only, then the number will be very small; if subacute cases with joint swellings are included, the proportion will be considerably larger; while if we admit cases of acute endocarditis without articular symptoms, and those of articular pains and joint stiffness but without swelling, the proportion will be very much increased. My own belief is that there is a very close connection between chorea and the rheumatic diathesis as manifested by all the symptoms above noted, and accompanied by a family history of rheumatism. On careful scrutiny, the number of cases of chorea in which unmistakable evidence of this diathesis is found, is very large, including in my own observations over one half the cases. There seems, then, to be a large group of cases which may be classed distinctly as rheumatic chorea. There are, however, many others in which no such element can be found.

My associate, Dr. F. M. Crandall, has analyzed 146 cases of chorea treated by us at the New York Polyclinic and elsewhere, with the following results: Of 111 cases in which the question of rheumatism was investigated there was a definite history of it in 63. In 41, rheumatism occurred before the chorea; in 13, the first evidence of rheumatism was coincident with the chorea; and in 9 it first occurred subsequently to the chorea, usually within three months. In about one third of the cases, attacks of rheumatism occurred during or subsequent to the chorea as well as before it. It may then be stated that previous rheumatism was evident in 37 per cent, concurrent rheumatism in 24 per cent, and subsequent rheumatism in 15 per cent of the cases. Excluding cases mentioned twice, and also all those in which there was a history only of "growing pains," there was evidence of articular rheumatism in 56·7 per cent of the cases. Many of these patients have been under observation now for several years, and it has been interesting to see, as time has passed, how the evidences of the rheumatic diathesis have multiplied the longer the cases were followed.

In the above statistics only articular symptoms have been accepted as

evidence of rheumatism. If the cases of endocarditis without articular symptoms were included, as I think they might fairly be, it would raise the proportion of rheumatic cases still higher. The great proportion of cardiac murmurs persisting after chorea, if not all of them, should, I believe, be classed as rheumatic, even if no articular symptoms have been present.

Overpressure in school is often an important factor in the production of chorea, as has been shown by Sturges (London). Anæmia, if not an essential factor, is certainly a very important one, and the great proportion of cases present very distinct evidences of it. Chorea may develop as a sequel of any of the infectious diseases, more particularly scarlet and typhoid fevers. It is seen quite often in cases of chronic malarial poisoning. Among the reflex causes may be mentioned phimosis, either lumbricoids or pinworms, delayed menstruation, and ocular defects,—although the latter more frequently cause a local spasm of the muscles of the eyes, which can hardly be considered choreic. It has been claimed that chorea may result from the reflex irritation arising from adenoids of the pharynx and enlarged tonsils. Whether this is directly or only indirectly a cause is not evident. The association of the two conditions is not very infrequent.

Hereditary influence is of considerable importance in the production of chorea. It is much more frequent in children of neurotic families, and very often several successive generations, or several children in the same family, may suffer from the disease.

The exciting cause of chorea in a certain proportion of cases is fright; occasionally it arises from imitation, and the disease has been known to occur epidemically in institutions. Choreiform movements may follow hemiplegia. Chorea and epilepsy may be associated in the same patient, or one disease may follow the other.

The causes which underlie the occurrence of chorea therefore, seem to be a rheumatic diathesis, a neurotic constitution, anæmia, and some severe disturbance of general nutrition. When these predisposing factors are present, an attack may be induced by many things. The greater the predisposition the less important may be the exciting cause. A very large number of the cases of chorea are in persons who present distinct evidences of rheumatism, although the explanation of this relationship is not yet understood. In another group the neurotic element predominates, and in these there may be no connection whatever with rheumatism.

Pathology.—The exact pathology of chorea is at the present time not settled. The seat of the morbid process is undoubtedly the central nervous system, probably the motor areas of the cortex. Like epilepsy, chorea may follow organic brain disease, especially hemiplegia from cortical lesions. In some severe cases which were fatal, owing to association with acute endocarditis, capillary emboli have been found in the

brain. They have, however, often been absent, and probably explain but a small number of cases, if, indeed, they explain any. The fact that in the great majority of the cases of ordinary chorea, complete recovery occurs in the course of a few weeks or months, speaks strongly against any important structural change in the nervous centres. It seems much more in harmony with what we know of the disease clinically, to seek an explanation of the symptoms in vascular changes in these parts, having their origin in disturbances of nutrition.

Symptoms.—An attack of chorea generally comes on gradually. At first the child is often considered simply as unusually nervous; if at school, there may be noticed a difficulty in writing, drawing, or in using the hands for other delicate operations. At home, the child is continually dropping things, has difficulty in feeding himself, sometimes in buttoning his clothes, and very frequently he is not brought to the physician until the symptoms have lasted a week or two. Sometimes the legs are first affected, and a history is given of frequent falls, a stumbling gait, difficulty in going upstairs, etc. At other times the spasm is first seen in the facial muscles, with disturbance of articulation, twitchings of the eye muscles, and the child may be punished for making grimaces. In most cases the spasmodic movements soon extend to all parts of the body. According to Starr, they remain limited to one side of the body (hemichorea) in about one third of the cases. When fully developed, the movements of chorea are quite unmistakable. They are irregular, jerking, spasmodic, never rhythmical, rarely symmetrical, and vary in intensity from an occasional muscular contraction to almost constant motion. The movements are not under the control of the patient's will, and are usually intensified by efforts to suppress them. They are increased by excitement, embarrassment, or fatigue, but do not as a rule continue in sleep.

Very often there is some weakness of the affected muscles, which may be so great as to lead to the suspicion that actual paralysis exists. Not infrequently I have had patients brought to the clinic for supposed paralysis, either of one extremity or of one side of the body, where the choreic movements have not been severe enough to attract the attention of the mother. This paralysis usually disappears in the course of a few weeks.

In severe forms of chorea the patient may be unable to help himself or even to walk, from the inability to co-ordinate muscular movements. The symptoms may be so intense as even to endanger life. Such cases, however, are dangerous, not from the choreic movements, but from the acute endocarditis with which they are frequently associated.

The mental condition of choreic patients is one of marked irritability. They are fretful, emotional, easily provoked to tears or laughter, and often very difficult to control. In extreme cases a mental disturbance bordering upon acute mania has been observed. All degrees of speech disturbances may be met with, from the slight difficulty in articulation

due to inability properly to control the movements of the tongue and lips, to a condition in which speech is almost impossible. In rare cases speech has been temporarily lost. Heart murmurs are frequent in chorea. Some of these are of anæmic origin, some possibly are due to chorea of the heart-muscle itself—although this is a matter of some uncertainty—but a large number, probably the majority, are due to concurrent endocarditis, as is shown by the fact that they are permanent, and are followed by all the signs of organic heart disease. During every attack the heart should be closely watched, especially in children in whom there is a strong predisposition to rheumatism.

