CHAPTER III.

DISEASES OF THE BRAIN AND MENINGES.

MALFORMATIONS.

The malformations of the brain are of great variety, and many of them are solely of anatomical interest, as the conditions are incompatible with life. Only the most frequent and the best-known types will be mentioned, and those which are of interest from a clinical point of view.*

Meningocele, Encephalocele, and Hydrencephalocele.—These three conditions have in common a protrusion of some part of the cranial contents through an opening in the skull. In meningocele (Fig. 110) there is protrusion of the membranes alone. These form a sac, which is usually, but not invariably, distended by fluid. In encephalocele (Fig. 111) there is a protrusion of a portion of the brain substance; this is connected with the rest of the brain by a constricted neck or pedicle. There may or may not be fluid present in the tumour. In hydrencephalocele (Fig. 112) there is a protrusion of a portion of the brain substance which contains within it a cavity filled with fluid, this cavity communicating with the distended lateral ventricles.

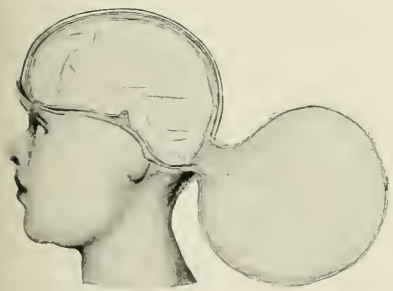

Fig. 110.—Meningocele.

Fig. 111.—Encephalocele.

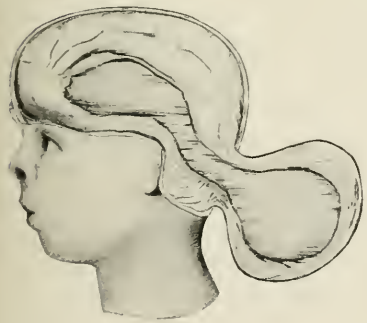

Fig. 112.—Hydrencephalocele.

* For other forms see Sachs, Nervous Diseases of Children, 1895, pp. 589–607.

In all these conditions there is a tumour, usually pedunculated, of a round or pyriform shape, with a smooth or lobulated surface. The ordinary size is that of a mandarin orange; it may be as small as a walnut, or as large as the patient's head. It is generally covered by the scalp, which is often denuded of hair; but it may be covered only by granulation-tissue, or it may show a central cicatrix, like that of spina bifida. Its coverings are usually thin and translucent. Other deformities, such as spina bifida, club-foot, and hare-lip, are frequently present.

All these conditions are rare, but the most frequent and most serious one is hydrencephalocele, this being usually associated with hydrocephalus. The next in frequency is encephalocele, which has the best prognosis. This is frequently termed *hernia cerebri*. It may exist without very serious alteration in the cranial contents. If fluid is present, it is external to the brain. Meningocele is the rarest form, and consists simply of an accumulation of fluid in the arachnoid cavity, which communicates by a small opening with the general arachnoid cavity of the brain.

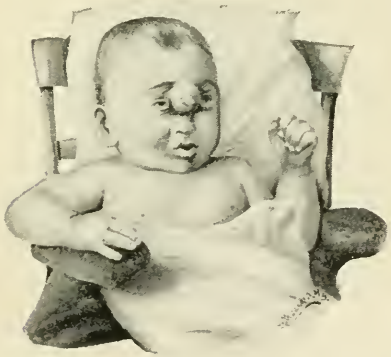

Fig. 113.—Naso-frontal meningocele (after Demme).

Of one hundred and five cases collected by Schatz, fifty-nine occupied the occipital region and forty-six were frontal. The aperture through which the occipital protrusion takes place is usually in the median line. It may communicate with the posterior fontanel, with the foramen magnum, or with the cleft of a spina bifida. The occipital bone may be divided in the median line, or rarely it may be absent.

In the naso-frontal form (Fig. 113) the tumour is usually at the root of the nose, a little to one side of the median line. The aperture is most frequently between the cribriform plate of the ethmoid and the frontal bones. It may be between the lateral halves of the frontal bone, causing a median tumour. The point of protrusion may also be the lateral region of the skull, generally about the lateral fontanel, or along the line of the sutures; it may project into the mouth or the pharynx. These anterior tumours are usually small, although large ones containing the anterior lobes of the brain, have been seen.

The theory of the origin of these malformations which is most widely accepted is that they are primarily cases of intra-uterine hydrocephalus, and as the cranial cavity has gradually been closed by the development of the bones, a certain portion of the brain has been left outside.

Symptoms.—The tumour is always congenital, although after birth it frequently increases very much in size. A typical tumour is round

and elastic, usually giving evidences of fluid; it pulsates synchronously with the heart; during screaming or forced inspiration, it increases in size; partial and in some cases complete reduction is possible, but this is usually followed by marked cerebral symptoms, even by convulsions. After partial reduction, an opening in the skull may often be made out. Microcephalus may be present, or there may be unequal development of the two sides of the head.

The following differential points given by Treves, indicate the most characteristic features of the three varieties: In meningocele, the tumour is at first small, but increases; it has a smooth surface; it is pedunculated; there is distinct fluctuation, perfect translucency, rarely pulsation; often it is completely reducible; compression of the tumour causes cerebral symptoms; the skull is normal. In encephalocele, the tumour is small and smooth; it is rarely pedunculated; fluctuation is absent; it is not translucent; there is distinct pulsation; it is usually reducible; pressure causes cerebral symptoms; the skull is normal. In hydrencephalocele, there is a large pendulous tumour with an irregular or lobulated surface; it is pedunculated; translucency is rarely complete; fluctuation is distinct; it is irreducible; pressure rarely causes symptoms; microcephalus and other deformities are often associated.

The occipital tumours are usually more serious than the frontal ones. The majority of cases die in the course of the first few weeks of life, death resulting from meningitis, convulsions, or rupture. In meningocele the tumour usually grows slowly, and ultimately may be shut off from the cranial cavity; but gradual thinning of the membrane may take place, and spontaneous or accidental rupture occur. In encephalocele the tumour grows slightly, or not at all. Most of these patients exhibit signs of mental impairment or other evidences of organic brain disease.

Treatment.—According to Treves, operation is justifiable only in case of impending rupture. The conditions present are essentially the same as in spina bifida. Meningocele may be aspirated, injected with iodine, or with Morton's iodine and glycerin solution (page 765); the sac may be laid open and a plastic operation performed for the closure of the communication with the cranial cavity; or the skin may be divided, and a ligature or clamp applied to shut off the communication with the brain. All these methods have been at times successful, but cure has in many instances been followed by the development of chronic hydrocephalus. Encephalocele is to be treated by protection and compression. Aspiration may be resorted to if fluid is present. In hydrencephalocele the prognosis is absolutely bad under all circumstances. Schatz* gives the following statistics, showing the results with and without operation, all varieties being included: Of twenty-four occipital tumours not operated on, three

* Berlin. klin. Wochenschrift, No. 28, 1885.

recovered; of thirty-five operated on by excision, ligation, or injection, six recovered. Of forty-six frontal tumours, there were six recoveries in thirty-two cases without operation, and two recoveries in fourteen cases with operation.

Microcephalus.—This is generally regarded as due to premature ossification of the skull; but this theory is certainly inadequate to explain all the cases. In many children suffering from marasmus, the sutures ossify and the fontanels close much earlier than in healthy infants of the same age, chiefly because, with the rest of the body, the brain also has ceased to grow. So it is true of some of the cases, at least, of microcephalus, that the early ossification of the skull is due to arrested growth of the brain, and not the reverse. The reasons for the developmental arrest in the brain are for the most part unknown. The condition usually dates back to intra-uterine life, although in some cases it appears to begin after birth.

It is well known that there is not an invariable relation between the size of the head and the size of the brain, although generally the two correspond. If the circumference of the head is much below the average for the age (page 20), and relatively much less than the measurements of the rest of the body, microcephalus may be assumed to exist. Sachs calls attention to the fact that the circumference of the head may be nearly normal and yet the essential conditions of microcephalus exist, owing to imperfect development of the anterior part of the brain.

The symptoms of microcephalus are those of idiocy and cerebral paralysis, existing in all possible combinations and with variable degrees of severity.

A new surgical interest in these cases has been awakened during the last few years by the operation of craniectomy. The purpose of this operation, which was devised by Lannelongue, is to relieve the intracranial pressure by making a longitudinal opening in the skull, on one or both sides. The opening made is usually about half an inch wide and four or five inches long. It is one or two inches from the sagittal suture, to which it is parallel. For the time being the cranial capacity is increased, but it is doubtful if even this is permanent. Jacobi* gives a report of thirty-three cases operated upon by American surgeons, with fourteen deaths and nineteen recoveries. At the time of report the condition in the cases which survived the operation was as follows: no improvement in seven; slight, in seven; "some," in one; much, in two; no history, in one; uncertain, in one. I quite agree with him that such results do not justify the performance of this operation.

Congenital Hydrocephalus.—These cases may fairly be considered as belonging to this category, although they have been discussed elsewhere.

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* New York Medical Record, May 19, 1894.

Porencephalus (literally, a hole in the brain) is a condition in which there is a large depression in some part of the brain, but with surrounding parts well developed. Such depressions may involve a whole lobe, and they may be deep enough to reach the lateral ventricles.

Porencephalus is described as congenital or acquired. In the congenital form, the defect is usually found in the anterior or middle part of the brain. The origin of these conditions is still a disputed question. They are probably due to early vascular changes. Children sometimes live several years with very large defects, the symptoms depending upon the seat of the lesion. The acquired form of porencephalus is usually one of the late results of meningeal hæmorrhage. It may affect one or both sides. Such cases present the symptoms of spastic paralysis—usually diplegia. In all cases with large brain defects, the space is filled with fluid.

PACHYMENINGITIS.

Pachymeningitis, or inflammation of the dura mater, occurs both as an acute and a chronic disease.

Acute Pachymeningitis.—This is very rare in children. Only pachymeningitis externa is generally included under this term, as acute pachymeningitis interna does not occur alone, but usually with inflammation of the pia mater (leptomeningitis). It may be associated with disease or injury of the bones of the skull, but is most frequently seen in connection with middle-ear disease. It generally begins as a localized process, but the inflammation may extend to the inner layer, and to the pia mater; or it may remain circumscribed, and terminate in the formation of an abscess between the dura mater and the bone.

The symptoms of acute pachymeningitis are distinctive only when the process is localized. They are then usually associated with middle-ear disease, and are indistinguishable from those of cerebral abscess. The treatment is surgical.

Chronic Pachymeningitis.—This, in children, almost invariably affects the inner layer (pachymeningitis interna); it is also known as *pseudo-membranous* and as *hæmorrhagic pachymeningitis* or *hæmatoma of the dura mater*. Its causes are for the most part unknown. It is not very rare, being usually discovered at autopsy in children, chiefly cachectic infants, who have died of other diseases. In the Report of the New York Pathological Society for 1890 Northrup records six such cases. I have seen five similar ones, as well as one other associated with chronic hydrocephalus.

Two classes of cases are to be distinguished,—those with, and those without extensive hæmorrhages. In the latter group there is found a thin, translucent, vascular membrane lining the inner surface of the dura. It may be only a delicate film which can be scraped off; it may be as thick as ordinary blotting-paper, or even twice that thickness. The membrane

is often œdematous; it is exceedingly vascular, and the vessels have very thin walls. There are usually scattered, punctate hæmorrhages, and there may be a few of larger size. This membrane may cover the whole inner surface of the dura, but in most cases it is principally over the convexity and may be found only here; it is apt to be more upon one side than upon the other. In cases of long standing there may be adhesions between the dura and the pia. When large hæmorrhages have taken place, quite a different pathological appearance is presented. The lesions found in a case upon which I made an autopsy in the New York Infant Asylum, are fairly typical: The infant was six months old, and the symptoms had existed for six days. The fontanel was bulging to a marked degree, and the sagittal and coronal sutures were separated. A thin recent clot from one eighth to one fourth of an inch in thickness covered nearly the whole of the right hemisphere and part of the convexity of the left. The entire dura was lined both at its convexity and base by a pseudo-membrane of grayish color, about one sixteenth of an inch in thickness. The brain was anæmic.

In cases of longer standing partial organization of the clot may be seen; in more recent ones the blood is partly or entirely fluid. I once found acute leptomeningitis with a purulent exudation, associated with hæmorrhagic pachymeningitis. In cases where life is prolonged for years, there may be partial or even complete absorption of the clot, followed by the formation of cysts, considerable inflammatory thickening of the pia with deposits of blood pigment, and finally atrophy and sclerosis of the cortex. The source of the hæmorrhage may be the rupture of a single large vessel, but more frequently the blood comes from many small vessels.

Symptoms.—These are due to the hæmorrhage, and not to the inflammatory process. Until hæmorrhage occurs there are no symptoms by which the disease can be recognised. Thus in many of the cases in which pachymeningitis is found at autopsy, its existence is not suspected during life. The occurrence of hæmorrhage is sometimes marked by vomiting or convulsions, and usually there is loss of consciousness. It may be a question whether the convulsions are the cause or the result of the hæmorrhage. In most cases they seem to be the result. They are usually general and repeated. If the hæmorrhage occurs slowly, there may be stupor without convulsions until nearly the close of the disease. In the fatal cases the symptoms generally continue from two days to a week. There are dulness, stupor, and finally coma, death occuring in coma or convulsions. If the hæmorrhage is diffuse—and this is apt to be the case—there is rigidity of all the extremities; if it is of one side only, the rigidity affects only one arm and leg. The pupils are more frequently contracted, but may be dilated or unequal. There is diplegia, hemiplegia, or monoplegia, according to the seat and extent of the hæmor-

rhage. The respiration is slow and irregular and may be of the Cheyne-Stokes variety. The pulse is slow, irregular, and sometimes intermittent. The temperature is at first normal, but rises slowly until death occurs, when it is from 100° to 103° F. Generally the cranial nerves are not affected, and opisthotonus is absent. The knee-jerk is often exaggerated. In cases which do not prove fatal—these being chiefly in older children—we have a similar onset, but after a few days consciousness is regained, and only hemiplegia or monoplegia remains. The course of the paralysis is that seen after meningeal hæmorrhage due to other causes. Wagner has reported a case in which recurring hæmorrhages took place at intervals of several months, the autopsy showing distinct evidences of both old and recent lesions.

Pachymeningitis, I believe, plays a much more important rôle in the production of meningeal hæmorrhages in children than has generally been accorded to it. From the frequency with which this lesion is found as a cause of sudden meningeal hæmorrhages which are fatal, it is not unlikely that many of the cases which recover with hemiplegia or monoplegia, may be due to the same cause.

The prognosis depends upon the age of the patient and the extent of the hæmorrhage. Extensive hæmorrhages are usually fatal in infancy, but small ones are seldom so, for they are rarely at the base. The prognosis of the paralysis in cases not terminating fatally, is the same as after meningeal hæmorrhage due to other causes, with perhaps an added liability to recurrent attacks.

Without large hæmorrhages, pachymeningitis interna can not be diagnosticated; and it is impossible to differentiate the hæmorrhagic cases from other varieties of meningeal hæmorrhage. It is important to make a diagnosis between pachymeningitis with hæmorrhage, and acute simple meningitis. In the former we have a sudden onset; stupor occurring early, usually on the first day, gradually diminishing in cases of recovery, or deepening into coma in fatal cases; localized or general paralysis, also occurring early; there is no fever in the beginning, and only moderate fever at the close. In acute meningitis we usually have a higher temperature, especially early in the disease; coma develops later, and rigidity of the extremities is less pronounced. In certain cases, however, where the hæmorrhage occurs in the course of some other disease, a differential diagnosis may be impossible.

Treatment.—The treatment of pachymeningitis hæmorrhagica is symptomatic. The indications are, to relieve cerebral congestion by applying ice to the head, to allay irritative symptoms by the use of bromides, and to keep the patient perfectly quiet.

ACUTE MENINGITIS.

Acute inflammation of the pia mater, or acute leptomeningitis, is seen under a variety of circumstances:

1. It occurs epidemically. It is then usually associated with the same process in the cord, and is known as *cerebro-spinal meningitis*, or *spotted fever*, being regarded by many as a general infectious disease with a local lesion.

2. It occurs sporadically as a primary disease, with symptoms and lesions which may be identical with those seen in the first group of cases.

3. It occurs as a secondary disease, complicating other acute infectious diseases and local inflammations.

At the present time we are not able to separate absolutely these three groups by the clinical symptoms, the pathological findings, or even by a bacteriological study of the micro-organisms which are concerned in the process. All the forms will therefore be considered under the same general head.

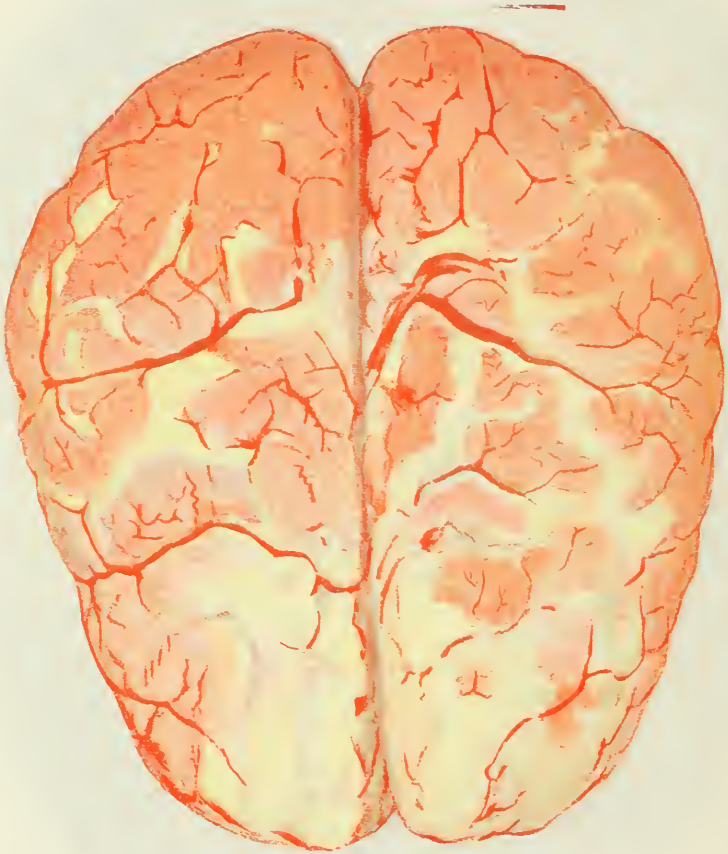
Etiology.—Epidemic meningitis occurs especially in winter and spring; it affects children of all ages, but males more often than females. It is attributed to overcrowding, especially in damp, ill-ventilated apartments, and, in some epidemics, to bad drainage and sewer-gas poisoning. It is not contagious, in the ordinary acceptance of the term. Epidemics are usually separated by intervals of several years, and when they occur the number of persons attacked is rarely large. In New York cases are seen every year; but in some seasons the number is quite large, and the disease is then said to be epidemic.