The urine in chorea has recently been studied with care by Herter and Smith, who have shown that in very many cases there is an excessive elimination of uric acid. This is neither the cause nor the effect of the chorea, but is to be regarded as evidence of a profound disturbance of nutrition, of which the choreic movements are but another manifestation.* The general condition of choreic patients is usually much below normal. They are anæmic; the appetite is poor, often capricious; they sleep very badly; they suffer frequently from headaches; they are easily fatigued by slight muscular exertion; and in short they have all the symptoms of a greatly disturbed nutrition.

Course and Duration.—The ordinary form of chorea tends to spontaneous recovery in from six to ten weeks. Exceptionally it may last for three or four months. In a small number of cases the disease may become chronic and continue indefinitely. Certain forms of local spasm, particularly choreiform movements of the muscles of the face, eyes, or neck, may be permanent. In any case of chorea which lasts longer than the usual time, the patient should be carefully examined for some cause of peripheral irritation. The tendency to relapses and second attacks is very marked. Later attacks are likely to occur in the spring succeeding the first illness, and in a small number of patients attacks may come every year for four or five years.

Diagnosis.—There is little difficulty in recognising chorea from the sudden, irregular, spasmodic contraction of the muscles coming on under the circumstances indicated. No other movements of childhood are likely to be confounded with it. The form of chorea following hemiplegia is usually more athetoid than choreic, yet at times it closely simulates ordinary chorea. The difficulty in distinguishing between the two is often increased by the fact that the weakness of simple chorea may, if unilateral, closely simulate hemiplegia. The existence of rigidity, contractions,

---

* Dr. Herter has called my attention to the fact that in many cases of well-marked chorea the urine contains a peculiar reddish colouring matter called hæmato-porphyrin. This is also found in many cases of rheumatism, another evidence of the close relationship existing between these two diseases.

and increased reflexes belongs exclusively to hemiplegic cases, and these will usually suffice to clear up all doubt with reference to the diagnosis.

Prognosis.—As a rule this is favourable, and complete recovery can be predicted, the exceptions to this being few in number. Parents should always be warned of the tendency of the disease to return in succeeding years, and the fact should be stated that in a certain proportion of cases the disease may be permanent. The prognosis of the cardiac murmurs occurring in chorea should always be guarded, although some of these are functional and disappear with recovery from the chorea; but the number of those which do not disappear is sufficiently large to make one always apprehensive as to the ultimate result. Acute chorea accompanied with endocarditis may be fatal; a number of such cases are on record in which there has been no other evidence of rheumatism.

Treatment.—The general management of the case is equally important with the administration of drugs. A child with chorea should at once be taken from school, and should never be subjected to punishment or to ridicule on account of the movements. Special attention should be given to the patient's diet and general nutrition. Tonics, especially iron, are indicated in most cases. The food should be simple and nutritious, and all stimulants, particularly tea and coffee, should be absolutely prohibited. While fresh air is desirable, exercise should be prescribed with great caution and its effect should be carefully watched. It should never be carried beyond the point of slight fatigue. A certain amount of moral restraint is absolutely necessary; thus it often happens that choreic patients do very badly at home where they are indulged and receive sympathy, while in a hospital, where they are under restraint and made to control themselves, they begin to improve immediately. Gymnastics, although useful in some of the milder cases, may do positive harm in those which are severe. They should be regularly and systematically practised twice a day, but not continued too long. In all severe cases the "rest treatment" should be employed, and equal benefit is also seen in the milder ones,—the patient is put to bed, and complete mental and physical rest secured. This may be combined with gentle massage for fifteen or twenty minutes a day. The daily use of warm baths, either alone or in conjunction with massage, is decidedly beneficial. In other cases the regular use of cold sponging is of the greatest value.

With reference to the use of drugs, it is advisable to separate from other cases those in which the connection with rheumatism is very close. In the rheumatic cases, salicylate of soda is often efficient, while the drugs usually employed may be absolutely without effect. In a case recently under observation, arsenic had been continued for two weeks without the slightest improvement, when the patient had an intercurrent attack of subacute rheumatism for which salicylate of soda in full doses was given, with the effect of controlling the choreic symptoms promptly and perma-

nently. In the non-rheumatic cases, arsenic is almost universally admitted to be the most valuable remedy we possess. The method of administration is important; failure most frequently results from the use of too small doses. Beginning with four drops of Fowler's solution three times a day for a child of eight years, the daily quantity may be increased by two drops each day until a disturbance of the stomach or bowels is produced, with puffiness under the eyes. The drug should now be stopped for two or three days, and then the same doses resumed and gradually increased, usually up to twelve drops three times a day, sometimes to fifteen, and even twenty drops, unless the movements cease before that time; but when this occurs the drug should be stopped. Arsenic should always be given after meals, and largely diluted, the dose being taken in a full glass of water, but not necessarily drunk at one time. The possibility of arsenical poisoning should be remembered, although it is extremely rare. Semple has reported a case in which multiple neuritis and general pigmentation of the skin occurred after four weeks' administration of the drug.

In the event of the failure of arsenic alone, it should be combined with the rest treatment. Drugs which sometimes succeed where arsenic fails are antipyrine and strychnine. From twenty to thirty grains of antipyrine should be given daily in divided doses to a child of eight years. There are a certain number of cases in which striking improvement follows the use of this drug if given in the full doses mentioned. To a child of eight years strychnine should be given in doses of $\frac{1}{60}$ of a grain three times a day, the dose being gradually increased until double this quantity is given; sometimes even larger doses than these are well borne. Galvanism is of some value in cases not relieved by drugs. Acute chorea of great severity may require opium, bromide and chloral, or even chloroform.

In estimating the value of drugs in the treatment of chorea, the natural course of the disease should be kept in mind, since those drugs which are taken after the third or fourth week are much more likely to be thought beneficial than those used in the early period of the attack.

There is no doubt that chorea may be dependent upon some ocular defect, and a correction of this will then form an essential part of the treatment, although few, if any, cases are cured by attention to the eyes alone.

Chorea has a strong tendency to recur, especially in the spring of the year. Children who have had one attack should be closely watched, particularly with reference to their work in school. They should not be crowded in their studies, they should have long vacations, and the nervous system should not be put upon any severe tension for a long time.

OTHER SPASMODIC AFFECTIONS.

Habit Spasm.—This term was, I think, first suggested by Gowers, to describe certain muscular movements of a spasmodic character which at

first are only occasionally noticed, but which sometimes persist until they become habitual and almost entirely involuntary. The condition was previously called "habit chorea" by Weir Mitchell. The movements usually affect the muscles of the face, but they may be seen in almost any part of the body. The most frequent varieties consist of blinking or sudden frowning, raising the eyebrows, or some peculiar grimace. At other times there is sudden twisting of the head, shrugging of the shoulders, or jerking of the hands. It is not often seen in the leg, but the muscles of respiration are quite frequently affected. There may be a half-sigh, a sort of sob, or a peculiar dry, laryngeal cough.

These movements are at first only occasional; but as the habit becomes more firmly fixed the spasm recurs every few minutes, and in severe cases it may be almost continuous. In nearly all cases it increases by observation. The same form of spasm does not always continue, but after a time one may subside and another take its place. The condition may last for months or years, and it may even be permanent.