Sporadic cases of meningitis may result from traumatism or sunstroke, or they may occur without assignable cause after the disease has prevailed epidemically, or even where there has been no epidemic. In the great majority of cases no adequate cause can be discovered.

Acute meningitis occurs as a secondary disease, complicating pneumonia, scarlet fever, variola, influenza, and typhoid fever. I once saw acute simple meningitis as a complication of pulmonary tuberculosis. It not infrequently complicates acute nephritis, especially when this has followed scarlet fever. It may be secondary to otitis media, erysipelas of the scalp, or abscess of the brain.

The bacteriological findings in the cases of cerebro-spinal meningitis thus far have not been uniform. The micro-organism most frequently found has been the pneumococcus (micrococcus lanceolatus). Some recent writers are inclined to regard this as the characteristic germ of epidemic meningitis. The pneumococcus, however, is found in sporadic cases, even in pure culture, as in one of my own patients, an infant of thirteen months; but, on the other hand, during the winters of 1893 and

PLATE XV.



ACUTE MENINGITIS, COMPLICATING PLEURO-PNEUMONIA.

Child twenty months old; on twenty-third day of a protracted attack of pneumonia, vomited six times, and the temperature, which had been nearly normal for four days, rose to 103° F. On the following day general convulsions, which were repeated frequently during the next few days; temperature, 101° to 104° F.; death in convulsions on twenty-eighth day.

Autopsy.—Pleuro-pneumonia of left side; lung resolving. Anterior portion of brain enveloped in lymph and pus, more marked at the convexity, but present also over the base.

1894, when the disease was regarded as epidemic in New York, Biggs found that in cases observed in one hospital (Bellevue) with similar symptoms and with the same gross lesions, there was no uniformity in the bacteriological findings. The pneumococcus was present in some, in others the streptococcus or staphylococcus, each form usually existing in pure culture in the case in which it was found.

While the pneumococcus is undoubtedly the micro-organism most frequently concerned in epidemic meningitis, it is certainly not the only one. In sporadic cases also it plays the most important part. Of twenty-five such cases studied by Netter, the pneumococcus was found in eighteen, the streptococcus pyogenes and staphylococcus pyogenes albus in four, and various other bacteria in the remainder. In the secondary cases, the pneumococcus is usually found when meningitis complicates pneumonia or influenza. Under other circumstances, any of the varieties of pyogenic bacteria may be met with.

Lesions.—In the most severe cases, and especially when the disease is prevailing epidemically, death may take place so early that the changes found at autopsy are slight. There may be only a serous exudation and intense hyperæmia, this being much less marked after death than during life. The microscope, however, may show, even in these early cases, an abundant exudation of leucocytes in the pia mater. In other cases, especially in infants, we may find an extensive purulent exudation where the symptoms have apparently lasted only twenty-four hours. In cases of three or four days' duration the lesions are quite uniform. The convolutions appear somewhat flattened from pressure due to distention of the ventricles. The inner surface of the dura is usually normal or only congested. There may be thrombi in any of the cerebral sinuses, or in the meningeal veins of the convexity. The brain is enveloped in an exudation of greenish-yellow lymph, which is usually abundant, and may nearly conceal the convolutions (Plate XV). It is generally most marked over the anterior half of the brain, and at the base, occurring elsewhere in patches. Exceptionally it may be found only at the base or at the convexity, but usually it is very extensive. There is an increase in the quantity of cerebro-spinal fluid. The ventricles are moderately distended with serum or sero-pus, and their walls may be slightly softened. To the naked eye the brain substance may show no changes except some congestion of the superficial layers of the cortex. In the meninges of the cord lesions similar to those of the brain are usually seen. The exudation is principally upon the posterior surface, and may extend throughout the entire length of the cord, or be limited to its upper or to its lower portion. In some cases the cord lesion is overlooked, because the whole cord is not examined.

Microscopical examination shows the exudation to consist of fibrin and pus cells, which infiltrate the pia mater and may cover its surface.

The superficial layers of the cortex in the inflamed areas sometimes show minute hæmorrhages and very marked cell-infiltration. Extension from the meninges to the substance of the cord is less common. Inflammatory products may be found in the central canal of the cord, and occasionally in the walls of the lateral ventricles of the brain. The lesions most frequently found in other organs, are acute parenchymatous degeneration of the liver, spleen, and kidneys, pneumonia, pleurisy, and peritonitis.

In sporadic cases of meningitis the lesions are identical with those above described. In the secondary cases, as a rule, the cord escapes, although the lesions in the brain are usually the same as when the disease is primary. When meningitis occurs as an extension from otitis, it begins in most cases as a localized process, and afterward becomes general. It is usually complicated by septic thrombosis of the lateral sinuses.

In the cases of meningitis which recover, there is an absorption of the greater part of the inflammatory products; but the pia mater may be thickened and adherent to the brain; areas of sclerosis may develop in the cortex, and chronic hydrocephalus may follow. I have three times had the opportunity of making autopsies upon cases which died at periods varying from four months to a year after the original attack of meningitis. There were found in all of them, thickening and cloudiness of the pia mater, usually most marked at the base. No remains of the exudation were seen except small deposits of fat occurring in irregular patches at the base and the convexity, not unlike miliary tubercles. This was seen in regions where the lesions had been most intense. In one case dying six months after the acute attack, the pia was adherent over the entire cortex of the brain;* the microscopical examination showed a thickening of the pia mater with an exudation of cells between the pia mater and the brain, and in places a commencing secondary encephalitis. A continuance of such a process as this may give rise to a localized or a diffuse sclerosis which may impair the functions and growth of the brain. Such lesions are most frequently seen over the frontal and temporo-sphenoidal lobes.

Symptoms.—Few diseases are so irregular in their course or present so many atypical forms, as does acute meningitis.

1. *The common form.*—Most of the sporadic and epidemic cases are of this type. The acute symptoms are sometimes preceded by a prodro-

* The clinical features of this case were also interesting. The patient was a bright little girl of four and a half years, who had in May a typical attack of meningitis of moderate severity. She made a very slow convalescence, but at the end of two months recovery was perfect in everything but her mental condition. She remembered nothing which she had previously learned in the kindergarten, where she had been an exceptionally bright pupil. Her mind was a blank. She was dull, listless, and her face had a vacant, idiotic expression. The special senses seemed unaffected, and speech was retained. She died during an attack of convulsions in November.

mal stage of one or two days, characterized by general weakness and indefinite *malaise*, but in the majority this is wanting, and the attack begins suddenly with vomiting or convulsions, headache, and high fever. The initial temperature is from 102° to 105° F. There are present intense headache, marked prostration, pain in the back of the neck and along the spine, general hyperæsthesia, opisthotonus, constipation, retraction of the neck, and rigidity of the cervical muscles. Later, more intense nervous symptoms develop. There is delirium, which is often active, to which are added muscular twitchings, and sometimes convulsions; or there may be dulness, apathy, and finally complete coma. The respiration is slow, sometimes irregular. The temperature is elevated, usually between 101° and 104° F. There are seen in a few of the cases fine petechial spots upon the face, abdomen, or all over the body. The pupils are irregular; there may be strabismus or nystagmus. The pulse is weak, and sometimes slow, sometimes rapid.

After these symptoms have lasted from two to ten days, the patient may become completely comatose, with general relaxation and dilated pupils, and may die in this condition or in convulsions. In other cases he passes into a typhoid condition, and death occurs from exhaustion or complications, particularly pneumonia. The usual duration of these attacks is from one to two weeks. If the case recovers there is a gradual subsidence of the nervous symptoms and sometimes quite a rapid convalescence; or the disease may pass into a subacute form, lasting from three weeks to two or three months, improvement being slow and interrupted by relapses. Severe cases may be followed by deafness, localized paralysis, or an impaired mental condition.

2. *Abortive cases.*—In every epidemic there are seen attacks which begin precisely like those above described, but where the symptoms last only two or three days and then subside rapidly, the case going on to a complete and permanent recovery. In some epidemics the number of such cases is quite large.

3. *Malignant or fulminating cases.*—These also occur principally in epidemics, but are not confined to them. The onset in this type is very abrupt, and the patient may be overcome by the poison and die in from twelve to thirty-six hours. These cases often begin with convulsions and very high temperature, from 104° to 106·5° F. There is very great prostration and frequently cyanosis. There may be opisthotonus and general hyperæsthesia, or these may be absent. The patient may pass in a few hours into a condition of collapse, with general relaxation, feeble, irregular pulse, and cold extremities, followed by convulsions and death. If life is prolonged, there may follow after a few hours a period of reaction, in which irritative symptoms are prominent,—headache, photophobia, contracted pupils, general hyperæsthesia, and active delirium. The eruption may appear within the first twenty-four hours after the onset.

In most of these cases a positive diagnosis is impossible, as the general toxic symptoms mask the local evidences of cerebral inflammation. The diagnosis is not likely to be made except when the disease is prevailing epidemically.

4. *Acute primary meningitis occurring sporadically* does not differ in any essential particulars from the epidemic form. The fulminating and the abortive cases are, however, less frequent than when the disease is epidemic.

5. *Acute secondary meningitis* presents quite a different clinical picture, and the symptoms are greatly modified by those of the original disease. In general, its course is shorter, and it is more uniformly fatal than is primary meningitis. The diagnosis is difficult, and in many cases the lesions are found at autopsy where no marked cerebral symptoms have existed during life. This is particularly true where the process is mainly at the convexity. The onset is generally with convulsions, after which there may develop quite rapidly stupor and finally coma, with dilated pupils, slow pulse, and irregular respiration. Convulsions and gradually deepening stupor may be the only symptoms; or there may be opisthotonus, retracted abdomen, and rigidity of the extremities. The duration of these cases is quite short, being rarely more than three or four days, and often but one or two. Death usually occurs in convulsions.

The nervous symptoms.—Headache is a frequent symptom of meningitis and is often severe; it is more likely to be frontal than elsewhere, although it may be general and associated with vertigo. There may also be pains in the back of the neck, along the spine, or in the muscles, which may be so intense as to cause the patient to scream out. Pain may be present only in the early stage, or continue throughout the disease. With this there may be tenderness along the spine, and often general hyperæsthesia, which may be so acute that any movement causes agonizing cries. Delirium is frequent in the severe cases after the first day; it may be wild and active, or low and muttering. After delirium there follows usually a stage of apathy which may develop into complete coma; deep coma, however, is not often present in cases that recover. Convulsions mark both the onset and the close of the disease, but rarely occur during its progress. Tonic spasm of the various muscles gives rise to deformities which may continue through the attack. The rigidity and contraction of the muscles of the neck produces cervical or general opisthotonus; there may be tonic flexion or extension of the extremities, especially of the legs. In some epidemics opisthotonus is seen in nearly every case, in others it is infrequent. In most of the protracted cases localized paralysis is present in the course of the disease. It may affect one side of the body, or one extremity.

Special senses.—The eyes are affected in almost all severe attacks. The pupils in the early stage are generally contracted, later they may be irregular, and toward the close they are usually widely dilated. External

strabismus is by far the most frequent form of ocular paralysis. The fundus is rarely normal. In a study of thirty-five cases, Randolph (Baltimore) noted the following changes: The fundus was the seat of venous engorgement and tortuosity, with more or less congestion of the optic disc in nineteen cases; there was optic neuritis in six cases; retinitis with thrombosis of the central vein in one case. Of the seven cases in which the fundus was normal, one had strabismus, one nystagmus, and one greatly dilated pupils. Inflammation of the conjunctiva is also very frequent. Deafness is common during the acute stage of the disease, and is its most frequent sequel. It may be due to the cerebral lesion, to otitis media, or to otitis interna. The last mentioned may result from an extension of inflammation along the course of the auditory nerve.

Speech is disturbed in most of the protracted cases. Bulging of the fontanel is one of the regular symptoms in young infants. Marked prostration is always present; it may come very abruptly, and may be followed by collapse, or may last but a short time and be followed by a period of reaction.

The *temperature* is always elevated, being especially high at the onset. In the fulminating cases there may be hyperpyrexia,—105° or even 106° F. The usual range is between 100° and 104° F. In cases terminating in recovery, the fever usually lasts from one to two weeks and gradually falls to normal. There is no regular or typical curve. The height of the temperature may bear no relation to the severity of the other symptoms. It may be low throughout, even in the fatal cases. A subnormal temperature is also a bad sign.

The *respiration* is slow and irregular as the disease progresses, and it may be of the typical Cheyne-Stokes variety. Cyanosis is often present in cases where no cause for it can be found in the heart or lungs; it is especially frequent in the fulminating cases. Pneumonia is one of the most common complications.

The *pulse* in the early stages is full and rapid; later it becomes slow, irregular, and feeble, and may be intermittent.

The examinations of the *blood* made by Barker and Flexner (Baltimore) showed the presence of marked leucocytosis in every fatal case examined. Epistaxis is not uncommon as an early, and sometimes as a late, symptom.

Digestive system.—Vomiting is frequent at the onset and may be persistent. The bowels as a rule are constipated, although there may be diarrhœa, and as a complication even dysentery has been observed. The tongue is often coated; sometimes it is dry and glazed, or covered with sordes. Deglutition is sometimes difficult on account of the retraction of the neck. The spleen is usually not enlarged. Jaundice occurs in a small proportion of the cases.

Eruptions.—In the majority of cases, the skin presents no changes

In others there is herpes of the lips, face, or nose, or an eruption over the face or body consisting of fine purpuric spots, and sometimes larger extravasations. These are particularly significant when seen upon the face or the ears, and from this symptom the name "spotted fever" has arisen. In some cases a general erythema is present. The petechial eruption may be seen during the early part of the disease, even in the first twenty-four hours. Late in the protracted cases there may be fine punctate hæmorrhages over the abdomen, as in any exhausting disease.

The large joints, particularly the knees, are often swollen, tender, and painful, the symptoms resembling those of acute rheumatism. Incontinence of urine and fæces may occur in the late stages of the disease, associated with low delirium and other typhoid symptoms. Retention of urine is not infrequent, and often overlooked.

Course, Termination, and Prognosis.—The duration of the disease in the fatal cases is usually less than a week. In epidemics many deaths occur within forty-eight hours. In infants also the course is very short. Of the cases which terminate in recovery, if we exclude the abortive cases, the majority last at least two weeks, and very many run a protracted course. After three or four weeks, there is in such cases a gradual subsidence of the fever and of most of the acute nervous symptoms; but the child remains emaciated, very weak, with occasional attacks of headache, general pains or hyperæsthesia, and often with some localized paralysis. This may slowly disappear, or it may be permanent. The child may recover perfectly so far as all the physical functions are concerned, but be mentally deficient.

The sequelæ of meningitis relate chiefly to the nervous system. There may be hemiplegia or monoplegia, followed by contractures, which may be temporary or permanent. Of the special senses, hearing is most liable to be affected, deafness being quite common after severe attacks, and deaf-mutism not an infrequent result in young children. Blindness is rare, and may be due to optic-nerve atrophy or rarely to the cerebral lesion. Speech is sometimes affected; and all grades of mental disturbance are seen after an attack. As a late result epilepsy may develop.

Meningitis is usually more fatal when it occurs epidemically than in sporadic cases. The mortality in different epidemics varies from thirty to seventy-five per cent. The younger the patient the worse the prognosis, and in infants the disease is usually fatal.

Diagnosis.—The diagnosis of acute meningitis presents unusual difficulties in young children, because of the frequency with which cerebral symptoms are seen in all forms of acute disease, both at the onset and late in their course. In infants the usual mistake made is to diagnosticate meningitis where there is none, rather than to overlook it when it is present. The symptoms most to be relied upon for diagnosis, are continued stupor or coma, opisthotonus, slow pulse and irregular respiration

—especially if associated with high fever—localized paralysis, rigidity of the extremities, and a retracted abdomen. Cases where the principal lesion is at the convexity are particularly obscure, and the diagnosis often is not made during life. There is no opisthotonus or cranial-nerve symptoms, and irregularity of pulse and respiration are rare.

At the onset, meningitis is most likely to be confounded with pneumonia, scarlet fever, and influenza. Pneumonia is recognised by the accelerated respiration and the physical signs; scarlet fever, by the congestion of the throat and the eruption; from influenza the diagnosis may be almost impossible except from the course of the disease. From all other diseases, meningitis is differentiated by the continuance and the severity of the nervous symptoms, rather than by the presence or absence of single or special symptoms.

Quincke's procedure of lumbar puncture of the spinal canal * furnishes a means of differential diagnosis of considerable value. It is especially useful in distinguishing meningitis from other diseases accompanied by marked cerebral symptoms. In meningitis there is invariably found, according to Wentworth (Boston), a distinct cloudiness of the cerebrospinal fluid. In some cases this is very marked, in others it is so slight as to require careful comparison with distilled water in a test-tube, to make it apparent. In addition there may be found during inflammation an excess of albumin, a deposit of leucocytes, and any of the various bacteria which produce meningitis.

A differential diagnosis between epidemic meningitis and the sporadic form is impossible. The diagnosis of simple from tuberculous meningitis is easy in typical cases, but in certain forms of the disease it is extremely difficult and sometimes impossible. The most striking points of contrast are, that in simple meningitis the onset is usually abrupt; the temperature is high; the disease develops rapidly; and in forty-eight hours—sometimes in twenty-four—nearly all the severe nervous symptoms may be present; pain in the spine and general hyperæsthesia are quite frequent. Usually the patient is a child who has been in perfect health up to the beginning of the disease; or there is present some local cause, such as middle-ear disease, or traumatism; or an epidemic may be prevailing. In tuberculous meningitis, the onset is usually insidious; the temperature low; the pros-

* Puncture is usually made between the third and fourth lumbar vertebræ a little to one side of the median line. The smallest exploring needle may be used, and for convenience it may be attached to a syringe as a handle, as it is not necessary to aspirate. The canal is reached at a variable depth, usually about one inch from the skin. The body should be flexed during the operation so as to separate the vertebræ, and unless the patient is comatose an anæsthetic is advisable. All observers agree that with a clean needle lumbar puncture is harmless. See Jacoby, New York Medical Journal, December 28, 1895, and January 4, 1896; Caillé, New York Medical Journal, June 15, 1895; and Wentworth, Transactions of the American Pædiatric Society, 1896.

tration not marked for the first few days; the evolution of the nervous symptoms is often slow and irregular, and the child may be sick a week before he appears to be seriously ill; pain in the spine and general hyperæsthesia are rare. The child is usually one who has a history of hereditary tuberculosis; or who has been previously delicate, or who has suffered already from some other form of tuberculosis, in the lungs, bones, or lymph nodes. In cases of sporadic meningitis which are apparently primary, the tuberculous is much more frequent than the simple form,—in my experience fully three to one.

Treatment.—The treatment of acute meningitis is quite unsatisfactory, and it is very doubtful whether the result is greatly modified by any special plan of treatment; it seems to depend upon the age of the patient, and the nature and severity of the attack, rather than upon its management. The treatment directed toward the inflammation consists in the constant use of an ice-cap to the head, and at times an ice-bag along the spine. Counter-irritation may be maintained by painting the nape of the neck and the spine daily with a strong tincture of iodine, or by blisters, but best of all by the Paquelin cautery. The bowels should be kept freely open by calomel or saline cathartics. Internally, ergot and iodide of potassium should be given in as full doses as will be tolerated by the stomach.

Of the symptoms which call for special treatment, the most prominent one is pain, which when severe requires morphine, often in large doses. It is often best to give it hypodermically. For other nervous symptoms—delirium, sleeplessness, etc.—the bromides and chloral, sulfonal, or trional may be given, or warm sponge baths. Stimulants are required in most of the cases at some time in the course of the disease. They are indicated by weak, rapid, and irregular pulse. Alcohol and digitalis should be used, but not strychnine. The difficulties in feeding these patients are sometimes great, but they can often be overcome by the use of gavage (page 62), which may be advantageously employed as a routine practice in the most severe cases. The physician should be on the watch for bedsores, and endeavour to prevent them by cleanliness, frequently changing the patient's position, etc. The bladder also must not be forgotten, as retention of urine is not uncommon, and may require the use of the catheter.

For the residual paralysis, massage, warm baths, and friction should be employed, but electricity only when all symptoms of central irritation have subsided. The prolonged use of iodide of potassium seems to have considerable influence in promoting absorption of the inflammatory products in cases where there is a persistence of symptoms for two or three months.

TUBERCULOUS MENINGITIS.

Synonyms: Acute hydrocephalus; basilar meningitis; water on the brain.

Tuberculous meningitis is a tuberculous inflammation of the pia mater of the brain, sometimes involving also that of the cord. It is doubtful if it ever occurs as the only tuberculous lesion of the body. It is quite frequently seen, and is more uniformly fatal than any other disease of early life. In infancy it is usually associated with general or pulmonary tuberculosis; in older children with tuberculosis of the bones, joints, or lymph nodes. Of my own cases, twenty-five per cent of all deaths from tuberculosis in children, were due to meningitis.

Lesions.—The lesion consists in the production of miliary tubercles, with which are frequently found tuberculous nodules of variable size, and in almost every case there are also the products of ordinary inflammation of the pia mater—lymph and pus—together with an accumulation of fluid in the lateral ventricles of the brain. Frequently there are tubercles in the pia mater of the upper portion of the cord. The miliary tubercles appear as small gray or white granules, situated along the vessels of the pia mater. When few in number they are usually only at the base, especially along the Sylvian fissures and in the interpeduncular space. When numerous they are most abundant at the base, but are also seen scattered over the convexity in small groups. In about half of my autopsies they have been limited to the base, and in no case were they seen exclusively at the convexity. Tubercles are often found in the choroid coat of the eye. The amount of lymph and pus present is rarely great, and never equal to that seen in simple acute meningitis. It is often a matter of surprise at autopsy to find the lesions so few, after very marked symptoms. The inflammatory products are most abundant at the base. In addition to the patches of greenish-yellow lymph, there are adhesions between the lobes of the brain and thickening of the pia. In cases which have lasted for several weeks, the pia mater in places is often very much thickened, owing to cell infiltration and the production of new connective tissue, and it is studded with miliary tubercles, sometimes with small yellow tuberculous nodules; frequently there is arteritis, which is sometimes obliterating.

In the most acute cases the brain substance immediately beneath the pia is intensely congested, slightly softened, and shows under the microscope a superficial encephalitis. The lateral ventricles are usually distended with clear serum, sometimes with serum containing flocculi of lymph or pus; the amount present varies from one to four ounces in each ventricle, being always greater in the subacute cases. The walls of the ventricles may be softened. The distention of the ventricles leads to flattening of the convolutions from pressure against the skull, to bulging

of the fontanel, and sometimes to separation of the sutures, if they are not completely ossified.

Tuberculous nodules varying in size from a small pea to a walnut are frequently seen associated with meningitis in older children, but not so often in infants. These nodules may be connected with the meninges, or they may be situated within the brain substance, usually in the cerebellum. The larger ones are classed as brain tumours. Inflammatory products are rarely found in the spinal canal.

Although it is not infrequent to see meningitis without symptoms of tuberculosis elsewhere, I have never failed at autopsy to find other tuberculous lesions in the body. In my own experience the following are those most often met with, given in the order of frequency:

(1) In infants, associated with general or pulmonary tuberculosis; (2) in children from three to twelve years of age, with tuberculosis of the vertebræ, hip, knee, or ankle; (3) at any age, with tuberculosis involving only the tracheal, bronchial, or cervical lymph nodes; (4) much less frequently with the pulmonary tuberculosis of older children. Meningitis has been reported when it was secondary to tuberculosis of the skin or mucous membranes. I have not, however, met with such cases.

Etiology.—Tuberculous meningitis is produced only by the transportation of the tubercle bacilli to the brain. They may find their way by the blood-vessels or lymphatics.

The following table shows the age at which the disease is most frequently observed:

Age.	Personal cases.	Oxley.*	Total.
Under one year	14	3	17
One to two years	9	16	25
Two to five years	24	26	50
Five to nine years	15	18	33
Nine to sixteen years	5	0	5
Totals	67	63	130

In this series, males were a little more frequently affected than females. In two or three instances traumatism was apparently an exciting cause. Tuberculous meningitis is occasionally seen in young children who were previously healthy, whose family history is free from tuberculosis, and where no exposure can be traced. It is probable that in all such cases there has been latent tuberculosis somewhere in the body, and that the exposure was long antecedent to the symptoms. In the majority, however, this is not the case. There is usually a history of hereditary tuberculosis or of exposure to infection; or there have been previous evidences of tuberculosis in the lungs, bones, or lymph nodes.

* Liverpool, Medico-Chirurgical Journal, July, 1885.

Symptoms.—In forty-three of sixty-three cases the onset was gradual; but in a considerable number of those classed as sudden, careful inquiry elicited a history of previous indisposition. The most frequent early symptoms are disinclination to play, or drowsiness; sometimes there is constant fretfulness or irritability. Often a distinct change in disposition is seen. In a case recently under observation this was most striking; from being devoted to her mother, a little girl could not endure her presence in the room. There is loss of appetite, and usually constipation. Sleep is restless and disturbed; there may be grinding of the teeth. Older children often complain of headache. At all ages a suggestive symptom is frequent attacks of vomiting without apparent cause. In addition to these there may be a slight but continuous elevation of temperature. Indefinite symptoms may last for four or five days, or they may be spread over two or three weeks without perhaps being sufficiently severe to attract much notice. Finally, unmistakable evidence of brain disease develops, and then it is recollected that symptoms like the above had existed for some time. These early disturbances are often ascribed to dentition, to worms, or to indigestion; and sometimes they are regarded simply as the result of the constipation.

In the midst of such indefinite symptoms there may come an attack of convulsions, and, in the course of a few hours, deep stupor. The early symptoms of the active stage are indicative of cerebral irritation. There is headache, often located in the frontal region, and occasionally photophobia; sometimes there is sudden screaming out at night without waking. The skin is usually somewhat hyperæsthetic; the reflexes are apt to be exaggerated; the muscles of the neck may be rigid and the head is drawn back, or there may be rigidity of one or more of the extremities. The pupils are normal or contracted; there may be nystagmus. The child is fretful, wishes to be left alone, and cries if disturbed; but otherwise is apt to be unnaturally drowsy. Such symptoms may continue for a day or two, or even for a week. If prolonged, they are likely to alternate with periods of more marked apathy and dulness. During this stage there is occasional vomiting, and the bowels are obstinately constipated. The pulse is usually somewhat accelerated, but may be slow and occasionally is irregular. The respiration is of normal frequency, but a careful observation during sleep or perfect quiet will often show a slight irregularity which is very significant. This becomes more marked as the disease progresses. The temperature is invariably elevated, but never very much so, generally being from 99° F. to 101° F. When a high temperature is seen, it is usually due to tuberculosis elsewhere than in the brain.

During the intermediate or second stage, the irritative symptoms subside, and stupor becomes deeper and more continuous. If undisturbed, the child may sleep a great part of the time, but can be roused, and then appears quite rational. Later the stupor becomes so profound that the

child can not be roused at all; or, again, this condition may alternate with periods of complete lucidity. Active delirium is rare. The pupils respond slowly to light or not at all; they may be unequal; occasionally there is seen strabismus, ptosis, or paralysis of the face. More often there is hemiplegia, or paralysis of one arm or leg. Such paralyses are often transient, disappearing after a day or two. Automatic movements of the extremities, particularly of the arms, are frequent. Muscular twitchings may be noticed. Opisthotonus is marked and well-nigh constant. In infants the fontanel is tense and bulging; the abdomen is retracted, giving the typical "boat-belly." On drawing the finger-nail along the skin of the abdomen, there appears, after a few seconds, a distinct red streak one or two inches wide, which remains for three or four minutes. This is the *tache cérébrale*, and while not pathognomonic, it is almost always present. Other vaso-motor disturbances may be seen. The reflexes are variable; in the early part of the disease they are usually increased, later they are diminished or abolished. The pulse now becomes slow and irregular, often intermittent. The respiration assumes the characteristic type, which consists in the movements becoming deeper and deeper until there is a long sigh, then a complete arrest of respiration for several seconds, after which the movements begin again, at first shallow, but gradually increasing in depth until the sigh is repeated. The accompanying tracing illustrates the type (Fig. 114). An examination with the ophthalmoscope usually shows the presence of choked discs.

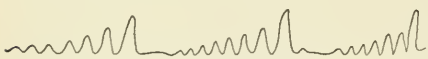


Fig. 114.—Tracing of respiration in tuberculous meningitis.

The duration of this stage is from three to ten days. The progress is irregular, and subject to great variations, especially as regards the mental symptoms. Sometimes a child will be seen in quite deep stupor, and on the following day will be sitting up in bed playing with its toys. Such a course is to be expected, and the physician should never raise any false hopes of recovery because of these periods of temporary improvement.

In the third stage there is complete coma. The child can not be roused at all. The pupils are widely dilated, and do not respond to light. There is general muscular relaxation. There may be retention of the urine. Deglutition is difficult, sometimes almost impossible. The boat-belly and opisthotonus are still marked. The respiration is more rapid, but still irregular. There are sordes on the lips and teeth, emaciation, and anæmia. Toward the end the temperature rises rapidly to 104° F., sometimes to 106° or 107° F. (Fig. 115). The pulse becomes very rapid and feeble, often 160 to 180 a minute. Death usually takes place from exhaustion in deep coma; or convulsions develop and continue from twelve to twenty-four hours until death. The duration of the stage of coma is

from two days to a week. Often the patient will live for four or five days in a condition of prostration so extreme that death is hourly expected. A rapidly rising temperature or the occurrence of convulsions indicates

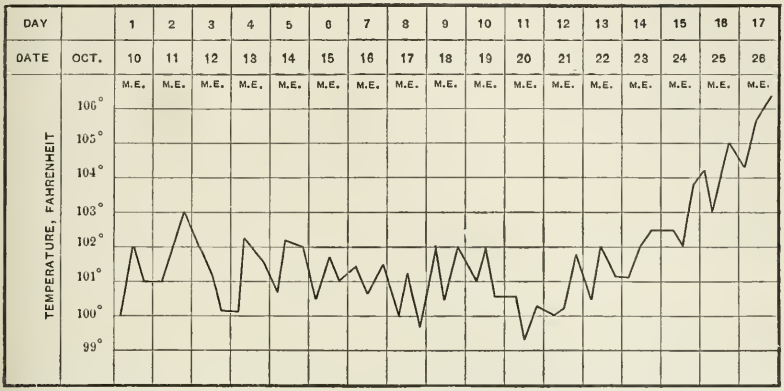


FIG. 115.—Fairly typical temperature curve in tuberculous meningitis; boy, twenty months old; death on seventeenth day.

approaching death. Of fifty-seven cases, fifty died in coma, seven in convulsions.

The entire duration of the disease from the beginning of definite symptoms, in sixty-five of my own cases, was as follows:

One week, or less	17
One to two weeks	15
Two to three weeks	17
Three to four weeks	14
Five weeks	2
	65

Variations in the course of the disease.—There are few diseases which present a greater variety of symptoms than tuberculous meningitis. Typical cases like those above described are seen most frequently in children over two years old, in whom the cerebral symptoms predominate over those of general tuberculosis. In infancy, especially when the disease follows acute tuberculous pneumonia, the duration of the cerebral symptoms may be only three or four days. The stages then are not marked. The onset is usually with convulsions, and in less than twenty-four hours there may be marked stupor, and all the symptoms belonging to the third stage of the disease.

In some cases the course is much longer than that described, the symptoms lasting from four to eight weeks. In character they are much the same as those in the typical cases, except that the irritative symptoms are less marked, and there is less fever. If the child is young, there is great bulging of the fontanel, or even an increase in the size of the head.

In older children the symptoms are chiefly those of a general pressure upon the cortex. These are due to the great accumulation of fluid in the lateral ventricles. The symptoms of general compression are persistent drowsiness, but rarely deep coma, rigidity of all the extremities, and sometimes paralysis. The pupils are usually contracted, but there are no symptoms which are distinctly focal. Opisthotonus is nearly always marked in these cases.

Diagnosis.—There are no diagnostic symptoms in the first stage. If the patient has previously suffered from local or general tuberculosis, and symptoms develop which are enumerated as prodromal, meningitis may be suspected with a strong degree of probability. If the child has previously given no evidence of tuberculosis, a diagnosis is impossible. The indefinite symptoms that belong to the early stage of the disease are frequent in young children suffering from chronic indigestion associated with constipation. In nine out of every ten cases, such will be the explanation of the indisposition rather than incipient meningitis. Disturbances of nutrition, classed as cyclic vomiting (page 287), may present many of the symptoms of meningitis. I have seen two cases in which a differential diagnosis was impossible for two or three days.

The most frequent symptoms of tuberculous meningitis enumerated in the order of their occurrence in fifty-eight cases, were as follows: obstinate constipation, persistent drowsiness, irregular respiration, vomiting without apparent cause, irregular pulse, convulsions, opisthotonus, and fever which was usually slight. Equally important for diagnosis, and especially significant when associated with the above, are strabismus, facial paralysis, and loss of the pupillary reflexes.

The discovery of tubercle bacilli in the fluid drawn by lumbar puncture (page 713) is conclusive. However, this does not add greatly to our means of diagnosis, as the bacilli are never numerous and always difficult to find, and in a number of undoubted cases they can not be found at all. Without finding bacilli we may be quite certain, from the other conditions present, that meningitis exists, but we can not with any certainty separate the simple from the tuberculous cases. The symptoms which distinguish these from each other have already been considered (page 713).

The cerebral symptoms of ileo-colitis and other diarrhœal diseases, sometimes closely resemble those of tuberculous meningitis; but whenever in a young child there is another disease present which may furnish an explanation for the cerebral symptoms, the diagnosis of meningitis should be made with great caution. The development of meningitis in the course of an ordinary attack of pneumonia may simulate very closely pulmonary tuberculosis with tuberculous meningitis. A diagnosis may be impossible during life. In doubtful cases the probabilities are greatly in favour of tuberculosis, since it is so much more common.

Prognosis.—It is still a matter of dispute whether tuberculous menin-

gitis ever ends in recovery. Such a result is certainly so rare as not to be expected. I have never seen it. In certain cases simple meningitis may so closely simulate the tuberculous variety that a differential diagnosis can not be made, and it is possible that the cases of alleged recovery were simple and not tuberculous. Gibney has reported a case of meningitis occurring in a boy with double hip-joint disease, which certainly, so far as symptoms went, should be classed as tuberculous, and yet recovery took place. The child died several months later, of amyloid disease. I was present at the autopsy, and there was found no trace of cerebral tuberculosis. On theoretical grounds there seems to be no reason why recovery may not sometimes follow from meningitis as well as from other forms of local tuberculosis, but as a matter of clinical observation such a result is extremely doubtful.

Treatment.—From what has been said regarding prognosis, it follows that if the diagnosis is correct the case is practically hopeless, no matter what treatment is employed; but as a positive diagnosis is not always possible, all cases should be treated like those of simple meningitis.

CHRONIC BASILAR MENINGITIS IN INFANTS.

Basilar meningitis is generally tuberculous. Not very infrequently there is, however, seen in infants a chronic form of basilar meningitis which is not tuberculous. Attention was first called to these cases by Gee and Barlow, who in 1878 published, under the title of "Cervical Opisthotonus in Infants," six cases of simple basilar meningitis in which the diagnosis was confirmed by autopsy. Since that time a number of other cases have been reported by various writers. I have followed two such cases to the post-mortem table, one of which was undoubtedly syphilitic. I have seen others of a similar nature which have recovered, one of these also being in a syphilitic infant. Not all these cases are syphilitic, but the etiology of the other cases is unknown.

Lesions.—This process is usually limited to the base of the brain. The pia mater is thickened about the interpeduncular space, also over the medulla, pons, and cerebellum. These different parts may be adherent to each other, or to the inner surface of the dura. The cranial nerves may be compressed. The openings in the fourth ventricle are usually obliterated, and there results a distention of the lateral ventricles with clear serum, sometimes in sufficient amount to be regarded as hydrocephalus. Rarely, pus may be found in the ventricles. The lesions thus are very much like those seen in the protracted cases of tuberculous meningitis, minus the tubercles.