The causes are those of neuroses in general. In the beginning, at least, there is usually a somewhat depreciated general health. The patients are nervous children of neurotic antecedents. There may be a history of some definite exciting cause, such as illness or overwork in school. The spasm of the muscles about the eyes may be associated with pathological conditions of these organs. This may be enough to start the spasm, if not to continue it. Both sexes are affected. In boys, masturbation may sometimes be an exciting cause.

Habit spasm is to be differentiated from chorea: this is usually easy, from the limitation of the movements to one part or group of muscles and from the duration of the disease.

Treatment is quite unsatisfactory after the habit has become fixed, hence it is of the utmost importance that it should be arrested at the earliest possible age. Punishments are of no avail, and usually aggravate the condition. Rewards are much more effectual. The general health should receive attention and nerve tonics should be given, especially strychnine.

Athetosis and Athetoid Movements.—This term, introduced by Hammond, is used to describe a chronic form of spasm usually seen in the hand, but sometimes also in the foot, and even the face. It may affect both sides, but in most cases it is unilateral. The movement is slow, irregular, and inco-ordinate—a sort of "mobile spasm," as it has been called—and there may be associated a certain amount of muscular rigidity. Such movements may occur in persons otherwise healthy, but are usually seen as a sequel of cerebral palsies, generally hemiplegia. Recovery from the hemiplegia may be so nearly complete that the athetoid movements are looked upon as primary. In some cases the movements are more rapid and somewhat resemble those of chorea,—a condition which

is sometimes classed as *post-hemiplegic chorea*. Athetosis is not influenced by treatment.

Rotary and Nodding Spasm of the Head.—These are rare forms of irregular movements usually observed in infancy. The condition was described long ago by Henoch, and since then cases have been reported by Hadden,* Peterson, and others. The most frequent is the rotary spasm, which consists in a side-to-side oscillation of the head, which may be slow or rapid, and in some cases is almost continuous. Some children have at times the nodding spasm also, and in others this is the only movement seen. Nystagmus is frequently associated, and may be of one or both sides. In a few of the reported cases convergent strabismus was present.

The causes of the condition are extremely obscure. It is usually seen in infancy between the third and eighteenth months, and, like most nervous symptoms of this period, has been ascribed to dentition, but without any special reason. In three of the cases reported by Hadden, it followed an injury to the head, and might perhaps be regarded as a result of cerebral concussion.

As a rule, the condition lasts for several months and improves,—in fact, recovery generally occurs. The prognosis is then usually favourable. In most of the reported cases improvement has followed the use of bromides; from ten to twelve grains daily should be given.

Nystagmus.—This term is applied to rhythmical, involuntary, oscillatory movements usually of both eyes. They are caused by the alternate contraction of opposing muscles. Nystagmus may be either vertical or horizontal. It is most often seen in infants a few months old, and is a symptom of irritation which may be general or local. In some cases the movement is almost continuous, occurring even in sleep; in others, it is only noticed at times of special excitement.

The etiology of nystagmus is obscure, and it may occur in quite a variety of conditions,—sometimes referable to the eye, at other times to the central nervous system. On the part of the eye, nystagmus may be due to blindness from any cause, to congenital cataract, corneal opacity, disease of the choroid or retina, or to errors of refraction. It may be seen in almost any organic disease of the nervous system, both with focal and diffuse lesions, especially in chronic hydrocephalus, insular sclerosis, tuberculous meningitis, and in diseases in which sight is impaired. Nystagmus may be of reflex origin, as in a case recently occurring in the Babies' Hospital, where an infant with a severe diarrhœa had repeated attacks, which disappeared each time after intestinal irrigation. While it is of no importance as a localizing symptom, nystagmus usually indicates something more than functional disturbance. An exception to this may perhaps be made when it follows cerebral concussion. In such cases it is

* Lancet, June 14, 1890.

usually temporary, disappearing in a few days or weeks. Under most other conditions it may continue indefinitely.

The condition of the eyes should be investigated in every case of nystagmus; it is only when the cause is here, and can be removed, that habitual nystagmus is amenable to treatment.

Hiccough (Singultus).—This is a spasm of the diaphragm which is usually seen in young infants. In them it is in most cases due to some irritation in the stomach. It is seen after eating, and may depend upon overfilling of the stomach by food, swallowing of air, etc. In other cases it has no relation to the taking of food, and is to be regarded as a form of reflex spasm, which may occur from a variety of causes, such as cold feet, chilling of the surface during bath, or suddenly taking an infant from a warm bed into a cold room. In cases like the above, hiccough, though sometimes annoying, is of little importance. It may be associated with gastric indigestion, with intestinal flatulence or inflammation, with peritonitis or intestinal obstruction. With the last two conditions it is always an unfavourable symptom. In older children hiccough sometimes occurs as a pure neurosis.

The object of treatment is to remove the cause. In infants this is to aid in the expulsion of the gas from the stomach by manipulation, position, or the other means useful in gastric colic. Where it is a nervous symptom only, it may be arrested by holding the breath, prolonged forced expiration, as in blowing a trumpet, and sometimes may require the use of such drugs as control muscular spasm—e. g., antipyrine or chloral.

Thomsen's Disease (Congenital Myotonia).—This rare disease is usually congenital. It may occur in several members of the same family, and is often hereditary. The characteristic symptoms are a peculiar rigidity of the muscles which is observed when they are first brought into action after repose. This rigidity is spasmodic, and usually continues but a few moments. It may recur when voluntary movements are again attempted. If, however, muscular effort is persisted in, it soon passes off. It is increased by apprehension, excitement, or cold, and by observation. The legs are most frequently affected, the condition being often noticed when the patient starts to walk; any of the voluntary muscles, however, may be involved. It may be greater upon one side of the body than upon the other. The muscles are abnormally sensitive to mechanical stimulation, and often to galvanism. They are above normal size, and the fibres themselves are enlarged.

The pathology of this disease is, according to Gowers, an altered functional condition of the muscle fibres, and an abnormal functional state of the nerve cells of the cord and the cortex. It is incurable, although the symptoms may be improved by active muscular exercise.

Cervical Opisthotonus.—This is usually a symptom of disease at the base of the brain, occurring with simple, tuberculous, and chronic basilar

meningitis, sometimes with tumours of the posterior fossa of the skull. However, in certain cases it occurs as a form of reflex spasm, particularly in young infants who are suffering from diarrhœal diseases or marasmus. In these cases it may last for days or weeks. The deformity is produced by a contraction of the superior fibres of the trapezius and by the posterior group of cervical muscles.

Torticollis—Wry-Neck.—Torticollis is usually produced by a tonic spasm of one sterno-mastoid muscle, with which may be associated spasm of the posterior cervical muscles, including the trapezius. In recent cases there is simply a condition of muscular spasm; in those of long standing there may be permanent shortening of the affected muscle, atrophy, and partial paralysis. A somewhat similar deformity may be caused by cicatricial contraction of the tissues of the neck following burns.

Fig. 109.—Spasmodic torticollis from malaria. Trapezius and sterno-mastoid of the left side are affected.

The deformity varies somewhat according as the sterno-mastoid muscle is alone affected, or the posterior muscles also, and as to which predominates. In simple sterno-mastoid spasm the head is inclined to the affected side and rotated toward the opposite side; the chin is raised, and the ear approaches the clavicle. When other muscles are involved the deformity is modified. If the trapezius is affected (Fig. 109) there is less rotation of the head, but it is drawn to the affected side and somewhat backward, while the shoulder is raised and the spine curved. Both of these symptoms may be seen to a slight degree in almost any marked case of sterno-mastoid spasm. Sometimes the spasm of the posterior muscles affects both sides; the head is then drawn backward and held rigidly but without rotation. In most of the recent cases the deformity can be partially or entirely overcome by passive force; but after a time this is impossible, owing to muscular shortening. In recent cases also localized pain and tenderness are frequently present, and sometimes they are severe.

Etiology.—Spasmodic torticollis may be produced by anything causing irritation of the trunk or the branches of the spinal accessory nerve; the source may be in the spinal canal, in the cranium, along the course of the nerve trunk, or of any of its peripheral fibres.

Cases are usually divided into congenital and acquired. Whitman,* from the records of the Hospital for the Ruptured and Crippled, New York, for nineteen years, gives the following statistics of 264 cases,—torticollis from Pott's disease not being included: Males, 109; females, 155; congenital, 32; under two years, 33; from two to ten years, 153; over ten years, 46; acute (i. e., of less than two months' duration), 77; chronic, 60, of which number 22 had lasted two years or longer.

Regarding the cause of congenital torticollis there is some dispute. Such cases have often been attributed to the contraction resulting from hæmatoma of the sterno-mastoid (page 94). My own experience coincides with Whitman's, that this is rarely if ever the case. While it is possible that the deformity is sometimes the consequence of injury received during delivery, the cause of most of the congenital cases goes back to conditions existing before birth. It may be compared to club-foot, and may be due to a faulty position of the child *in utero*, or it may come from more serious conditions, such as malformations, or unequal development of the two sides of the body.

One of the most frequent causes in the acquired cases, is irritation of the spinal accessory nerve by an enlarged cervical lymph gland; this was the cause assigned in nearly half of Whitman's cases; such is the usual etiology of torticollis following scarlet fever, measles, or diphtheria. I have seen it in the early stage of quinsy, and it may occur in cellulitis of the neck. A cause which the physician should always have in mind is cervical Pott's disease; torticollis may be the earliest, and for several weeks sometimes almost the only, objective symptom of this disease. Torticollis coming on acutely is most frequently due to cold (rheumatism?) or malaria. I have notes of eight cases clearly traceable to malaria, and have seen at least a dozen others. In several of these there was a distinct periodicity in the spasm, it recurring regularly at about the same time each day until quinine was given; in some cases it was accompanied by fever, in others not. In the so-called rheumatic torticollis, muscular pain and soreness are rather more prominent than in the other forms. In fourteen of Whitman's cases the spasm was attributed to injuries other than burns; and in only nine was it associated with some other disease of the nervous system, most frequently with chorea.

Prognosis.—The result in a case of torticollis depends upon the cause, the severity, and the duration of the deformity. Most of the acute cases from malaria, rheumatism, etc., recover, under appropriate treatment, in the course of a few weeks, sometimes in a few days, and not a few recover spontaneously. The congenital cases with slight deformity are usually amenable to mechanical or postural treatment if begun early. There is, however, in most of the other varieties a disposition of the de-

* Observations upon Torticollis, Medical News, October 24, 1891.

formity, if untreated, to persist, and even to increase. If it has lasted several months the probabilities of spontaneous recovery or even of improvement are small.

Treatment.—The first indication is to remove or treat the cause where one can be found. Malarial cases require quinine; rheumatic cases are benefited by rest in bed, hot applications, counter-irritation, friction, and sometimes by anti-rheumatic remedies. Cases which have lasted a month usually require some orthopædic head-support, and those which have lasted six months or more are rarely cured without a surgical operation. This may be either a subcutaneous tenotomy or myotomy of the sterno-mastoid, or an open incision. Whitman gives the result of thirty-two cases admitted for treatment to the hospital mentioned, as follows: In 17 in which the deformity had lasted less than six months, 10 were cured, the average duration of treatment being three months; 4 were improved, and 3 not improved, the average duration of treatment in these cases being eleven months. Of 15 cases in which the deformity had lasted over six months, none were cured and only 6 improved, after an average of about eight months' treatment. In the foregoing series of cases the treatment consisted mainly in the use of orthopædic apparatus; later results from incision have been considerably more favourable. But these figures show how serious a matter is an old case of torticollis, and emphasize the importance of resorting to efficient measures early in the disease.

HYSTERIA.

This is not a disease of childhood, but one which is occasionally seen in early life. All that will be attempted in this chapter is to point out the most common manifestations of hysteria when it occurs in young children. After puberty it is essentially the same as in adults.*

Etiology.—Hysteria is very rare before the seventh or eighth year, and most of the cases seen in children occur after the tenth year. As to sex, there is no such predominance of females as in later life, although even in childhood they are more frequently affected than males. Hereditary influences play an important part in the production of this disease. It is seen in children who inherit a nervous constitution, or in whose parents nervous diseases, such as insanity, or hysteria, or alcoholism have been present. Of the other etiological factors the most important are a disordered nutrition, frequently with anæmia or chlorosis, and overpressure in schools. Masturbation or phimosis may act as an exciting cause, or, indeed, anything which leads to an exalted nervous irritability and depreciation of the general health. It is occasionally associated with tuber-

* For a fuller discussion of this subject, and references to recent literature, see Mills, in Keating's Cyclopædia, vol. iv.

culosis; it may follow any of the acute infectious diseases; or it may be excited by injury, fright, or imitation.

Symptoms.—There is scarcely any disease in which the clinical picture presented is so varied as in hysteria. It may simulate almost any form of organic disease of the brain, lungs, digestive organs, bones, or joints. The most common symptoms may be grouped under four general heads. These are, however, seen in almost every conceivable combination.

1. *Psychical symptoms.*—Where these predominate there may be seen periods of mental depression of longer or shorter duration, a change in disposition, an indifference to surroundings, a capricious humour, or a nervous condition of extreme irritability with irregular paroxysms of laughter or weeping without cause. There may be great excitability of temper, and fits of passion almost maniacal in their severity. There may be various hallucinations. Sleep is frequently disturbed, sometimes by attacks resembling ordinary night-terrors; sometimes somnambulism is present. There is often a disposition to deception about the most trivial matters, which may last for weeks. There is a tendency to imitate the symptoms of various diseases, which the patients may have witnessed in others or about which they have read.