Symptoms.—These in all cases are quite uniform. The two most prominent symptoms are cervical opisthotonus and moderate hydrocephalus. The opisthotonus is constant and may be quite extreme. In one of my cases the cervical spine for weeks formed nearly a right angle with the

body. The accompanying illustration (Fig. 116) is from a photograph of this patient. From time to time the opisthotonus varies in intensity, but it never entirely disappears. The degree of hydrocephalus is generally not extreme. It causes the usual symptoms of enlargement of the head, separation of the sutures, and bulging of the fontanel. Mental dulness or apathy is less liable to be present when the disease begins in early infancy, and the cranial bones yield more readily to the increased pressure, than when it comes so late that the sutures are firmly ossified. In addition to these two cardinal symptoms, there are often seen nystagmus, occasional attacks of vomiting without apparent cause, and convulsions more or less

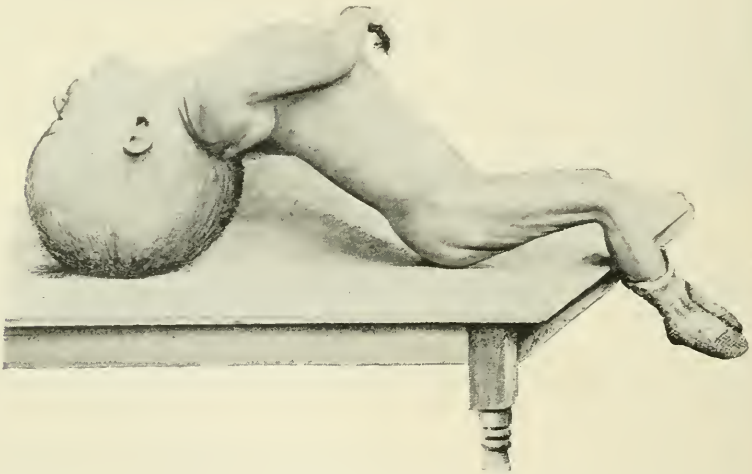


Fig. 116.—Chronic basilar meningitis; a patient in the Babies' Hospital (diagnosis confirmed by autopsy).

severe. There may be tonic rigidity of the extremities, with exaggeration of the reflexes. Febrile symptoms, as a rule, are wanting. The course is essentially chronic. The duration varies usually from one to four months; exceptionally it may last a year. Patients may die from convulsions or from the effects of the hydrocephalus, but more frequently waste and die from marasmus. The prognosis is bad, except in the cases which are due to syphilis, where recovery may take place. How large a proportion of the cases are syphilitic has not yet been determined.

Diagnosis.—The disease is to be distinguished from tuberculous meningitis, and from the opisthotonus of reflex origin, which is occasionally seen in infants suffering from marasmus. It differs from tuberculous meningitis in its more protracted course, in the absence of fever, paralysis, and the evidences of tuberculosis elsewhere in the body, and also in the greater prominence of the opisthotonus and hydrocephalus. The opisthotonus which is seen in cases of marasmus is never so extreme or so continuous,

and is not accompanied by any enlargement of the head, or by other cerebral symptoms.

Treatment.—This consists in the administration of potassium iodide. Although this has little or no influence upon cases not syphilitic, it may cure those which are syphilitic. As it is impossible to distinguish between syphilitic and non-syphilitic cases, every child should have the benefit of a thorough trial of this drug in full doses. At least fifteen grains daily should be given for several weeks to an infant six months old, and still larger doses if the stomach will tolerate it.

THROMBOSIS OF THE SINUSES OF THE DURA MATER.

This is not very frequent. It may depend upon certain general conditions, when it is usually classed as cachectic or marantic thrombosis; it may be associated with local pathological processes, when it is known as inflammatory or septic thrombosis.

Cachectic Thrombosis.—This is seen in infants and young children, but is very rare after the age of five years. It occurs in the course of various diseases, the most frequent being pneumonia, pertussis, diphtheria, nephritis, tuberculosis, and the acute intestinal diseases. In connection with the last-mentioned group, altogether too much has been made of it, as it is really rare, and in only a very few cases does it explain the cerebral symptoms present. This statement is made from personal observations upon over two hundred autopsies upon cases of acute intestinal disease. The actual cause of the thrombosis is the altered condition of the blood and the feeble circulation, as the walls of the sinuses are normal.

The most frequent seat of cachectic thrombosis is the superior longitudinal sinus. At autopsy one must be careful not to confound the soft, partly-decolorized, non-adherent thrombi of post-mortem origin, with those of ante-mortem formation. The latter are firm, and when of long standing may be very hard and even show a laminated structure. They usually fill the sinus completely, and are adherent. The thrombus extends from the sinuses to the veins emptying into it, which stand out like dark worms upon the surface of the brain. The brain itself may be deeply congested, or it may be covered with a diffuse hæmorrhage, but more frequently the brain and the membranes are simply œdematous.

The *symptoms* of cachectic thrombosis are few and uncertain, and in a large number of cases the disease is latent. Very rarely is a positive diagnosis possible during life. When the thrombosis occurs just before death, its symptoms are so mingled with those of the original disease that they can not be separated. In some cases there may be localized or general convulsions, or paralysis, loss of consciousness, and strabismus.

The *prognosis* is bad, cases generally proving fatal in the course of a few days. The diagnosis is so uncertain and obscure that the *treatment*

must be symptomatic, and directed toward the general rather than the local condition.

Inflammatory Thrombosis—Septic Thrombosis—Sinus-Phlebitis.—This condition is most frequent in children in connection with acute meningitis. It may exist either with the simple or the tuberculous variety. It also follows otitis—especially old and neglected cases—usually with necrosis of the petrous bone, but sometimes without it. It is much less frequently associated with disease of the ear in children than in adults. It may arise from traumatism, necrosis of the cranial bones, or from septic processes involving any of the cavities or any of the structures adjacent to the brain, such as the scalp, orbit, nasal fossa, mouth, or pharynx. Infection from the mouth or pharynx is most frequent in children in connection with scarlet fever or diphtheria; while usually secondary to otitis it may occur without it, the infection being carried by the blood-vessels. Infection from the nose may have its origin in ulceration from syphilis or tuberculosis. In the orbit, the source may be malignant disease.

The seat of the thrombosis will depend upon the original disease. If this affects the cranial bones or the scalp, it will be the longitudinal sinus; if the ear, the lateral sinus; if the base of the skull, the orbit, the mouth, the jaw, or the nose is affected, it will be the cavernous sinus. When thrombosis occurs with meningitis the lesions are much the same as in the cachectic form, with the exception that there are sometimes slight changes in the walls of the sinuses. If the patient has suffered from a local septic process, there may be puriform softening of the clot, and general pyæmia, with the development of secondary abscesses in the brain, in the lungs, and in other organs. With such cases there may be associated a general or localized meningitis.

Symptoms.—The symptoms of septic thrombosis are more decided than those of the cachectic form. When occurring in the course of meningitis, it usually adds no new symptoms to those of the original disease. In the pyæmic form the symptoms are more characteristic, particularly when associated with otitis. There are recurring chills with very high and widely-fluctuating temperature. There is headache, and often localized tenderness of the scalp; the other symptoms which are present are usually the same as those of meningitis. If metastasis occurs, there may be evidences of abscesses of the brain or in other organs, and sometimes there are signs of suppuration in the jugular vein.

The local symptoms of the thrombosis differ somewhat according to the sinus affected: if its seat is the superior longitudinal sinus, there may be cyanosis of the face, dilatation of the temporal and frontal veins, and sometimes epistaxis; if the lateral sinus is involved, the process may extend to the jugular vein, which may be felt in the neck as a hard cord, and there may be dilatation of the veins of the mastoid region, and even localized œdema; when the cavernous sinus is affected, there may be pro-

trusion of the eyeball of the affected side, œdema of the lid, and with the ophthalmoscope the retinal veins appear enlarged and tortuous, sometimes being the seat of thrombosis. The process may affect either one or both sides. The course of septic thrombosis is rather irregular, varying from a few days to three weeks. In fatal cases death takes place from meningitis, cerebral abscess, or pyæmia. The prognosis is very grave, unless the disease is so situated that it is accessible to surgical operation.

Treatment.—The only successful treatment is surgical. Operation is easiest in thrombosis of the lateral sinus, being much more difficult if involving the superior longitudinal sinus. So many cases are now on record of successful operation upon septic thrombosis of the lateral sinus, that it should always be urged when the diagnosis is clear. Recurring chills and high, fluctuating temperature, associated with disease of the ear, either with or without symptoms of meningitis, are sufficiently characteristic to justify operative interference.

CEREBRAL ABSCESS.

Cerebral abscess is quite rare in children, decidedly more so than is cerebral tumour. In Gowers' collection of 223 cases, only 24 were under ten years of age. In infants, abscess is one of the least frequent diseases of the brain, and up to five years it is exceedingly rare.

Etiology.—By far the most frequent cause in children is otitis. This is the origin of the great majority of the cases. Abscess rarely complicates acute otitis, but is seen with the chronic form. Exactly how otitis causes cerebral abscess it is not always easy to determine. Toynbee was the first to call attention to the fact that cerebellar abscess was most frequent with disease of the mastoid cells, and cerebral abscess with otitis media. Usually there is caries of the petrous bone, but there may be none. The infection may extend through the small veins traversing this bone, or along the lateral sinuses to the cerebellum. Abscess is often attributed to the retention of pus in the ear, but it may occur when the discharge is free.

Traumatism is the second important etiological factor. Abscess may be associated with fracture of the skull, or follow simple concussion. The abscess is generally in the neighbourhood of the injury, but occasionally is produced by *contre coup*. In one instance, reported by Wagner, thrush was believed to be the cause of cerebral abscess, the same fungus that existed in the mouth being found in the brain, which in this case was studded with small abscesses. Abscess may be the result of infectious emboli, associated with general pyæmia, though this is rare in early life; and finally it may occur without any assignable cause.

Lesions.—The most frequent seat of the abscess is, first, the temporosphenoidal lobe; secondly, the cerebellum; thirdly, the frontal lobes. Other locations are very rare. Abscesses are usually single. In size they

vary from that of a small cherry to an orange. One case was observed by Meyer, in which an abscess occupied one entire hemisphere. The contents are usually thick greenish-yellow pus, which may be very fetid. When abscesses have lasted for some time they are usually surrounded by dense pyogenic membrane, and may become encysted. The pathological process may be slow, and often is apparently stationary for a long period. Abscesses may rupture into the ventricles, less frequently upon the surface of the brain, causing meningitis, or the pus may even escape externally through the auditory meatus, as in Lallemand's case.

Symptoms.—These are general and local. The general symptoms are much the more important for diagnosis, and often are the only ones present. The local symptoms are those of a tumour. The clinical history of a case of abscess of the brain may be divided into three stages: First, the period of onset, or early acute inflammatory symptoms, fever, etc., which attend the formation of pus. Secondly, the latent period, or period of remission, in which very few symptoms are present. In many acute cases this stage is wanting altogether; in the chronic cases it may last for months, or even years. Thirdly, the final period, with recurrence of active cerebral symptoms, followed by death in a few days.

The onset may be accompanied by symptoms so slight as almost to escape notice. In most cases, however, headache and fever are present. The headache is usually severe, and often localized upon the affected side; in cerebellar abscess it may be occipital. The fever is moderate in intensity, and continuous. In addition there may be vertigo, vomiting, general convulsions, and cessation of the aural discharge, if one has been present. The duration of this stage is variable; it may be only a few days, or several weeks. It is shorter in traumatic cases, and in those which are due to pyæmia.

The latent stage, or period of remission of symptoms may be quite short—only a few days' duration—and it is often absent. During this period the temperature may fall quite to the normal, and the headache disappear, or be only occasional and slight. However, if any focal symptoms have been present they remain unchanged.

The symptoms of the terminal stage are due to a rapid extension of the inflammatory process, with œdema and softening about the abscess, sometimes to rupture into the ventricle, and sometimes to meningitis. The fever now returns, and may be high. There is headache, often very intense and continuous; there may be delirium and convulsions, and the gradual development of coma. In addition there may be vomiting, paralysis, opisthotonus, retracted abdomen, and the other symptoms of meningitis. Occasionally all the earlier symptoms may be latent, and the terminal symptoms may be the only ones present. In infants, the fontanel is usually large and bulging; convulsions are rather more frequent than in older children.

The local symptoms of abscess are rather indefinite, owing to its usual situation. Abscesses of considerable size may exist in the temporo-sphenoidal lobe, in the central part of the frontal lobe, or in the cerebellum, without any definite local symptoms. If the abscess is near the motor area, there are the usual symptoms of disease in this location, spasm, or paralysis of the face, arm, or leg. A cortical or sub-cortical abscess is likely to cause convulsions. Cerebellar abscess may give rise to occipital headache, frequent vomiting, and when the abscess is large enough to press upon the middle lobe, there may be inco-ordination of the muscles of the extremities. Optic neuritis may be present, but other symptoms relating to the cranial nerves are rare. Localized tenderness over the scalp, when persistent, is a symptom of importance, and may serve to locate the abscess, if it is superficial.

Diagnosis.—Of the general symptoms, the most important for diagnosis are fever, headache, delirium, and terminal coma. These become particularly significant when following otitis or traumatism. The differential diagnosis of abscess is to be made principally from tumour and meningitis, and from these conditions more by the history and general course of the disease than by any special symptoms. The diagnosis of abscess from tumour is considered in connection with the latter disease. It is more difficult to distinguish between meningitis and abscess, since the two processes are often associated. With meningitis convulsions are more common, but they are rarely localized; rigidity and the inflammatory symptoms are more intense; the course is usually more rapid and more regular, being rarely interrupted, as is the course of abscess. From the cerebral symptoms occurring with otitis it is extremely difficult to distinguish abscess, for, according to Gowers, optic neuritis may be present in the former as well as in the latter condition. The more intense and prolonged are the cerebral symptoms and the more marked the neuritis, the greater are the probabilities of abscess.

Prognosis.—The prognosis in cerebral abscess is always grave, unless accessible to surgical operation. The progress may be slow, or rapid, but it is inevitably from bad to worse, and sooner or later the disease, if not interfered with, proves fatal.

Treatment.—The medical treatment of abscess in its active stage is that of any acute intracranial inflammation,—ice to the head, absolute quiet, free catharsis, and full doses of the bromides or antipyrine or morphine, if pain is intense. The absolutely hopeless condition of these cases when left to themselves, and the recent brilliant results from surgical operations, should lead the physician to urge operation in every case.*

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* For a discussion of the surgical aspects of this question, see "Brain Surgery," by M. Allen Starr, M. D., and "Pyogenic Infectious Diseases of the Brain and Cord," by William McEwen, M. D.

CEREBRAL TUMOUR.

Very little has been added to our knowledge of cerebral tumour in children since the exhaustive monograph of Starr, which appeared in Keating's Cyclopædia in 1890. It is to this article that I am indebted for most of the facts in this chapter.

Varieties and Location.—Tumour of the brain is not very infrequent, and may be seen even in infancy. From this time up to puberty there is no period of special susceptibility. In two hundred and sixty-nine of the cases in Starr's collection, in which the nature of the tumour was stated, the following were the varieties:

Tubercle	152 cases.
Glioma	37 "
Sarcoma	34 "
Glio-sarcoma	5 "
Cyst	30 "
Carcinoma	10 "
Gumma	1 "
	269 "

Tuberculous tumours are more often multiple than are other varieties. Their most frequent seat is the cerebellum; next to this the pons and crura cerebri. They are rarely cortical or central. Glioma is most often found in the cerebellum or in the pons, and next in the cortex; but it is rarely central. Sarcoma is most frequently in the cerebellum; next to this, in the order of frequency, in the pons, the basal ganglia, and the cortex. Cystic tumours are either central or cerebellar. Taking the cases as a whole, the most frequent seat of tumour in children is, first the cerebellum, second the pons, third the centrum ovale.

Tuberculous tumours are occasionally seen in infancy, but they occur most frequently between the ages of five and twelve years. They are usually secondary to tuberculosis elsewhere, especially in the lungs and in the bronchial lymph nodes. They most frequently start from the membranes, rarely being centrally situated, and extend inward, infiltrating the superficial portion of the cerebellum or cerebrum. There is almost invariably localized meningitis at the site of the tumour; there may be adhesions between the dura and pia mater, and the disease may extend to the cranial bones. In size, these tumours vary from a small pea to a child's fist. They may be softened and broken down at the centre, or cheesy throughout. They are the result of a localized tuberculous inflammation, which does not differ essentially from that seen in other parts of the body.

Glioma is not infrequent in infancy. It is probably connected in every case with the ependyma of the ventricle. It repeats the structure of the neuroglia, being composed of connective tissue and branching cells.

Sarcoma may be of the spindle-celled or the mixed variety. It grows much more rapidly than glioma. The two varieties are not infrequently combined in the same tumour—glio-sarcoma.

Cystic tumours are sometimes sarcomatous in origin, the wall of the cyst containing sarcoma cells, and they may also be parasitic, from the growth of the echinococcus. They may be found in any part of the brain.

The other varieties of sarcoma, gumma and vascular tumours, are exceedingly rare until after puberty.

As the tumour grows, secondary lesions are produced in most of the cases. These are the result of pressure upon arteries, causing localized anæmia, or even cerebral softening; or upon veins, producing congestion and œdema. When affecting the middle lobe of the cerebellum, pressure upon the venæ Galeni may lead to effusion into the ventricles. Localized meningitis over tumours superficially situated is the rule, and this may be the cause of some of the symptoms. Rarely, cerebral hæmorrhage may be associated.

Etiology.—The causes of cerebral tumours are for the most part unknown. In a few instances there is a history of definite traumatism. Sarcoma or carcinoma may be secondary, and tuberculous tumours are probably always so.

Symptoms.—These may be divided into two groups: first, the general symptoms which are common to tumours of all varieties, and are independent of location; secondly, the local symptoms depending upon the situation of the growth.

General symptoms.—One of the most frequent is headache. Though it varies much in its severity, character, and position, it is rarely absent. It is apt to be severe, and may continue for a long period, or it may be intermittent. The location of the pain has no definite relation to the situation of the tumour. It may be accompanied by sensations of tightness compression, or tension in the head. It may be associated with localized tenderness of the scalp; when this is constant it is a valuable symptom for diagnosis, as it often occurs with tumours superficially located.

General convulsions often occur in the early stage, but separated by quite long intervals; they become more frequent and more severe as the disease progresses. All degrees of severity are seen, from slight twitchings and temporary loss of consciousness, to typical epileptiform seizures. They are most common when the growth is rapid and when complicating meningitis is present. Attacks of localized spasm may for a considerable time precede general convulsions; and in a single attack there may be first localized and then general convulsions.

Mental symptoms are generally present in great variety and complexity. There may be only fretfulness and irritability, or a marked change in disposition. These symptoms are so frequent from other causes in children that they excite no apprehension, unless to them are added dulness,

apathy, and somnolence. Later in the disease there may be attacks of hypochondriasis, or of melancholia; there may be periods of wild, almost maniacal excitement; and, finally, the mental impairment may approach a condition of imbecility.

Optic neuritis and optic-nerve atrophy are very frequent, occurring, according to Starr, in eighty per cent of the cases. This is only recognised by the ophthalmoscope, as there may be no disturbance of vision. The optic neuritis is generally double, appears earlier, and is more constant in basal tumours than in those at the convexity, or those centrally located.