2. *Sensory symptoms.*—These are the most frequent manifestations of hysteria in early life. There is often general or local hyperæsthesia, which may be so great as to simulate inflammation of the various internal organs. Anæsthesia is much less common, although it may be seen in children as young as eight or nine. Headache is an occasional symptom, and is sometimes associated with great tenderness of the scalp. There may be neuralgias in the different parts of the body, or sharp epigastric pain, sometimes accompanied by vomiting. Sometimes the special senses are affected, giving rise to hysterical blindness or deafness, usually of short duration.

3. *Joint symptoms.*—These are really a variety of sensory disturbances. They are not uncommon, and are often most puzzling. The symptoms may be referable to the spine, or to any of the large joints, particularly those of the lower extremity. All forms of organic disease of these joints may be simulated, and these patients are often treated for months with orthopædic apparatus, with the belief that they are suffering from Pott's disease, lateral curvature of the spine, club-foot, or ostitis of the hip, knee, or ankle. Cases of this sort have been very fully described by Gibney,* and by Shaffer, whose articles should be consulted for fuller details. They are usually seen between the ages of ten and fourteen years, and occur in both sexes. There may be lameness referred to one of the large joints, curvature of the spine, or torticollis. The symptoms are most frequently

* Gibney, Transactions of the American Neurological Association. 1877. Shaffer, Archives of Medicine, New York, December, 1879, February and April, 1880.

referred to the hip, and next to the knee, the ankle, or the spine. The pain is often acute. It is increased by motion, and by attempts at overcoming the deformity, if any is present. There is a marked hyperæsthesia of the whole limb, and sometimes of the body. In nearly every case there is marked tenderness of the spine upon pressure, especially in the dorsal region. The deformity may be very slight from spasm of the flexors only, or it may be severe, and followed by contracture, so that the thighs may be flexed tightly against the abdomen with the heels against the buttocks. Such deformities may last for months. There may be considerable muscular atrophy, but only that which comes from disuse. A special difficulty in diagnosis arises from the circumstance that these symptoms occasionally follow an injury.

Organic disease of bones and joints may usually be excluded by attention to the following points: The mode of onset is more abrupt than is seen in bone disease, and the course of the disease is quite irregular. The degree of deformity is greater than is seen in bone disease of the same duration. There are general hyperæsthesia of the limb, acute tenderness of the spine upon pressure, and undue sensitiveness to heat or cold. The deformity varies from time to time, being always more marked when examination is attempted. If the patients are closely watched, other evidences of hysteria may be seen. Under complete anæsthesia the contractures may disappear entirely. There is no enlargement of the articular ends of the bones, no swelling of the soft parts, and no evidence of active inflammation or of suppuration. All the symptoms except the deformity are subjective. Under proper treatment there is in most cases perfect recovery, often in a surprisingly short time.

4. *Motor and convulsive symptoms.*—In the milder forms we may see many varieties of tonic or clonic spasm. There may be seen local spasm of the eyes, face, or mouth, spasm of the muscles of the neck producing torticollis, of the muscles of respiration causing dyspnœa, which may be constant or paroxysmal. There may be hiccough, or spasm of the larynx causing hysterical aphonia. A very common symptom is hysterical cough, which may be so frequent and so severe—even accompanied by hæmoptysis—that grave disease of the lungs is suspected; the chest, however, is free from the physical signs of disease. There may be frequent attacks of vomiting with eructations; these may be continued sometimes even for months, and in rare instances blood has been vomited. There may be dysphagia from spasm of the œsophagus, or regurgitation of food on attempts at swallowing. In more severe cases we may have the symptoms of chorea major and attacks of hystero-epilepsy. The latter are rare in children and do not differ essentially from such attacks in older patients. There are usually prodromal symptoms. The convulsive movements are exceedingly varied in type. There are painful sensations and sensitive areas, by pressure upon which hysterical symptoms may be in-

creased or even convulsions excited. The respiration may be rapid or irregular. All variations in tonic and clonic spasm may be seen. Opisthotonus is frequent. Consciousness is not fully lost, but is disturbed, and hallucinations are present. The temperature is normal.

Hysterical paralysis is not common in children, but it may be seen even in the very young. Gillette has reported the case of a child eighteen months old who exhibited the symptoms of hysterical palsy of one arm. Other symptoms occasionally seen in hysteria, are persistent anorexia, polyuria, sometimes incontinence of urine, disturbance of the secretion of saliva or perspiration, and very rarely hysterical fever.

The general condition of hysterical patients is usually below the normal. They are poorly nourished and anæmic; they sleep badly; they have capricious appetites, feeble digestion, and faulty assimilation.

Diagnosis.—Hysteria is apt to be overlooked because its occurrence in children is not considered as often as it should be. In most cases the diagnosis is easy if hysteria is suspected. A combination of vague disconnected symptoms is usually present which admits of no other explanation. Organic disease can be excluded only by careful and repeated examinations. It is to be borne in mind, however, that hysteria not infrequently complicates organic or constitutional disease. Much importance is to be attached to a family history of hysteria or of other neuroses. From poliomyelitis, hysterical paralysis is differentiated by the presence of faradic contractility even though atrophy exists. Hysterical convulsions are differentiated from true epilepsy by the absence of any elevation of temperature, of biting of the tongue, evacuation of the viscera, of a violent fall, and often by the rapid disappearance of the symptoms under appropriate treatment.

Prognosis.—This is better than in adults, especially if the cases are taken in hand early, before the disease has become deeply seated. Very much depends upon how well the directions for treatment can be carried out. The prognosis is less favourable where the hereditary tendency is strongly marked. In many cases there are relapses later in life.

Treatment.—Prophylaxis is of much importance. When a hereditary tendency to nervous diseases exists in a family, or whenever very nervous children are placed under the physician's care, every means should be taken toward muscular development, keeping the nervous system in the background. Such children should lead an out-of-door life as much as possible, preferably in the country; they should keep early hours, have regular exercise, and their education should be directed with moderation and judgment; special attention being paid to regularity of work, and the prevention of overpressure in schools. Theatres and exciting books should be avoided. All stimulants, including tea and coffee, should be absolutely forbidden. The diet should be plain and nutritious. It is highly important that such children should be removed from association with a hysterical mother, when this is possible.

In the general management of a case of hysteria, it is of the first importance that the child should be cared for by a person of firmness, who can exercise proper control. Hysterical children are always managed more easily when they are removed from their homes and placed under the charge of a good trained-nurse. Isolation is absolutely essential in many cases. The general health should be carefully looked after, and arsenic, iron, cod-liver oil, and other tonics given according to indications. Horseback exercise and other out-of-door sports should be encouraged, and every means taken to interest the child in something which requires physical exercise. In cases of simulated disease, the child should be put to bed, no books or toys allowed, and no effort made toward his amusement. No sympathy should be exhibited, but the child treated with kindness and firmness. This moral treatment is quite as important as any other part of the therapeutics. In cases with hysterical joint symptoms the most valuable thing is counter-irritation to the spine, preferably by the Paquelin cautery. Some cases are benefitted by galvanism. The moral effect of hypodermics, even of cold water, is sometimes striking. Under no circumstances should mechanical force be used to overcome deformity. Many cases of hysteria improve under hydrotherapy; the cold douche, the cold pack, or the shower bath may be used. This is valuable in conjunction with massage and the "rest treatment."