Vomiting is very frequent, but diagnostic only when it occurs suddenly without assignable cause, and without nausea or other symptoms of indigestion. It is especially significant when frequently repeated, and of more importance in older children than in infants.

Vertigo is often associated with vomiting. At first it is occasional and seen upon changing position, but later it may be quite constant, especially with tumours in the posterior fossa.

Disturbances of sleep are frequent. There is usually insomnia, but sleep may be broken by hallucinations, accompanied by attacks of screaming; rarely is there persistent drowsiness until toward the end of the disease.

Local symptoms.—These depend upon the situation of the tumour, but not at all upon its anatomical character. Local symptoms may be wanting entirely, and they may vary much in different cases even with tumours in the same situation. They are modified by the size and by the rapidity of growth, and by the existence of local meningitis.

In tumours of the cortex, the meninges are likely to be involved, especially with tuberculous and gliomatous growths. The pathological process may extend from within outward or from without inward. The most frequent general symptoms in such cases are headache, circumscribed tenderness of the scalp, convulsions, and mental symptoms. Optic neuritis, vomiting, and vertigo are not so common. Tumours situated in the frontal lobe, as a rule, present few symptoms and may be entirely latent. Irritation of the frontal lobe may extend to the motor area and cause convulsions either local or general; but not often is there paralysis. Tumours of the left side (of the right side in left-handed persons) in the third frontal convolution may cause motor aphasia.

Tumours in the motor convolutions along the fissure of Rolando produce the most definite and uniform local symptoms. When situated at the upper portion the leg is affected, at the middle portion, the arm, and at the lower, the face. Irritative symptoms, such as rigidity or clonic spasm, commonly precede for some time the paralysis which results from pressure or destruction. These attacks of localized convulsions may begin in the face, arm, or leg; but they usually extend more or less rapidly

until all three are involved. There is no loss of consciousness, but there may follow a slight transient paralysis. Such attacks are known as "Jacksonian epilepsy," and form one of the most diagnostic symptoms of cerebral tumour. Localized spasm may be associated with anæsthesia or other disturbances of sensation. The paralysis generally first affects one extremity—the arm or leg, according to the location of the tumour—and afterward it may involve the entire side, including the face.

If the tumour is centrally located, or at the base, hemiplegia may be an early symptom from pressure on the motor tract. With cortical paralysis there may be associated ataxia and anæsthesia.

Tumours of the parietal lobe may give no local symptoms. At times there are disturbances of muscular sense, tactile sensibility, or sensations of pain and temperature. If the inferior parietal lobule of the left side is affected, there may be word-blindness, or inability to understand written language.

Tumours of the occipital lobe produce, as the only constant local symptom, hemianopsia. This is usually bilateral, affecting the same side of both eyes, being on the side opposite to that of the lesion—i. e., a tumour on the right side causes blindness in the left half of both eyes, so that the patient sees nothing to the left of a line directly in front of him. Instead of hemianopsia, there may be only irritation and various disturbances of sight.

Tumours of the temporo-sphenoidal lobe may be latent, or, if on the left side, may cause word-deafness—i. e., inability to understand the significance of spoken language.

Tumours in the island of Reil when situated upon the left side (right side in left-handed persons) may cause motor aphasia or disturbances of speech. If they are large they may produce symptoms by pressure upon the motor tract,—hemiplegia or monoplegia.

Tumours of the basal ganglia cause marked general symptoms, but none of a definitely local character. The important symptoms relate to the various tracts or bundles of fibres which pass from the cortex through the internal capsule. These include the motor and the various sensory tracts, the olfactory, auditory, visual, and speech tracts. Any of these may be pressed upon, and the nature of the symptoms will depend upon the size of the tumour and the extent of the pressure. If only the anterior part of the capsule is affected there may be no symptoms; if the middle fibres, hemiplegia and disturbances of articulation; if the posterior fibres, hemianæsthesia. All these may be associated, and any of them may be complete or partial. Tumours in this situation are apt to implicate the cranial nerves. Optic neuritis is quite constant, and appears early. Localized or general convulsions are rare.

The peculiar symptoms pointing to tumours of the crura cerebri are nystagmus, strabismus, and loss of pupillary reflex, sometimes with general

muscular inco-ordination, and a staggering gait. There is usually third-nerve paralysis on the side of the tumour, and on the side opposite to the hemiplegia with which it is often associated. This variety of crossed paralysis is quite diagnostic. The symptoms of third-nerve paralysis are external strabismus, dilatation of the pupil, and ptosis. In these cases optic neuritis appears early. There may be a complicating hydrocephalus. While hemiplegia is commonly present with large tumours, it may be absent with small ones, or may appear later than paralysis of the third nerve.

Tumours of the pons are quite common. The diagnostic symptoms consist in crossed paralysis, the cranial-nerve symptoms being on the side of the tumour, and the general motor and sensory symptoms on the opposite side. When the seat is the upper half of the pons, the third and fifth nerves are apt to be implicated, giving rise to ptosis, dilatation of the pupils, external strabismus, trophic disturbances such as ulceration of the cornea, and neuralgic pain in the face. Tumours in the lower half of the pons involve the sixth, seventh, and eighth nerves, causing internal strabismus, contracted pupils, facial paralysis, sometimes deafness, and auditory vertigo. Other symptoms associated with tumours of the pons are headache, vomiting, and optic neuritis; convulsions being rare.

Tumours of the medulla are recognised by the involvement of the glossopharyngeal, pneumogastric, spinal accessory, and hypoglossal nerves. There are difficulty of deglutition, irregular respiration, irregular pulse, and vaso-motor disturbances, such as flushing of the face and perspiration. There may be projectile vomiting, polyuria or glycosuria, opisthotonus, difficulty in articulation or in sucking, and in protrusion of the tongue. When large, these tumours may produce symptoms of pressure upon the motor or sensory tracts,—paralysis, partial anæsthesia, with rigidity and exaggerated reflexes.

Tumours of the cerebellum are especially important, this being the most frequent location in childhood. When only one hemisphere is affected there may be no local symptoms. Tumours involving the middle lobe, or those large enough to produce pressure upon the middle lobe, give rise to vertigo and cerebellar ataxia. Vertigo is especially frequent; it may occur with headache. Cerebellar ataxia is different from the ataxia due to a spinal-cord lesion, and strikingly resembles that of intoxication. It may increase until the patient is unable to walk, although there is no loss of muscular power. Vomiting is a frequent symptom, as are also optic neuritis, and headache which is usually occipital. When there is secondary hydrocephalus, as is not uncommon, mental symptoms are present, and there may be enlargement of the head. Opisthotonus is occasionally seen, but general convulsions are rare.

Diagnosis.—The size of the tumour is to be determined mainly by the general symptoms, special attention being given to the order of their development. A diagnosis as to the nature of the tumour is really not of

much importance; but some information upon this point may be gained from the consideration of its etiology, the rapidity of its growth, and the age of the patient. Cerebral tumour may be confounded with abscess, tuberculous meningitis, chronic basilar meningitis, and chronic hydrocephalus. The symptoms distinguishing tumour from abscess are the following: Tumour may occur at any age; without definite etiology, excepting when tuberculous; the progress is steady, but generally slow, new symptoms being continually added; headache is more constant and more severe; optic neuritis more frequent; cranial nerves more often involved; mental disturbances more marked; focal symptoms are often definite; fever is absent; duration, six months to two years. As compared with the above, abscess is not so frequent, being especially rare in infancy; there is a definite history of traumatism or ear disease; progress more irregular; symptoms often intermittent; headache less severe; mental symptoms less marked; optic neuritis and involvement of the cranial nerves less frequent; focal symptoms usually indefinite; localized tenderness over the scalp more constant; fever present except in the latent period; the most frequent complication is acute meningitis.

Cases of tuberculous meningitis which may be confounded with tumour are those of slow course sometimes seen in older children. The difficulty in diagnosis is increased by the frequent association of tuberculous tumours with tuberculous meningitis. The main points of difference are that in tumour the symptoms are more localized and the course generally much slower. Almost every individual symptom, however, may be present in the two conditions.

Chronic basilar meningitis may produce symptoms almost identical with those of tumour in the posterior fossa. It is, however, confined to infancy, and is frequently syphilitic. Hydrocephalus and opisthotonus are much more marked than are usually seen with tumour.

Chronic hydrocephalus may resemble tumour; this occurs so frequently as a lesion secondary to tumour that the question often arises whether there is only hydrocephalus, or there is in addition a tumour. Primary hydrocephalus is usually congenital, and the symptoms appear during the first year. It commonly attains to a greater degree than is seen in secondary hydrocephalus; but the symptoms in the two forms may be identical.

Prognosis.—The prognosis in cerebral tumour is absolutely bad; except in syphilitic cases, which are among the rarest forms seen in childhood, there is no prospect of recovery, and but little of improvement. The symptoms usually progress steadily from bad to worse, and more rapidly in children than in adults. Death occurs from exhaustion, coma, convulsions, or from respiratory failure, sometimes suddenly from unknown causes.

Treatment.—If there is any reason to suspect syphilis, the iodide of potassium should be given in large doses and continued for a long period;

the effect of this drug even in tumours not syphilitic is sometimes beneficial. Starr refers to a case in which symptoms of six months' duration, including optic neuritis, entirely disappeared under the use of mercury and the iodide. The tumour was supposed to be gumma, but an autopsy obtained six months later showed it to be a sarcomatous cyst. For a discussion upon the surgical aspect of the treatment of brain tumours, the reader is referred to Starr's work on Brain Surgery.

HYDROCEPHALUS.

Hydrocephalus or "water on the brain," consists in an accumulation of serum in the cranial cavity. This may be between the dura mater and the pia (external hydrocephalus) or in the ventricles of the brain (internal hydrocephalus). The former is secondary and is quite rare, while the latter is not uncommon. Hydrocephalus may be acute or chronic.

Acute Hydrocephalus is secondary to basilar meningitis, which is usually of tuberculous origin. The terms tuberculous meningitis and acute hydrocephalus are sometimes used synonymously. A moderate distention of the ventricles is frequent in all varieties of acute meningitis. The amount of fluid in acute hydrocephalus is not great, there being rarely more than three or four ounces present.

Chronic External Hydrocephalus is extremely rare, and is probably always a secondary lesion. It is found with certain congenital malformations and with atrophy of the brain, and it may follow meningeal hæmorrhage or pachymeningitis. On incising the dura mater a few ounces, or sometimes even a pint, of serum may escape. The convolutions are somewhat flattened, and may be greatly atrophied. Other lesions are found either in the brain or in the dura mater. There may be some degree of internal hydrocephalus associated. External hydrocephalus may cause enlargement of the head and separation of the sutures, and in fact most of the symptoms of the internal variety; but usually it is not severe enough to give rise to any decided symptoms. It is so rare that it need not be considered at length.

CHRONIC INTERNAL HYDROCEPHALUS.

This is the important variety, and when no qualifying term is mentioned this is the form of hydrocephalus which is always understood.

Etiology.—This occurs both as a primary and a secondary condition. When secondary it is usually associated with tumours of the base of the brain or with chronic basilar meningitis, either simple or tuberculous. It is in these cases a mechanical condition caused by pressure which obliterates the openings from the lateral ventricles into the fourth ventricle, or the foramen of Magendie.

The causes of primary hydrocephalus are as yet very little understood. In a large proportion of the cases the disease is congenital, generally

beginning in the latter months of intra-uterine life. Some of these cases are clearly syphilitic. D'Astros * has collected nine cases and added three others, in which hydrocephalus was associated with lesions undoubtedly syphilitic. When due to syphilis, the disease may at the same time be congenital. Rickets and hydrocephalus are occasionally associated, but so infrequently as to make a definite etiological connection between them very doubtful. The rachitic head has been so often mistaken for hydrocephalus that an erroneous notion has arisen as to the frequent association of these two diseases. This point will be referred to more fully under diagnosis. Chronic hydrocephalus is often attributed to tuberculosis, but here again the connection is a very doubtful one. Heredity is a factor of some importance; numerous instances are on record where two children in the same family have been affected. Hydrocephalus not infrequently develops after successful operations upon spina bifida or encephalocele.

Lesions.—The difference between the primary and secondary cases is chiefly one of degree. The amount of fluid in secondary cases is rarely more than three or four ounces. In primary cases it is usually from half a pint to one pint, but it may be very great. In one of my own cases there was removed from the head of a child, who died at four months, five pints of fluid. Larger quantities than this have been reported, but not at so early an age. In composition this resembles the cerebro-spinal fluid. An examination in one of my cases showed it to be a clear, translucent fluid, slightly alkaline in reaction, specific gravity 1005, containing sodium and potassium chlorides, alkaline phosphates, and a trace of albumin. In some specimens sugar is found. In cases of inflammatory origin the amount of albumin is generally larger, and the fluid may be slightly turbid. The effusion may become purulent from accidental infection resulting from operation, from rupture, or, as in one of my cases, from infection through the sac of a spina bifida with which it was complicated, the process extending to the brain through the central canal of the cord.

The changes in the brain result from the gradual accumulation of fluid in the ventricles. The septum lucidum is usually broken down, and all the avenues of communication between the ventricular cavities are greatly enlarged. The continuous distention results in a gradual thinning of the brain substance which forms the ventricular walls; often these are found only one fourth of an inch in thickness, or even less than this, the cortex being a mere shell (Fig. 117). In one of my autopsies the ependyma of the ventricle and the pia mater were in places actually in contact, all of the brain tissue having been absorbed; the brain resembled a large double cyst. In a case of Peterson's, with the exception of a small portion of one temporo-sphenoidal lobe, all

* Revue Mensuelle des Maladies de l'Enfance. ix. 481, 543.

of both hemispheres had disappeared, the cerebellum and basal ganglia alone being intact. The brain is always anæmic, and the gray and white substance may be indistinguishable. The changes are largely mechanical, the microscope showing, in my case just referred to, only granular matter and round nuclei evidently from broken-down nerve cells. In less severe cases the changes may be slight. It is, however, always surprising to see the amount of compression which the cortex will tolerate without interference with its functions, provided the pressure comes gradually. The ependyma may be normal, but it is usually somewhat thickened and pale, sometimes granular, and may be infiltrated with new cells. When infection takes place an acute ependymitis may be set up. Chronic inflammation of the ependyma is thought to be the essential lesion in many of the primary cases, whether of simple or syphilitic origin.

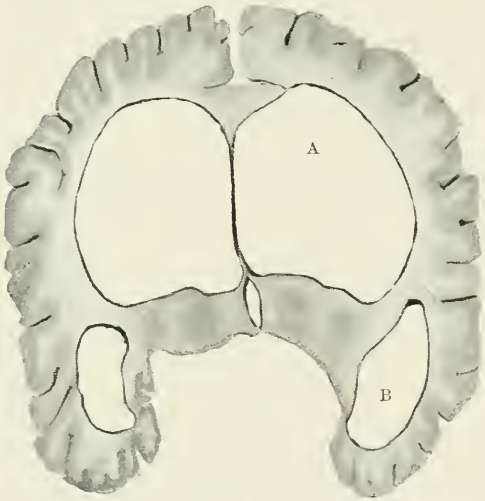

Fig. 117.—Vertical transverse section of a brain in congenital hydrocephalus, from a child who died at the age of three weeks. *A*, distended lateral ventricle; *B*, its descending horn.

The bones of the skull are markedly affected; the sutures at the vault are widely separated, and sometimes even those at the base. After the removal of the fluid the head collapses, giving an appearance which has been well likened to a bag of bones. It should not be forgotten, however, that hydrocephalus may coexist with premature ossification, in which case the head may be small. In the cases which recover, the wide gaps in the skull may be closed by the development of wormian bones; but ossification is often not complete until the fifth or sixth year.

The most frequent lesion associated with congenital hydrocephalus is spina bifida, in which cases there may also be a patency of the central canal of the spinal cord; more rarely meningocele or encephalocele are met with. Sometimes there are deformities in other parts of the body, such as club-foot or hare-lip.

Symptoms.—Hydrocephalus may exist with a small head. In this condition there is usually premature ossification of the cranial bones. Four such cases have come under my notice, one child having lived to be fourteen months old. These children are usually idiotic, and die at an early age, often from convulsions. In such cases other malformations of the brain are frequently associated.

Hydrocephalus, with the exceptions mentioned, is recognised by the increased size of the head. In order to estimate the amount of enlargement, it must be remembered that at birth the circumference of the normal head is about 14 inches, and at one year from 18 to 19 inches. The degree of enlargement in hydrocephalus may be very great. In one of my cases, the head at four months measured 24½ inches. In another at ten and a half months, 26¾ inches (Fig. 118). Steiner has reported a re-

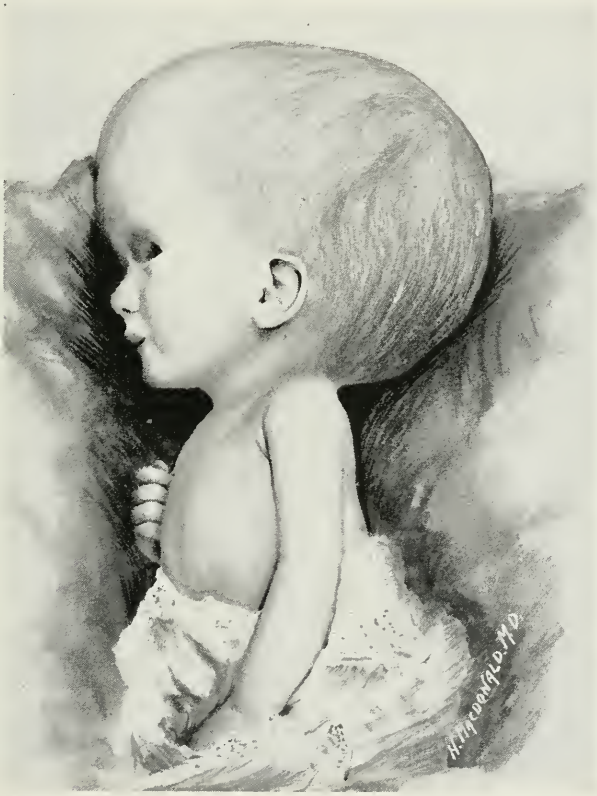

Fig. 118.—Chronic hydrocephalus of a severe type; head of a globular shape; child, ten and a half months old.

markable case in which the head at eight months measured 32¾ inches. When the enlargement of the head is not great the diagnosis is not so easy. Hydrocephalic enlargement is commonly symmetrical and in all directions. The head is sometimes globular in outline (Fig. 118) and sometimes pyramidal (Fig. 119). The forehead is exceedingly high and projecting, and there is a prominence at the root of the nose seen in no other form of enlargement. The sutures may be separated from half an inch to two or three inches; the fontanel is very large, tense, and bulging;

the veins of the scalp are enlarged and prominent. In marked cases fluctuation may be readily obtained, and the head may even be distinctly translucent.

In the acquired form all these symptoms are less marked, and if ossification of the skull has taken place it is often impossible to discover any increase in size. The rate of growth of the head varies much in different cases, and it is the surest measure of the progress of the case. The increase in circumference is usually from one to three inches a month.

The primary cases are for the most part of congenital origin, and the child may die *in utero*. At other times the process may have advanced so

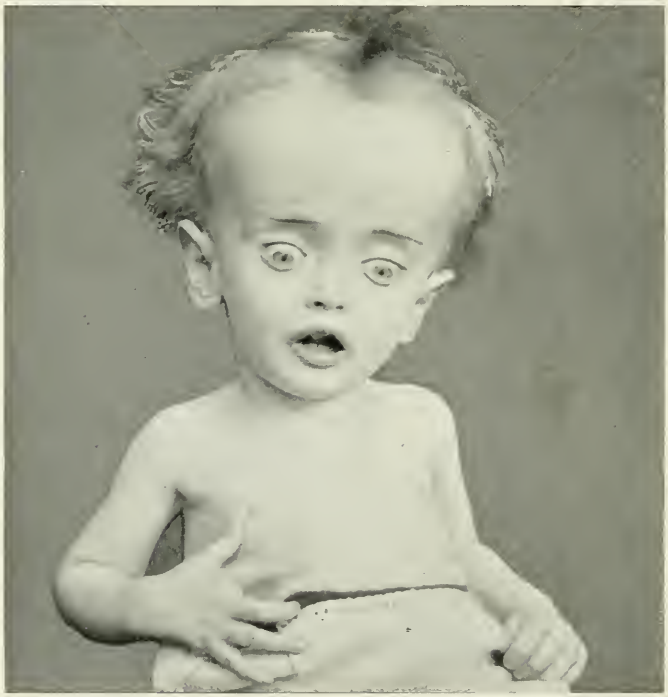

Fig. 119.—Chronic hydrocephalus of average severity; head of pyramidal shape; showing characteristic expression of the eyes.

far before birth that puncture of the head is necessary before delivery is possible. In perhaps the majority of cases no symptoms are observed at birth, or the head is only slightly larger than normal. Usually nothing is noticed until the child is two or three months old, when it is discovered that the head is increasing in size at an abnormal rate. If the progress is rapid, other symptoms are soon evident: the infant can not hold up its head; it is lethargic, and all its perceptions are dulled, sight and hearing included; there may be a general flaccid condition of all the

muscles of the extremities due to a slight general paresis, but more often there is rigidity, which is usually most marked in the legs, but sometimes in the arms; the hands are often clenched, with the thumbs adducted; the reflexes are exaggerated; the pupils are generally contracted and equal, though they may be dilated; nystagmus and convergent strabismus are often present. Convulsions may occur from time to time, or may be deferred until near the close of the disease. As the head enlarges the body usually wastes, and the disproportion between the two may seem greater than it really is.

Such congenital cases rarely see the end of the first year, and are often fatal during the first six months. The causes of death are marasmus, convulsions, and intercurrent disease, rarely rupture of the head.

In the cases which develop more slowly, the symptoms are quite different. The head may not attain at eighteen months the size reached in the other cases at the third or fourth month. The surprising thing about many of these cases is that the distinctly cerebral symptoms are so few. Where the pressure develops gradually, the brain seems able to tolerate an almost indefinite amount of it. The more readily the bones of the skull yield to pressure the fewer are the nervous symptoms; hence, other things being equal, they are less marked where the disease begins before the sutures are firmly ossified than in the later cases. A comparatively small amount of effusion may cause very marked symptoms in a child two or three years old, while a much larger amount in an infant of a year, may produce much less disturbance. It is for this reason that secondary hydrocephalus causes such striking symptoms, although the accumulation of fluid is small.

Whether the progress of these cases is slow or rapid, the development of the children is greatly retarded. Many are not able to support the head until two or three years old; frequently they do not walk until five or six years old. The special senses are generally not noticeably affected, but intelligence in most cases is interfered with,—in some only slightly, in others very markedly, while some are idiotic. Contractions of the extremities are occasionally seen, but usually more of the hands than the legs. Sensation is not often affected. The course is a very chronic one. From time to time there are exacerbations of the symptoms, and even intercurrent meningitis may be excited.

Prognosis.—Recovery is rare. It is quite exceptional that a hydrocephalic child reaches the age of seven years. In some cases the process goes on up to a certain age and then ceases spontaneously, and the child may go through life with a head very much larger than normal, usually with a mental condition somewhat impaired. Retrogression of the symptoms is, however, never to be looked for.

Diagnosis.—The most important symptom is the enlargement of the head, and this can only be arrived at by careful measurement and com-

parison with the normal size. The rapidity of growth is quite as important for diagnosis as the fact of enlargement. If the head grows more than an inch a month there can be little doubt. Hydrocephalus without enlargement of the head can not be diagnosticated. The enlargement most frequently confounded with hydrocephalus is that which occurs in rickets. In the latter disease it is almost invariably irregular; there are prominences over the two frontal eminences and over the parietal bones, often with furrows between them; the size of the head is chiefly due to thickening of the bones of the skull; the marked prominence of the forehead is not seen, and the increase in bi-parietal diameter is not present; furthermore, there are other signs of rickets.

Treatment.—Almost every sort of local treatment has been adopted for hydrocephalus, including incision, aspiration, cranial puncture with the trocar, lumbar puncture, blisters, strapping, and counter-irritation. Up to the present time there does not exist sufficient evidence to show that any one of these means is curative. If aspiration is done, the fluid reaccumulates very quickly, while incision or cranial puncture is almost certain to be followed by meningitis. If there is any reasonable suspicion of syphilis, mercurial inunctions to the head should be employed, and even in other cases a few favourable results have been reported. Convulsions and other functional symptoms are to be treated upon general principles, as they arise. At the present time I believe it is better to refrain from all operative measures unless rupture seems likely to occur.

INFANTILE CEREBRAL PARALYSIS.

Synonyms: Spastic diplegia, paraplegia, or hemiplegia.

Under the term cerebral paralysis are included several groups of cases with causes quite dissimilar, but having certain definite clinical features in common. While the symptomatology is quite clear, there are many questions relating to the pathology that are not yet fully settled, although much has been added to our knowledge within the last few years. Paralysis depending upon cerebral tumour, abscess, or hydrocephalus is not included in this chapter.

The cases of cerebral paralysis may be divided into three groups, according as the paralysis depends upon conditions existing prior to birth, upon those connected with birth, or upon those of subsequent development.

I. **Paralysis of Intra-Uterine Origin.**—This is the least frequent condition. In such cases there is some congenital defect in the brain, due sometimes to arrested development, at others to such intra-uterine lesions as hæmorrhage or thrombosis. There may be porencephalus, or cysts extending deeply into the substance of the brain, sometimes communicating

with the ventricles. The origin of this condition is for the most part unknown. In rare cases the paralysis is due to cortical agenesis,* a condition in which the brain may seem normal to the naked eye, but the microscope shows a complete arrest in the development of the cells of the cortex, usually affecting both hemispheres. In still other cases there are found gross defects in development in the motor centres of the cortex. Such a lesion is shown in Fig. 124, page 751. Cases in which there is conclusive evidence of intra-uterine hæmorrhage are very rare.

Symptoms.—In most of the paralyses due to intra-uterine lesions, loss of power is only one of the symptoms, and usually not the most prominent. It is rare that there is not some mental impairment, and usually idiocy is present. The type of paralysis is nearly always diplegic or paraplegic. Where this is due to arrested cortical development, a general flaccidity of the muscles may be seen instead of the rigidity so characteristic of the other forms of cerebral paralysis.

II. **Birth-Paralysis.**—Cerebral birth-paralysis is due in nearly all cases to meningeal hæmorrhage. The primary lesions and the early symptoms have already been described (page 105) in connection with the Diseases of the Newly Born. The secondary lesions present considerable variety. There may be found (1) meningo-encephalitis, (2) atrophy and sclerosis of the cortex, (3) cysts upon the surface, (4) secondary degenerations in the spinal cord.

1. Meningo-encephalitis.—This lesion is often quite diffuse. There is thickening of the pia mater, and it is usually adherent to the brain substance. The cortex is involved to a variable degree, depending somewhat upon the time which elapses between the initial lesion and the autopsy. The following were the microscopical changes found by Sachs † in the brain of a child in my wards at the Babies' Hospital, who died at the age of one year of measles: The lesions were found everywhere in the cortex. The pia was universally adherent, and showed general cellular infiltration; its blood-vessels showed marked cellular proliferation, and the veins in the sub-pial space were dilated and filled with blood. In the pia dipping in between the convolutions similar changes were present. In the cortex few if any normal pyramidal cells were found, but in the outer layers were an enormous number of small glia cells. Many of the blood-vessels showed a cell-proliferation of their walls. There was also

* For fuller description, see Sachs's Nervous Diseases of Children, 1895, p. 601.

† The clinical features of this case are quite as interesting as the pathological findings. The child was a first-born, delivered after a dry labour of forty-eight hours. It was asphyxiated, and from the first days of its life it had attacks of convulsions, usually repeated many times a day. During one of these convulsions the photograph from which Fig. 122 was made, was taken by Dr. Peterson. The child had the symptoms of typical spastic paraplegia—the arms being, however, slightly involved—retarded mental development, and convergent strabismus.

a degeneration in the pyramidal tracts of the anterior columns of the cord.

2. Atrophy and sclerosis.—These changes vary much in extent and degree. There may be only a circumscribed area in which the convolutions are small, firmer than usual, and covered with an adherent pia, or there may be an atrophy so extensive as to involve a large part of one hemisphere (Figs. 120 and 121), or sometimes of both hemispheres. Usually the lesion is somewhat diffuse over the convexity of both sides, and much more frequently of the anterior than of the posterior half of the brain.

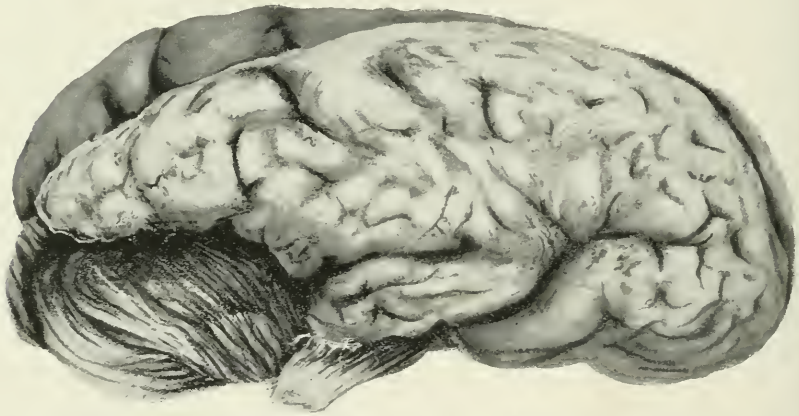

Fig. 120.—Extensive atrophy and sclerosis of the right hemisphere, from an infant seven and a half months old; probably the result of a meningeal hæmorrhage at birth (lateral view).

History.—Twelve hours after birth was seized with general convulsions, which continued for three days. No other symptoms noticed till one month before death, when weakness of left arm was observed. Never held head erect. Was plump and well nourished; died from erysipelas.

Autopsy.—Pia not adherent; a large cyst occupied the region of the occipital and posterior part of the parietal lobes, showing in its floor discolouration and pigmentation, evidently from an old hæmorrhage. Right optic nerve, tract, and crus much smaller than the left.

Where a depression of the brain exists the space is filled with cerebro-spinal fluid, and in many cases there is a deformity of the skull.

3. Cysts upon the surface may occur alone or in connection with the lesions just mentioned. These are usually small, about the size of a walnut, but they may cover a large part of a hemisphere. Such large cysts are sometimes classed as cases of external hydrocephalus.

4. Secondary degenerations of the internal capsule and the lateral columns of the cord are found in most of the cases associated with extensive atrophy and sclerosis, and in many of those in which only meningo-encephalitis is present.

Symptoms.—The type of paralysis will of course depend upon the extent and position of the original lesion. A diffuse lesion is followed by diplegia; one not quite so extensive by paraplegia; one affecting one side only by hemiplegia, or even monoplegia, though this is very rare. The

relative frequency of the different forms will vary according to the age at which the patients come under observation. Thus in the statistics of Sachs and Peterson,* there were twenty-seven cases of diplegia or paraplegia, and twenty-two of hemiplegia. These cases were drawn from miscellaneous sources, chiefly from a general neurological clinic. According to my own observations, which have been chiefly upon infants,

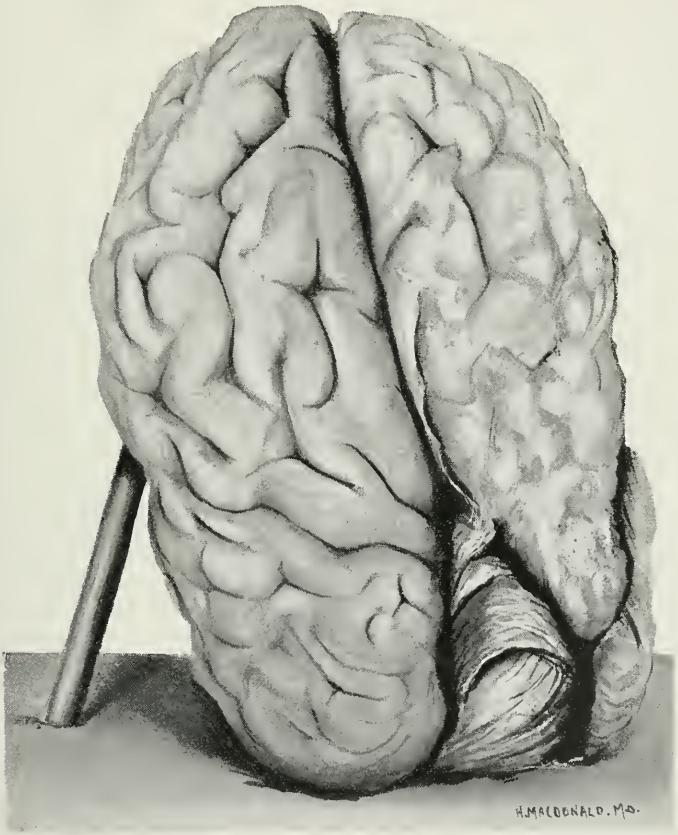

Fig. 121.—Atrophy of right hemisphere; same case as Fig. 120; superior view.

the cases of diplegia and paraplegia have outnumbered those of hemiplegia more than four to one. My belief is that the great majority of the congenital cases, or those due to hæmorrhage occurring at birth, are diplegias or paraplegias, and that very many of them succumb during the first two years, and never come under the observation of the neurologist; however, the cases of hemiplegia, because of the less serious lesion, live much longer, and hence are more likely to be seen by the specialist. Diplegia

* Journal of Nervous and Mental Disease, May, 1890.

and paraplegia will therefore be considered as the characteristic types of cerebral birth-palsy, as the cases of hemiplegia do not differ from those due to later causes—i. e., the acquired form.

In the most severe cases that survive the symptoms of the early days of life (page 107) there remains some rigidity of the extremities, chiefly of the legs, which is constant or intermittent, slight or well marked. There is often spasm of the muscles of the neck and trunk, giving rise to opisthotonus. In many cases there are frequent attacks of convulsions (Fig. 122). The general physical development of the child is often interfered with, so that it remains small and delicate, and perhaps dies of some acute disease in early infancy, never having been able to sit erect, or even support its head. In other cases the general nutrition is not affected,

Fig. 122.—Convulsions in spastic paraplegia: from a photograph by Dr. Frederick Peterson during an attack. (History on page 743.)

and the infants may be plump and well nourished. Such children may live indefinitely. There is always some degree of mental impairment; it may be so slight as not to be noticeable until the child is old enough to talk, and sometimes not until the age of four or five years; or the child may be idiotic. Speech is not only delayed, but is very imperfect. Hearing is frequently affected, but sight rarely. Often these children are not able to walk alone until they are four or five years old, and then with a peculiar cross-legged gait, owing to spasm of the adductors of the thighs. This may be so great as to entirely prevent walking, and while sitting or lying the thighs may cross each other. All the reflexes are greatly exaggerated. In one child under my observation the pharyngeal reflex was so much increased that swallowing of solid food was impossible, owing to spasm of the muscles. Alcoholic stimulants and medicines that were at

all pungent were taken only with the greatest difficulty. In some of the worst cases walking is impossible, owing to the shortened tendons and the contractures which have occurred in the muscles. The arms are in nearly all cases much less affected than the legs, and in about half the number, according to the observations of Sachs, they are not involved at all. The condition is not incompatible with long life.

In the mild cases it not infrequently happens that the early symptoms are so slight as to be overlooked, and nothing excites suspicion until the infant is six or eight months old. There is then discovered an unmistakable muscular weakness, as the child can not sit up, or even hold up the head when the trunk is supported. In most of the cases there is observed before this time a tendency to stiffen the body and to throw it backward, owing to spasm of the cervical or spinal muscles. This may be slight, or it may be very marked. The muscular weakness is not infrequently mistaken for rickets, and is sometimes regarded as simple backwardness. A closer examination usually discloses the presence of some rigidity of the extremities, particularly of the legs, and exaggeration of the knee-jerk. As the child grows older the other symptoms of late or imperfect development become more and more evident.

There are changes in the shape of the skull, this being usually smaller than normal in all its diameters, or there may be asymmetry. There is an arrest of development in the paralyzed limbs. These are both smaller and shorter than normal. There is marked muscular atrophy. In many cases abnormal movements are seen, which may be of an irregular choreic type, or they may be athetoid. According to various statistics, epilepsy develops in from 33 to 50 per cent of all the patients affected.

III. **Acute Acquired Paralysis.**—This is usually of the hemiplegic type, although diplegia and paraplegia may in rare instances be met with. This group includes cases developing at any time after birth, but the great majority of those seen in childhood, begin before the fifth year.

Etiology.—The etiology of many of these cases is very obscure. The paralysis sometimes follows traumatism. It is occasionally seen in the course of scarlet fever, measles, diphtheria, variola, and pneumonia. Much more frequently than with any of these diseases it occurs during pertussis, being usually the outcome of a severe paroxysm. Aside from the traumatic cases and those occurring with pertussis (and these include but a small proportion), the real cause is for the most part unknown. The frequency with which these cases are ushered in with convulsions has led many to assign this as the cause of the paralysis. It is more probable that the convulsions are the result than the cause of the lesion producing the paralysis.