In attacks of hystero-epilepsy the cold douche may be used, or pressure made upon the testicle or ovary. In severe cases ether may be given. In all hysterical cases the condition of the bowels should receive careful attention, as these patients are very prone to obstinate constipation.

HEADACHES.

Headaches are not common in little children except in connection with disease of the brain or meninges; in older children they occur from causes similar to those seen in adult life. The most frequent headaches may be grouped in the following classes:

1. *Toxic headaches.*—Such are the headaches resulting from uræmia, from carbonic acid in poorly ventilated rooms, and from malaria. But the largest number are due to absorption of toxines from the intestines, and are associated with chronic indigestion and constipation.

2. *Headaches from anæmia and malnutrition.*—These are most frequently seen in girls from ten to fourteen years old. Some are intellectually bright, and have been crowded in their school work; others are dull and learn only with difficulty, and in consequence worry over their work until their health becomes undermined. They sleep badly, lose appetite, and often become choreic. The anæmia may be either the cause or the result of these symptoms. The urine in these cases often contains a large excess of uric acid.

3. *Headaches of nervous origin.*—These may occur in children who are highly neurotic, either from their inheritance or surroundings, and in those who are the subjects of epilepsy or hysteria, and they may be symptomatic of organic disease of the brain, such as tumour or tuberculous or syphilitic meningitis. True facial neuralgia is rare in childhood except from carious teeth; from this cause, however, it is not infrequent.

4. *Headaches due to disease of some of the organs of special sense.*—In connection with the eyes there may be conjunctivitis, keratitis, iritis, errors of refraction, or strabismus; connected with the nose there may be polypi, hypertrophic rhinitis, or adenoid vegetations of the pharynx; connected with the ears there may be otitis or foreign bodies in the canal. Each one of these conditions requires special treatment.

5. *Headaches due to inherited gout or rheumatism.*—These are not very frequent, but they may be severe, and may at times simulate the onset of meningitis. They are often accompanied by pains in the joints, muscles, or nerve trunks; they may be associated with a urine which is highly acid and contains deposits of oxalates or of free uric acid.

6. *Disturbances of the genital tract* are rarely a cause of headaches in children, although this may be the case in girls about the time of puberty, especially where menstruation is delayed or difficult.

Diagnosis.—The diagnosis of headaches includes the discovery of the cause, and this is often difficult. In an infant or a young child, organic disease of the nervous system should always be suspected as a cause of severe headaches. In older children the important things to be considered, because the most frequent, are digestive disturbances, nervous exhaustion, malnutrition, and visual disorders. An absolute diagnosis in a case of persistent headache can be made only by a careful physical examination, not omitting a study of the urine; often there must be a close observation of the patient for some time.

Treatment.—The only successful treatment is that which is directed toward a removal of the cause. Each one of the different groups above mentioned is to be managed differently, according to the principles elsewhere laid down regarding the treatment of these conditions. For the relief of the symptom, cold to the head, a hot foot-bath, and phenacetine in moderate doses are perhaps the most certain of all remedies.

DISORDERS OF SPEECH.

In this chapter will be discussed only functional speech defects,* those depending upon organic conditions being considered in connection with diseases of the brain. The most common varieties are stuttering, stammering, lisping, alalia, backwardness, and functional aphasia. All

* See Wyllie, Edinburgh Medical Journal, October, 1891.

forms are much more frequent in boys than in girls, the proportion being more than four to one.

Stuttering.—This is the most common form of speech disturbance. Articulation is distinct and the separate sounds are properly produced, but there is a difficulty in connecting the consonant with the succeeding vowel; this seems like an obstacle to be overcome. Stuttering is occasionally seen in most children. It is more frequent in the third and fourth years, before speech is thoroughly mastered. At this age it is aggravated or produced by disturbances of nutrition, but is usually of temporary duration, lasting for a few weeks or months. Only recently a little boy of four was under my care, who became very anæmic, slept poorly, and suffered from malnutrition as a result of the confinement incident to a home in the city. He soon began to stutter, and in a short time it became painfully marked. After a few weeks in the country he improved very much in his general condition, gained four or five pounds in weight, and his stuttering completely, and I think permanently, disappeared. Such disturbances as this are analogous to chorea. In other cases stuttering follows some acute illness, and under such conditions also it is usually of short duration.

Most children who become habitual stutterers do not begin until they are six or seven years old, and sometimes even later. Stuttering may arise from imitation, and probably inheritance is an occasional factor. It is frequently a mark of degeneration.

It is important that all such cases receive early treatment before the habit becomes firmly fixed. The prognosis is good for spontaneous recovery in nearly all the cases seen in very young children, and also in those coming on after acute illness. Other cases in which the condition has become habitual, should have the benefit of systematic training under a competent teacher in breathing, vocal, and speech gymnastics.

Stammering.—This term is sometimes used synonymously with stuttering. Kussmaul makes the distinction between them that, in stammering, individual sounds are difficult of production, while in stuttering it is syllabic combinations. Stammering is often accompanied by some defect in the organs of articulation—the teeth, lips, tongue, or palate—which is not present in stuttering.

The treatment consists in careful training and in the correction of whatever abnormal local conditions may exist.

Lisping.—In this there is imperfect production of certain sounds, owing usually to a faulty position of the organs of articulation. The sounds may be so indistinct that they can not be understood. In this condition also there may be defective formation of some of the organs of articulation, although in the milder forms this is not the case. The treatment is similar to that of stammering.

Alalia.—This consists in a total inability to articulate. It is seen in all young infants during their earliest attempts at talking. In older children it is usually associated with some mental defect.

Backwardness.—Backwardness is carefully to be distinguished from a late development of speech due to idiocy. At two years old children not deaf are almost invariably able to speak. Speech may be late in consequence of prolonged or very severe illness, and where it has been acquired it may be lost from similar causes.

Functional Aphasia.—The term has been applied to a temporary loss of speech which sometimes occurs in chorea, and sometimes from severe fright or anything else which has produced a marked nervous impression. West records an instance in a girl of eight years, who was suffering from an attack of chorea induced by fright. Speech first became difficult and then was lost altogether. For a month the child could say only "Yes" and "No." The case very slowly improved, but at the end of nine weeks had recovered completely.

Loss of speech sometimes follows the acute infectious diseases, especially typhoid fever.

In all disorders of speech, the functional cases are to be distinguished from those which depend upon deafness and mental deficiency. The frequency with which these disorders are due to disturbances of general nutrition, and to local causes in the mouth and throat, should be borne in mind, and these conditions should receive their appropriate treatment early, before the habit of defective speech becomes firmly established. For the latter class of unfortunates, special training at the hands of a competent teacher should be advised, preferably in an institution.

DISORDERS OF SLEEP.*

Disturbed Sleep, Sleeplessness.—Disturbed or restless sleep is much more common in infancy and childhood than is true insomnia, although the causes of the two conditions may be the same.

Etiology.—In infancy these symptoms are most frequently due to hunger or to indigestion resulting from overfeeding or improper feeding. Very often disturbed sleep is the result of bad habits, such as rocking during sleep or night-feeding. Sometimes it arises from dentition, or the pain of colic or otitis; at other times it may be simply the expression of a condition of nervous irritability, the result of inheritance or of the child's surroundings.

In later childhood the first thing to be suspected when sleep is much disturbed is some derangement of the digestive organs; in this will be found the explanation of fully half the cases. The most frequent type,

* For the characteristics of the sleep of infancy, and the average amount taken at the different ages, see pages 5 and 6.

where the symptom is of long duration, is chronic intestinal indigestion, often associated with indicanuria, a condition in which the diagnosis of the mother is usually worms. Other cases are due to obstructed respiration from adenoid growths of the pharynx or enlarged tonsils, sometimes to nocturnal attacks of asthma. A lack of fresh air in the sleeping room, excessive or insufficient bedclothing, and cold feet, are other frequent causes. Disturbed sleep with "starting pains" is one of the earliest symptoms of hip-joint disease. In the nervous exhaustion resulting from overpressure in schools, and in malnutrition and anæmia, disturbances of sleep are well-nigh constant. They are also seen in organic cardiac disease and in all pulmonary conditions accompanied by dyspnœa or cough. Sleep may be disturbed in consequence of bad dreams which have their origin in exciting stories heard or read just before bedtime, or in too violent or exciting play. To discover the cause in almost any case it is necessary to investigate carefully the whole routine of the child's life.

Symptoms.—The condition may be one of real insomnia which may last for weeks or months; or the sleep may be simply disturbed and restless, the child waking many times during the night, and when asleep will not lie quietly, but constantly changes his position. Sometimes children wake suddenly with a scream, but immediately drop off to sleep again.

Treatment.—The essential treatment consists in the discovery and removal of the cause of the disturbance. This will often involve a radical change in the manner of feeding, in the hygiene of the nursery, and in all the surroundings of the child; but in this way only should these cases be managed. Under no circumstances should the physician countenance the use of drugs to promote sleep in children, except in the case of severe acute disease. Soothing syrups and all nostrums for "teething" should be absolutely forbidden. Mothers and nurses are only too ready to fall into the habit of using them, because the injurious effects are not appreciated. When the cause of sleeplessness is found and removed the child will sleep, but compulsory sleep obtained under other conditions is always productive of more harm than good. If food, diet, and all bad habits have been corrected, nervous causes must be investigated. When no cause can be discovered the treatment should consist in putting the child upon the simplest possible diet, and in attention to such general conditions as anæmia, malnutrition, and neurasthenia, some of which are almost certain to be present. In many cases a warm bath at bed-time will be found beneficial. A quiet, darkened room, plenty of fresh air, and the stopping of both eating and drinking during the night, are essential to a cure in most cases. When the condition accompanies some acute disease, the drugs which are most useful are codeia and trional. A child of two years may take $\frac{1}{20}$ of a grain of codeia or two grains of trional as an initial dose, to be increased if necessary.

Night Terrors—Pavor Nocturnus.—Two classes of cases have been grouped under this head, both having this in common, that sleep is disturbed by fright. In an excellent recent article upon this subject,* Coutts calls attention to the necessity of sharply distinguishing between them.

The condition in the first group partakes of the nature of nightmare. It may be due to partial asphyxia from adenoid growths of the pharynx, or to other causes mentioned under disturbed sleep, or it may be gastric or intestinal in its origin. These cases are quite frequent. Sleep may be disturbed from the outset, and the attack may be merely the culmination of such disturbance. The child wakes in a state of fright and excitement, and often says he has had a bad dream. His mind is clear, he recognises those about him, but it may be a long time before he is sufficiently calm to sleep again. The attack may be remembered perfectly the next day. Cases like this are to be managed in the same general way as cases of disturbed sleep above mentioned.

In the second group are the only cases to which the term "night terrors" should really be applied. These are relatively rare, but the condition is a much more serious one. The symptom is due to some disturbance of the central nervous system. According to Coutts, it occurs especially in those of neurotic antecedents, or those who have previously suffered from infantile convulsions, and it is often the precursor of other nervous attacks,—migraine, hysteria, epilepsy, and even insanity. The attack usually comes suddenly where a child has previously been sleeping quietly, and more frequently in the early part of the night than later. He is generally found sitting upright in his bed in a bewilderment of terror, being afraid of "the dog," or "the bear," or there is some other vision or hallucination which has produced the fright. Often this is associated with something of a red colour. The child does not recognise those about him, does not know where he is, and may go to sleep again without coming to full consciousness. The next day there is no recollection of what has happened. Usually no after-effects are seen, but sometimes a large amount of pale urine is passed. The attacks may be repeated at intervals of a few months, or they may occur every few nights; but whatever the peculiar nature of the vision, it is likely to be repeated in nearly the same form. Such attacks have something in common with epileptic seizures, and the diagnosis between them may at times be difficult. They are always to be regarded seriously, not only on account of what they are in themselves, but on account of what may follow.

Treatment.—All mental and nervous strain should be most carefully avoided, and where the attacks are frequent the bromides should be given at bedtime. Some person should sleep in the same room with the child, or in an adjoining one with the door open.

---

* American Journal of the Medical Sciences, February, 1896.

Excessive Sleep.—It is rare that either infants or children sleep an unnatural amount of the time unless one of two causes is present—organic brain disease or the use of drugs. The latter is always to be suspected if with the sleep there is associated obstinate constipation. Opium in the form of "soothing syrup" or paregoric, is the drug which has usually been given.

INJURIOUS HABITS OF INFANCY AND CHILDHOOD.

On account of the close connection of these habits with disturbances of the nervous system, they may be properly considered with the functional nervous diseases. Although some of these habits may not be of serious importance, yet as a group they have received altogether too little attention at the hands of the physician.

Sucking.—This is a very common habit in infants, and during the first few months it is seen to some degree in most of them. If they are carefully watched the habit is easily stopped; otherwise it may continue indefinitely. Young infants usually suck the fingers when hungry, and this can scarcely be considered abnormal, but an effort should always be made to stop it, lest the habit become fixed. Lindner* distinguishes between simple sucking and sucking with combinations. In the former, the child sucks some part of the body, such as the thumb, fingers, toes, tongue, lips, back of the hand or arm, or it may be some foreign substance, such as part of the clothing, the blanket, a rubber nipple, or a "sugar-teat." This is the most common form that is seen. In the second variety the sucking is accompanied by the rubbing of some other parts, which seems to afford a pleasurable excitement; this may be the ear, the genitals, or any other portion of the body. Sometimes sucking is accompanied by some practice which produces actual pain, such as pulling of the hair or scratching the body. Habits of sucking often persist throughout infancy, and not infrequently throughout childhood; they have often been known to continue up to puberty. The longer the habit has lasted the more difficult is it to break.