Lesions.—The lesions of acute cerebral palsy may be grouped under three heads: (1) those of the blood-vessels; (2) those of the membranes; (3) those of the brain substance.

1. Lesions of the blood-vessels.—There may be either hæmorrhage, embolism, or thrombosis. Hæmorrhage is by far the most important. It is usually meningeal, very rarely cerebral. It occurs more frequently at the convexity than at the base, and is often quite diffuse. Meningeal hæmorrhage may result from pachymeningitis. I have elsewhere stated my conviction that this is more frequent than is generally supposed. It may be due to traumatism, where it is also from the dura mater; or from the acute hyperæmia accompanying paroxysms of pertussis, where it may be from the dura or the pia; or it may be secondary to thrombosis of the superior longitudinal sinus. The association of hæmorrhage with sinus-thrombosis is not very infrequent. It was found in one of my autopsies upon a patient who died of pneumonia. The bleeding in these cases is usually from the pia. Cerebral hæmorrhage is extremely rare, but it occurs even in infants; I once saw it in one only two months old.

Embolism is rarely found unless associated with acute rheumatic endocarditis, and then usually in children who are over seven years old. As in adults, the usual seat of the embolus is a branch of the middle cerebral artery. It may be single or multiple. Thrombosis has been met with in a small number of cases, but it is extremely rare.

2. Lesions of the membranes.—These are generally the result of old cerebro-spinal meningitis; sometimes they may be of syphilitic origin. In both, however, the process is rarely confined to the membranes; it is a meningo-encephalitis.

3. Lesions of the brain substance.—Atrophy and sclerosis are terminal conditions found in a large number of the autopsies made upon cases where the paralysis has been of long standing. They vary in severity and extent, and are followed by secondary degeneration in the cord, as in cases of birth paralysis. There may be the same development of cysts of the pia mater, or an accumulation of fluid in the arachnoid cavity, these taking the place of the atrophied convolutions. What the primary lesion is in these cases is still a matter of debate. Strümpell believes many of them to be due to an acute poliencephalitis, analogous to acute poliomyelitis. Cases are not infrequently seen clinically, which this pathology seems to explain very satisfactorily. However, there is as yet lacking sufficient anatomical evidence to establish this view.

In this connection may be mentioned a case of acute paralysis in which no lesion was found. In the spring of 1894, there was admitted to my service in the Babies' Hospital, an infant with pneumonia, who had developed, a few days before, typical right hemiplegia. The pneumonia antedated the paralysis by several days. The latter came on suddenly, with convulsions, and involved the face, arm, and leg. The arm and leg appeared to be completely paralyzed, but in the face the paralysis was incomplete. The paralysis had begun to improve somewhat at the time of the child's death, which occurred a little over a week after its onset.

At the autopsy no gross lesion could be discovered. A careful microscopical examination was made by two expert pathologists, Drs. C. A. Herter and J. S. Thacher, who could find no explanation of the paralysis. Nothing abnormal was found except "a slight increase of small spheroidal cells about some of the meningeal and cortical vessels of the motor area. The frontal and occipital lobes were normal."

Symptoms.—While diplegia and paraplegia are occasionally seen, the great majority of cases of acquired cerebral palsy are of the hemiplegic variety. When diplegia and paraplegia occur, it is usually in early infancy, and their symptoms and course differ in no wise from the birth palsies. We may therefore regard hemiplegia as the chief manifestation of acquired cerebral palsy.

The onset of the paralysis is almost invariably sudden, with convulsions, which are usually repeated, and in severe cases followed by loss of consciousness. In the secondary cases these are generally the only symptoms. In one of my cases the patient went to bed apparently well, and awoke in the morning with hemiplegia. Such an onset, however, is very exceptional. When the paralysis is apparently primary, fever is usually present, and in addition to the convulsions there may be vomiting, delirium, and other symptoms, strongly suggestive of an acute inflammatory process in the brain, which continue for a variable time, usually two or three days, before paralysis is seen. The temperature in most cases is from 100° to 102° F., and the rise of temperature follows more frequently than precedes the convulsions. After the child recovers consciousness, and sometimes before this, the paralysis is discovered. If there is a very extensive lesion there may be diplegia, deep coma, and death, but this is very infrequent. Usually the lesion is more limited, and the symptoms are those of typical hemiplegia. It is rare that the face is much involved, and often it escapes altogether. The paralysis of the arm and leg is at first complete, but may improve very rapidly in the course of a few days. Disturbances of sensation are usually of a transient character. After a variable period, from one to several weeks, the patient begins to use the paralyzed extremities, the arm recovering more slowly than the leg, as in adult hemiplegia. The convulsions may be repeated for the first day or two, but prolonged or continuous convulsions are rare. With lesions of the left side of the brain, speech may be affected, and not infrequently in young children when the lesion is upon the right side. The reflexes are increased upon the affected side, and slight ankle-clonus may be present.

In the course of a few weeks the child may be able to walk, dragging the affected leg; the recovery in the leg is sometimes complete, but in most cases a slight halt in the gait remains. The arm usually recovers more slowly than the leg, and contractures are likely to develop after a variable time, generally two or three years. In Fig. 123 is shown a frequent deformity of the upper extremity. Contractures of the leg lead to various

forms of talipes, generally equinus, from shortening of the tendo-Achillis. Sometimes the arm or the leg recovers so perfectly that the case may be regarded as one of monoplegia. In old cases the paralyzed limbs are atrophied; there is more or less rigidity, and the spastic condition may be quite marked. I have seen this limited to a single group of muscles in the leg. Aphasia is common in right hemiplegias, and it is not very rare in those of the left side, because infants appear to use both sides of the brain with nearly equal facility.

Fig. 123.—Deformity of left hand the result of contractures following an attack of hemiplegia four years before; child seven years old.

The mental condition of these children is usually normal, in striking contrast with the cases of congenital diplegia. The earlier the paralysis occurs the more likely are mental symptoms to be present, since we have here not only the direct effect of the lesion, but an arrested development of some part of the brain. Epilepsy is not an uncommon sequel; it may be of the Jacksonian type, or there may be attacks of general convulsions. In other cases there are post-hemiplegic movements of a choreic or athetoid character, or irregular inco-ordinate movements.

Prognosis of Infantile Cerebral Paralysis.—In diplegia and paraplegia the outlook is always unfavourable. A very large number of these cases which are due either to intra-uterine or birth lesions, never reach the third year, but die in infancy of marasmus or acute intercurrent disease. Those who survive usually show serious mental defects, and many are practically helpless on account of the extreme spastic condition of the muscles of the extremities.

In hemiplegia the prognosis is much more favourable. In most of these cases the paralysis is of the acute acquired variety, and the later the period of onset, the less likely is the brain to be seriously damaged. In some of these patients complete recovery takes place; in others the residual paralysis is so slight as to be easily overlooked except on careful examination, the occurrence of epilepsy being perhaps the first thing which leads one to suspect that a previous paralysis has existed. The great majority of children who have suffered from infantile cerebral palsy have some degree of permanent paralysis and usually some deformities from contractures,

the extent of both varying, of course, with the severity of the primary lesion. In all cases seen in young infants it is exceedingly difficult to give a prognosis in regard to future mental development. As a rule, the impairment is directly proportionate to the extent of the paralysis and its intensity; although in exceptional cases we find a good deal of mental disturbance with only moderate paralysis, and *vice versa*.

Diagnosis.—The diagnosis between the congenital and acquired forms of cerebral palsy is of no great practical importance, and it may be impossible; for the symptoms in congenital cases are often not sufficiently marked to attract attention until children are old enough to sit alone or to walk.

It may be quite difficult to distinguish cerebral paralysis from infantile spinal paralysis. The history of an acute onset, the atrophied limbs, the deformities, and the absence of sensory disturbances, may be found in both conditions. Spinal paralysis is, as a rule, monoplegic, and often affects but a single group of muscles. Cerebral paralysis is either diplegic or hemiplegic in character, and even though only a leg or an arm may seem to be affected, a critical examination will usually reveal the fact that the other limb of that side has also suffered. The presence of rigidity and exaggerated reflexes is quite as important evidence of this as loss of power. The electrical reactions, however, are conclusive; the reaction of degeneration is absent in cerebral paralysis, while it is present in spinal paralysis.

Simple as the differentiation may seem in most cases, the mistake is frequently made of confounding cerebral diplegia, particularly of the flaccid type, with rickets. But a careful history and a thorough examination will usually dispel all doubt (see pages 232, 233). Cases of acute acquired paralysis at the onset may be mistaken for acute meningitis, but early loss of consciousness, the early development of the paralysis, its permanent character, and the short duration of the acute symptoms, distinguish cases of hæmorrhage from those of meningitis; but when it follows traumatism, and when it occurs in the course of some other disease such as pneumonia or scarlet fever, it may be difficult or impossible to make a diagnosis between the two conditions.

Treatment.—The course and the result of cerebral paralysis depend upon the extent of the injury to the brain, its nature, and the age at which it is inflicted,—all these being conditions which are beyond the power of the physician to modify or control. The treatment of cerebral palsy is therefore extremely unsatisfactory. For the congenital cases practically nothing can be done, except for the deformities and complications. The acquired cases during the acute onset are to be managed like all other cases of acute cerebral congestion or inflammation,—absolute rest, ice to the head, and bromides. Electricity is never to be used in early cases, and little or nothing is to be expected from it in the late ones. Much can be accomplished in an educational way for the mental derangements re-

sulting from cerebral palsy; this, however, belongs more properly to the subject of idiocy.

An important part of the treatment relates to the deformities. Many of these may be prevented by the early use of orthopædic apparatus. Serious deformities in old cases may be greatly benefited by tenotomy or myotomy, followed by the application of suitable apparatus. In fact, very little can be done for these patients except by the orthopædic surgeon. Epilepsy is to be treated as in cases depending on other causes.

FEEBLE-MINDEDNESS, IDIOCY, IMBECILITY.

By these terms are designated the different forms of mental impairment, seen in children as a result either of arrested development or disease of the brain. They differ in degree rather than in kind, and may be associated with a variety of pathological conditions. Following somewhat the classification of Ireland, these cases may be grouped as follows:

1. Those depending upon the arrested development of the brain as a whole, or upon that of the frontal lobes. An excellent example of this class of cases is shown in Fig. 124. Another form is "agnesia corticalis" (page 741).

2. Those associated with hydrocephalus.

3. Those associated with microcephalus, with or without premature ossification of the cranial bones.

4. The paralytic cases,—including the varieties which occur in the different forms of cerebral paralysis, the greater part of which are due to meningeal hæmorrhage at the time of birth, and associated with spastic diplegia or paraplegia; a smaller number are associated with acquired palsy, which is most frequently due to meningeal hæmorrhage.

5. Those of inflammatory origin. They follow cerebro-spinal meningitis, and possibly also there may be added a group dependent upon poliencephalitis (Strümpell).

6. Those associated with epilepsy, in which the condition is a result of changes in the brain produced by the repetition of the epileptic seizures.

7. Sporadic cretinism (page 752).

Cases of mental impairment probably do not follow ordinary attacks of infantile convulsions or traumatism without some definite lesion of the brain, and hence have been included in some of the foregoing varieties.

In addition to the etiological factors belonging to the separate conditions described, there are to be considered influences of heredity, nervous diseases in the family, alcoholism, syphilis, and some other inherited vices of constitution in the parents, and intermarriage among blood relations.

Most cases of idiocy exhibit to a greater or less degree, the stigmata of degeneration (page 757). In an examination of five hundred and

seventeen idiots by Howe, there were found blindness in twenty-one; deafness in twelve; some defect of the nose or mouth, such as hare-lip, high

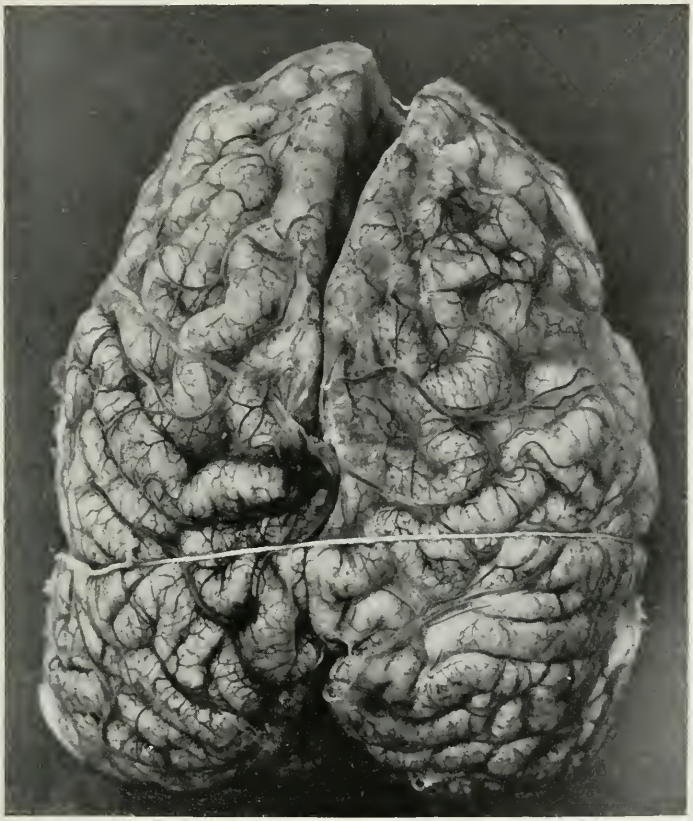

Fig. 124.—Arrested development of the frontal lobes of the brain, particularly of the right side, from an idiotic child twelve months old.*

palatal arch, or cleft palate, in twenty-three cases; and some deformity of the hands or feet in fifty-four cases; while in ninety-six there was paralysis of one or more limbs.†

* A microscopical examination by Dr. Martha Wollstein showed the cortex in the affected region to be only one-third the normal thickness; the cortical layers were ill-defined; there was a striking absence of the characteristic nerve cells, both the large and small pyramidal cells being few in number. There was no growth of connective tissue. The white substance was normal, as were also the dura and pia.

† For the symptoms of idiocy in detail, reference is made to works on diseases of the nervous system, especially to the Monograph of Langdon Down, and to the article by Brush in Keating's Cyclopædia, vol. iv, p. 1019, in which will be found references to recent medical literature upon the subject.

SPORADIC CRETINISM.

Synonyms: Cretinoid Idiocy; Myxœdematous Idiocy; Idiocy with Pachydermatous Cachexia.

Since the early description of this disease by Fagge, in 1871 and 1874, numerous cases have been published in England, on the continent of Europe, and in America, showing that the disease is not confined to any country. During the last six years, five cases have come under my own observation. While the disease is rare, cretins are much more common than was formerly supposed.

Etiology.—It is now well established that this condition depends either upon a congenital absence of the thyroid gland, or something which abolishes its functions. In Bramwell's series of forty-four cases, ten autopsies are reported; in nine of these no trace of the thyroid gland could be found, and in the tenth one lobe was the seat of a large tumour. The symptoms are practically identical with the myxœdema of adults which follows the removal of the thyroid gland. Regarding the causes which destroy the thyroid gland or abolish its functions little is as yet known. In most cases it is a congenital condition. In some instances it has followed acute disease. As a rule, only one case occurs in a family, the other members of which present nothing abnormal in mental or physical development.

Fig. 125.—A typical cretin, nine years old; height, 28¾ inches. (After Bramwell.)

Symptoms.—The symptoms of cretinism in most cases make their appearance during the first year, sometimes not until children are two or three years old, and occasionally none may be seen until the seventh or eighth year. The general appearance of the cretin is very striking, and so characteristic that when once seen the disease can hardly fail to be recognised (Figs. 125 and 126). The body is greatly dwarfed, and children of fifteen years are often only two and a half or three feet in height. All

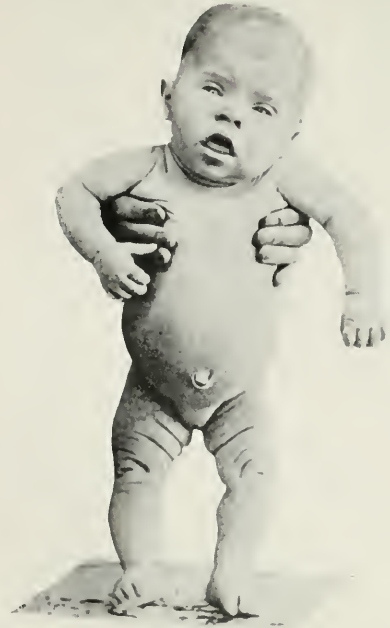

Fig. 126.

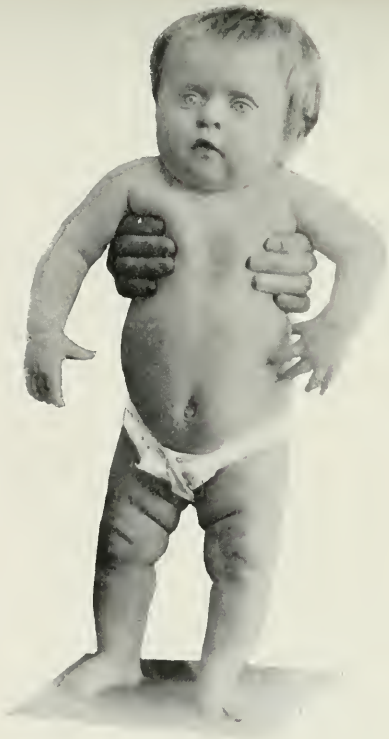

Fig. 127.

Fig. 128.

Dr. J. P. West's case of cretinism. Fig. 126, at seventeen months before treatment. Fig. 127, after six months' treatment with thyroid extract, having gained in weight 8 pounds, in height 4 inches, in circumference of head 2¾ inches. Fig. 128, after one year's treatment.

the extremities, the fingers and the toes, are short and stumpy. The subcutaneous tissue seems very thick and boggy, but does not pit upon pressure like ordinary œdema. The facies is extremely characteristic: The head seems large for the body, the fontanel is open until the eighth or tenth year, and it may not be closed even in adults; the forehead is low and the base of the nose is broad, so that the eyes are wide apart; the lips are thick, the mouth half open, and the tongue usually protrudes slightly; the cheeks are baggy, the hair coarse, straight, and generally light coloured. The teeth appear very late—in one of my cases none were present at two years—and are apt to decay early.