The results of sucking may be serious. Deformities of the thumb or finger, of the lips and teeth, and even of the jaws, are sometimes produced. I know a lady, now in advanced life, whose thumbs to this day show a deformity resulting from the habit of thumb-sucking while a child. In her case the habit was not broken until she was eight or nine years old. Probably the most pernicious result of sucking is its tendency to develop the habit of masturbation. Habitual sucking of one hand or finger may lead to spinal curvature.

Treatment.—In the management of these cases the most important thing is to arrest the habit early, before it becomes fixed. Too often the

* Jahrbuch für Kinderheilkunde, vol. xiv, p. 68.

habit of thumb-sucking, or of sucking a rubber nipple, is encouraged by mothers and nurses, because of the temporary quiet which is thereby produced; even physicians are sometimes accessory to this procedure. Under no circumstances should it be resorted to as a means of putting children to sleep or otherwise quieting the nervous system. Nurses and parents should be put on their guard. With infants, the only treatment which is at all successful is such mechanical restraint as will make sucking an impossibility. It is of no use to cover the part which is sucked with bitter solutions. My experience has been that children are not deterred even in the slightest degree by such procedures. The hands of young infants may be covered with mittens, or with the long sleeves of a night-gown which is pinned to the bed, so that it is impossible for the child to get the part to the mouth; or pasteboard splints may be applied at the bend of the elbow, so as to prevent flexion of the arms. Children must be carefully watched at all times, but particularly when going to sleep and when they first wake, since these are the times when sucking is most likely to be indulged in. In the milder cases the habit is often discontinued spontaneously when infants are eighteen months or two years old; but when it has been indulged until a child is four or five years old, it is broken only with the greatest difficulty and after prolonged effort. Punishments are of little avail, but rewards are often successful. The child's pride must be stimulated. Restraint should be encouraged by every means possible. On no account should this be passed over as a trivial matter either by the parents or the physician.

Masturbation.—This is not uncommon even in infancy. Many cases have been observed during the first year, and some as early as the seventh or eighth month. In the Babies' Hospital within the last three years at least half a dozen cases have been under observation in children under two years old, some of them most intractable ones. Masturbation is more frequent after the eighth or ninth year, but it is from the twelfth to the fifteenth that it is especially seen. At this age it is much more often seen in males than in females, although in girls it is particularly hard to control.

The symptoms which these older children exhibit who practise frequent masturbation, are usually marked and quite characteristic. They are pale and anæmic; they have dark rings under the eyes; they sleep poorly, are easily fatigued, and frequently complain of headaches. They become quiet, reticent, and easily embarrassed; they avoid the society of other children, and lose all animation and all interest in out-of-door amusements. They are absent-minded, and show an inability to concentrate the attention upon anything. Gradually they may become more and more morbid, and in extreme cases may develop melancholia, mental weakness, or even insanity. In other cases, attacks of convulsions and epilepsy may follow. I had recently under observation a boy of seven years who

was having from six to ten epileptic seizures a week, in whose case masturbation appeared to be the principal cause. I do not, however, think such cases are frequent. Sometimes hysteria and chorea are traceable to the influence of masturbation, this result being, of course, more likely to follow where there already exists a predisposition to these diseases. In addition to these effects upon the nervous system, where it is begun at an early age, masturbation may seriously interfere with the physical development of the child. The local symptoms of masturbation in the male, are redness and sometimes slight swelling of the prepuce; but very often there is simply a relaxed condition of all the genital organs. In the female there may be redness and swelling of the vulva, and in some cases a moderate vaginitis.

Among the local causes may be mentioned anything which excites undue irritation,—a long or adherent prepuce, phimosis, balanitis, vaginitis, any skin disease which causes itching of the part, thread-worms, and even constipation. Urine which is rendered irritating on account of excessive acidity or the presence of crystals of uric acid, is a not infrequent cause. Exercises in which the legs are rubbed together may lead to it, also posture or clothing which causes friction of the parts, and sometimes warm feather-beds. To these must be added as a potent cause, the habit of sucking. Masturbation often results from example or because the habit has been taught by other children, sometimes by nurses. Where it develops in a young child without local cause, it should not be forgotten that masturbation is one of the signs of degeneration, often an early one, and other stigmata (page 758) will usually be found if they are looked for.

In infants and very young children masturbation is often not recognised. At this age it is more frequently accomplished by thigh-friction, or by rubbing the genitals against a chair or some other object, than by the use of the hands. The variety of ways is almost endless. During the act there are usually noticed flushing of the face and some rigidity of the muscles of the trunk and lower extremities, which are followed by complete relaxation and often by perspiration.

The prognosis depends most of all upon how firmly rooted the habit has become before it is recognised. It is usually a simpler matter to stop it in infants and in young children, as they can be more easily controlled and more closely watched than those who are older. The outlook is much better where the cause is a local one capable of being removed, than where no such cause exists. It is also much better when in an older child it has been acquired by imitation, than where it is a symptom of degeneracy; in fact, the last-mentioned cases are rarely if ever cured.

Treatment.—The most important thing is an early recognition of the condition. The physician should put parents and nurses on their guard, and the first suspicions should be reported and the child carefully watched until all doubt is removed. In most cases seen by the physician the

habit is not difficult to arrest at the outset, but it becomes extremely so after it has been practised for years before it is discovered. In young infants much may be accomplished by mechanical restraint. The kind of restraint which is necessary will depend upon the manner of masturbating. If by the hands, these must be tied during sleep, so that the child can not reach the genitals; if by thigh-friction, the thighs must be separated by tying one to either side of the crib. In inveterate cases, a double side-splint, such as is used in fracture of the femur, may be applied. In children that are over three years old, all such contrivances are almost invariably unsuccessful. It is of the utmost importance in every case to have the child under the close surveillance of a competent and trustworthy person. He should be especially watched just after being put to bed and immediately after waking. Corporal punishment is often useful in very young children, but of little or no benefit in those who are over four years old. In fact, in such it may do positive harm, for deception and lying are soon added to the previous vice. The mother should secure the child's confidence, and in every way possible seek to strengthen his will and stimulate his self-control, using her influence to help him break the habit. The local causes, too, must be examined into and removed whenever found. Circumcision should be done if phimosis exists, and even where it is not, the moral effect of the operation is sometimes of very great benefit. Care should be taken that the clothing does not irritate the parts. The child should be removed from all vicious companions. In some cases hypnotism has been employed with excellent results. The general treatment should be directed to the child's condition. Cold bathing should be practised, iron and tonics administered where they are indicated by the general condition, and the child should be put under as healthful local surroundings as possible. The administration of drugs for the habit itself is of little or no value.

Nail-biting and Tongue-sucking are two forms of habit which are less frequent and less important than those already mentioned. The former is best remedied by keeping the nails cut very short; the latter seldom becomes a fixed habit, and the child usually ceases it of his own accord as he grows older.