Fatty tumours are quite constant in older children, although they were wanting in two of my infantile cases. They are seen in the supra-clavicular region, just behind the sterno-mastoid muscle, sometimes in the axilla, or between the scapulæ, and sometimes in other parts of the body. In distribution they are apt to be symmetrical, and are usually about the size of a hen's egg. The neck is short and thick. In some cases there is a depression corresponding to the location of the thyroid gland. The chest is not deformed. The abdomen is large, pendulous, and resembles that of rickets. The skin is dry, perspiration scanty, and eczema is common. The voice is hoarse and rough. Patients often do not walk until they are five or six years old, and then they waddle in a clumsy way. All the movements of the body are slow and lethargic, and everything indicates a mental and physical torpor. The rectal temperature is usually subnormal. I had once an opportunity to observe an attack of acute bronchopneumonia in one of these cretins two years old. The symptoms and physical signs were typical, but during the greater part of the disease the rectal temperature fluctuated between 95° and 98·5° F. Only once was a temperature above 99° F. recorded. On account of their low temperature and torpid condition these patients are very sensitive to cold. The mental condition is always impaired, and they are usually idiotic. Speech is acquired late, and in some cases not at all. Cretins are dull, placid, and good-natured, rarely troublesome or excitable; and when fifteen or eighteen years old they appear like children of two or three years. There is an absence of development of the sexual organs, and almost invariably they suffer from chronic constipation.

Diagnosis.—The diagnosis is usually easy, although the early cases are sometimes miscalled rickets. The low temperature, the facial expression, the torpor, and the fatty tumours are enough to differentiate the two diseases.

Prognosis and Treatment.—There is no tendency to spontaneous improvement. Many of these cases die in childhood, but a few live to adult life. Until within the last few years they have been considered hopeless. The improvement which followed the use of the thyroid extract in cases of adult myxœdema has led to a trial of this

remedy in sporadic cretinism. A sufficient number of cases have now been recorded to establish the fact that the thyroid extract is a specific remedy for this disease. Peterson and Bailey * have collected forty cases treated in this manner. No case failed to improve when the extract was properly given. In twenty-five cases the improvement was very striking, and in several it was truly remarkable (Figs. 126, 127, 128). After a few months' treatment the entire appearance of the child is in most cases changed: The idiotic expression of the features is lost; the thickening of the skin and subcutaneous tissues disappears; there is a marked increase in weight, and in the growth of the whole body; muscular power is rapidly developed, so that many soon become able to walk; and progress is seen in dentition, and in some older girls in the establishment of menstruation. Intellectual progress is much slower than physical changes; however, nearly all the children become brighter and more intelligent, and a few learn to talk. In none of the cases so far reported has treatment been continued longer than eighteen months, so that it is as yet impossible to say whether improvement will continue indefinitely, and whether complete recovery is to be expected. From present knowledge the latter seems very improbable. In all cases the thyroid extract must be given indefinitely, for otherwise improvement ceases at once, and cases may even relapse. The earlier the treatment is begun the more marked is the improvement usually noticed.

The preparation most used in America is Parke, Davis & Co.'s desiccated extract, prepared from the thyroid gland of the sheep. Of this from one half to one grain is given twice a day. Some disturbances are often seen at the beginning of the treatment—perspiration, fretfulness, and sometimes a rise in temperature—but these soon pass off. In some cases a smaller dose must be used at first, and the increase made very gradually.

INSANITY.

Insanity is so special a subject, that all that will be attempted here will be to mention the most frequent varieties seen in early life, with the important etiological factors which operate at this period. For a full discussion of the subject the reader is referred to works upon insanity, and to Sachs, in whose book † will be found quite a full bibliography of this branch of the subject.

Insanity is distinguished from idiocy in that it affects a mind previously sound, however, the two conditions may be associated. Undoubted cases of mental disease have been observed before the seventh year, but

* Pædiatrics, May 1, 1896. See also Osler, American Journal of the Medical Sciences, November, 1893; and Bramwell's Monograph on Cretinism.

† Nervous Diseases of Children, New York, 1895. See also Mills, in American Text-Book of Diseases of Children, edited by Starr, Philadelphia, 1894.

they are extremely rare. From this time up to puberty, however, nearly all the varieties seen in adult life occasionally occur, but they are very infrequent even at this period. The form which insanity in childhood most frequently assumes is mania.

Etiology.—Insanity is sometimes seen as a sequel of one of the infectious diseases, more often typhoid fever than any other, although it may follow measles, scarlet fever, diphtheria, or variola. Another cause is masturbation, although its effect is much more frequently seen after puberty than before. Hereditary syphilis is sometimes the cause of dementia, which comes on about the fourth or fifth year, or even later. Alcoholism, epilepsy, insanity, or other nervous diseases in the parents are important causes. Prolonged or continuous mental strain, the result of overwork in school, is a cause of considerable importance, especially in girls about the time of puberty. As exciting causes may also be mentioned various reflex conditions, such as intestinal worms, phimosis, delay in the establishment of menstruation, and abnormal conditions of the nose and throat; these, however, can not have much influence except where the predisposition is a strong one. Insanity may be associated with or may follow hysteria, chorea, or epilepsy. It has sometimes followed injury to the brain, acute meningitis, and occasionally other forms of brain disease.

Symptoms.—Certain forms of insanity are practically never seen in children, such as paranoia or primary delusional insanity, acute dementia, paretic dementia, periodic or circular insanity, and cataleptic insanity.

Mania is one of the most frequent forms, and is the most common variety of post-febrile insanity. Its symptoms may be quite intense, but are usually of short duration, lasting but a few days or weeks. In rare cases it may continue for months, and it may even be permanent.

Melancholia is not uncommon. It is seen as a result of prolonged mental strain in school, it may be due to fear of punishment, and sometimes may follow masturbation. It is usually associated with some very marked disturbance of the general health. It shows itself, as in the adult, by fits of depression, self-mutilation, and even by suicidal tendencies.

Epileptic insanity may follow epilepsy in children who were previously mentally sound, where it may take the form of true epileptic dementia, or there may be attacks of mania which occur in the place of an epileptic seizure or follow such a seizure. Transitory attacks of fury or frenzy coming on without apparent cause should always suggest the possibility of epilepsy.

Other forms which insanity assumes in early life are: transitory psychoses, such as delirium, night-terrors, attacks of sobbing or weeping, sometimes from fright; moral insanity, as shown by perversion of the moral sense from injury or disease, and by various vicious tendencies; morbid impulses, which may be homicidal or sexual, or a disposition to thieving, lying, pyromania, etc.; morbid fears, of which there may be an

almost endless variety. These are sometimes associated with a low state of physical health; this, however, is usually not the case.

Prognosis.—On the whole, insanity in childhood has a better prognosis than in the adult. In most of the cases of mania, melancholia, the various transitory psychoses, or the choreic and hysterical forms, recovery occurs with proper treatment. The outlook for the other varieties is much worse, especially in those in which there is a strong hereditary tendency to mental disease.

The treatment is to be conducted along the same general lines as in adults.

THE STIGMATA OF DEGENERATION.

These marks are of much importance in relation to the different forms of nervous disease in children, especially epilepsy, idiocy, and insanity. They are of great value in determining existing nervous disease, or as showing latent neuropathic tendencies.

The physician should be familiar with these various signs in order that he may connect them with each other and refer them to their proper source, and at the same time, by appreciating their significance, be able to advise parents with regard to the care, education, mode of life, and occupation of children, in whom to a greater or less degree these signs may be present. These stigmata are not of equal importance as marks of degeneration. Some of them, such as facial asymmetry and most of the deformities of the palate, are always to be so regarded; the speech defects are often so, while many of the others may or may not be, according to their association. The stigmata are divided into anatomical, physiological, and psychical. The following is the classification given by Peterson:*

Anatomical Stigmata.—Cranial anomalies: Facial asymmetry; deformities of the palate; anomalies of the teeth, tongue, lips, or nose.

Anomalies of the eye: Flecks on the iris; strabismus; chromatic asymmetry of the iris; narrow palpebral fissure; albinism; congenital cataract; pigmentary retinitis.

Anomalies of the ear.

Anomalies of the limbs: Polydactyly; syndactyly; ectrodactyly; symelus; phocomelus; excessive length of the arms.

Anomalies of the trunk: Herniæ; malformation of the breasts and thorax; dwarfishness; giantism; infantilism; femininism; masculinism; spina bifida.

Anomalies of the genital organs.

Anomalies of the skin: Polysarcia; hypertrichosis; absence of hair; premature grayness.

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* Deformities of the Hard Palate in Degenerates, by Frederick Peterson, M. D., International Dental Journal, December, 1895.

Physiological Stigmata.—Anomalies of motor function: Walking late; tics; tremors; nystagmus; epilepsy.

Anomalies of sensory function: Deaf-mutism; neuralgia; migraine; hyperæsthesia; anæsthesia; blindness; myopia; hypermetropia; astigmatism; Daltonism; hemeralopia; concentric limitation of the visual field.

Anomalies of speech: Mutism; defective speech; stuttering; stammering.

Anomalies of genito-urinary function: Enuresis; sexual irritability; impotence; sterility.

Anomalies of the instinct or appetite: Merycism; uncontrollable appetites for food, liquor, drugs, etc.

Diminished resistance to external influences and diseases.

Retardation of puberty.

Psychical Stigmata.—Insanity; idiocy; imbecility; feeble-mindedness; eccentricity; moral delinquency; sexual perversion.

DEAF-MUTISM.

Excluding the cases in which idiocy is present, which are not considered in this chapter, deaf-mutism may be due either to congenital or acquired conditions; the larger proportion of the cases belong in the latter class. When congenital, deaf-mutism may result from ostitis, or periostitis of the temporal bone, encroaching upon the cavity of the middle ear, from anchylosis of the ossicles, from absence of the internal ear or any of its parts. There may also be colloid degeneration of the labyrinth. It may result from atrophy of the auditory nerve, and it may be due to a lesion of the brain. These congenital conditions are often hereditary. Acquired deaf-mutism is most frequently the result of scarlet fever, and is due to otitis. The second important cause is cerebro-spinal meningitis, where it may be due to a lesion of the brain, the auditory nerve, or the ear. It occasionally follows mumps, diphtheria, measles, and other infectious diseases. It may result from repeated attacks of acute otitis associated with adenoid growths or chronic rhino-pharyngitis.

The younger the child at the time the deafness occurs the sooner the power of speech is lost. In most of the infectious diseases, if the attack occurs before the fifth year speech is lost. According to Love,* total deafness is rare among deaf-mutes; hearing for speech is present to a useful degree in about twenty-five per cent of the cases, while hearing by cranial conduction exists in nearly all cases. Deaf-mutism should be suspected if a child not idiotic shows at the end of two years no signs of beginning to talk. A careful distinction should be made between deaf-mutism and idiocy resulting either from congenital conditions or acquired disease.

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* Deaf-Mutism, by James K. Love. Macmillan & Co., 1896.

It is necessary that this condition be recognised as early as possible, in order that the child may have the advantages of proper training during its early years. The physician should insist upon the child being sent to an institution where it may be taught to speak as early as the third, and certainly by the fourth year.

The treatment is mainly prophylactic. The most important relates to the care of the ears in scarlet fever, and the removal of adenoid vegetations of the pharynx and other causes which produce attacks of acute or chronic otitis. For the condition itself education is the only thing to be considered.

CHAPTER IV.

DISEASES OF THE SPINAL CORD.

MALFORMATIONS.

Malformations of the cord are very frequently associated with those of the brain, and bear a certain degree of resemblance to them. (1) The cord may be absent (amyelia); this condition may exist alone or with absence of the brain. (2) The lack of development may be only partial (atelomyelia), as where some of the tracts are wanting. The most important one is defective development of the lateral tracts, which may be a cause of spastic paraplegia (Charcot). (3) There may be a malposition of some of the gray matter (heterotopia). (4) There may be a double cord (diplomyelia); the division is generally incomplete, and is attributed to an abnormal development of the central canal; it is usually associated with other deformities. All of these malformations are extremely rare and of very little practical interest.

There remains to be mentioned the only one which is really important—spina bifida.

Spina Bifida.—This is a malformation of the vertebral canal with a protrusion of some part of its contents in the form of a fluid tumour. The tumour is elastic, compressible, usually increased by crying, and sometimes by pressure upon the anterior fontanel. The contained fluid is clear serum, resembling in all respects the cerebro-spinal fluid. It is one of the most frequent congenital deformities.

According to Humphrey, spina bifida is due to an early failure in development,—in most cases before the cord is segmentated from the epiblastic layer from which it is developed. Hence it remains adherent to the epiblastic covering, and the structures which should be formed between the cord and the skin are undeveloped. For this reason we have in the wall of the sac a fusion of the elements of the cord, nerves, meninges, vertebral arches, muscles, and integument. If the error in development occurs

later, the cord and nerves may be attached to the sac, but not intimately fused with it; in still other cases the cord does not enter the sac at all. The malformation may occur before the central canal is closed; or, if closed, it may reopen from the accumulation of fluid. It is probable that the accumulation of fluid first occurs, and that this prevents the union of the parts of the vertebral arches.

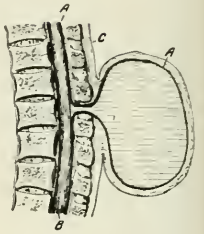

Fig. 129.—Meningocele (partially diagrammatic). *A*, the membranes; *B*, the spinal cord; *C*, the integument. The accumulation of fluid is behind the cord, which does not enter the sac.

Although the tumour is generally associated with a bifid spine, this is not necessarily the case. The protrusion may take place through the intervertebral notch or foramen, or there may be a fissure of the bodies of the vertebræ, and an anterior tumour projecting into the cavity of the thorax, abdomen, or pelvis,—spina bifida occulta. The principal anatomical varieties are meningocele, meningo-myelocele, and syringo-myelocele.*

Meningocele.—In this form there is a protrusion of the membranes only (Fig. 129). The accumulation of fluid is either in the arachnoid cavity or the subarachnoid space posterior to the cord. The opening of communication between the tumour and the spinal canal is small in this variety, usually being about one twelfth to one sixth of an inch in diameter. There may, however, be no communication. The skin is usually fully developed (Fig. 130). The tumour is frequently globular, sometimes pedunculated, and may attain a very large size, being as much as five or six inches in diameter. This is because spontaneous rupture is not likely to occur, and the tumour does not become infected except by operative interference. With such tumours patients may live to adult life. This variety is most frequently seen in the cervical region. It has the best chance of natural recovery, and in it operation gives the best results.

Fig. 130.—Meningocele, in a child one year old.

Meningo-myelocele.—This is by far the most frequent variety of spina bifida, occurring in thirty-five of the fifty-seven cases reported by Demme. It is the form usually seen in the sacro-lumbar region.

* See Report of London Clinical Society, 1885: and Humphrey, Lancet, March 28, 1885.

The accumulation of fluid takes place in the anterior subarachnoid space, less frequently in the anterior arachnoid cavity (Fig. 131). In this form the cord is contained in the sac, and usually forms a part of its wall. The tumour is smaller than the meningocele, the usual size being that of a mandarin orange. It is sessile, never pedunculated. As a rule it is only partly covered by skin, but has a central area, elliptical in shape, where there is only a thin, translucent membrane. This surface, which is known as the central cicatrix, is sometimes covered with granulations, and frequently ulcerates. The tumour often has a vertical furrow or a central umbilication, corresponding to the attachment of the cord on its inner surface. The usual relation of the parts is for the cord to run horizontally across the upper part of the tumour to the central cicatrix, with which it becomes blended, and from which again the nerves arise. These re-enter the canal at the lower part of the tumour, and are distributed below as usual. In other cases the cord joins the wall of the sac soon after its entrance, and its attenuated fibres are found spread out all over the sac, coming together again below and entering the spinal canal.

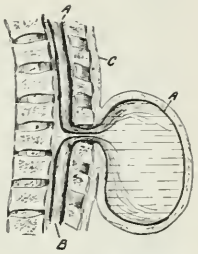

Fig. 131.—Meningomyelocele (partially diagrammatic). *A*, the membranes; *B*, the cord; *C*, the integument. The accumulation of fluid is in front of the cord, the filaments of which are spread out, forming a part of the wall of the sac.

The following case, upon which I recently made an autopsy, is a good example of the common variety: The child died on the third day after birth from rupture of the sac. The tumour occupied the sacral region. The first sacral vertebra was normal, and beneath this the cord passed, terminating in the cauda equina soon after entering the sac, and continued back to the central cicatrix. Here nerve filaments blended with the other tissues in an indefinite structure, from which again, with tolerable distinctness, they could be seen to pass over the wall of the sac and return to the canal. The afferent and efferent nerves and the part of the membranes they carried with them formed several septa, making a smaller separate sac within the larger one. The large sac was clearly a dilatation of the anterior subarachnoid space, and communicated freely with the same space in the cord above.

Syringo-myelocele.—In this variety the accumulation of fluid is in the central canal of the cord, the lining of the sac being here the attenuated and atrophied cord elements. This is the rarest form of tumour, but the one most frequently associated with hydrocephalus, and consequently having the worst prognosis. It is usually found in the dorsal or dorso-lumbar region, rarely in the lumbo-sacral (Fig. 132).

With spina bifida other deformities are frequently associated, the most common being club-foot, hydrocephalus, more rarely encephalocele or cerebral meningocele, and hare-lip. If hydrocephalus exists, there is in

most cases a dilatation of the central canal of the cord and a direct communication between the tumour and the lateral ventricles of the brain. Pressure upon the anterior fontanel causes an increase in the size of the tumour, and conversely. Club-foot is usually double, most frequently talipes equino-varus. In a number of cases there is a history of some deformity in other members of the family. I once saw two successive children in the same family with spina bifida.

Fig. 132.—Syringo-myelocele of the mid-dorsal region, in a child four months old, who also had hydrocephalus.

Symptoms.—The tumour is present at birth, and is most frequently situated just above the sacrum. Paralysis is frequent in myelocele and syringo-myelocele, but is not seen in meningocele; its degree and its location depend upon the situation of the tumour and the extent to which the cord is involved. It is rare in cervical tumours, and most marked in those situated in the lumbo-sacral region. In the worst cases there is complete paraplegia, with paralysis of the bladder and rectum. If the tumour is sacro-lumbar or sacral, only the cauda equina is likely to be involved, and this but partially, so that the paralysis of the extremities is incomplete, and the bladder and rectum may escape.

In Fig. 133 is shown a very remarkable case of sacral spina bifida in a boy of five years, who came under observation for incontinence of fæces. The tumour was a little more to the left than to the right side, and had been overlooked. It had evidently pressed upon the lower branches of the sacral plexus, so as to involve the sphincter and the gluteal muscles of the left side. The atrophy was very marked, as shown in the illustration.

Fig. 133.—Sacral spina bifida.

The natural course of spina bifida

is to increase steadily in size; and if the tumour is covered by skin, its growth may be almost unlimited. It has been known to attain a circumference of twenty-two inches. If the integument is wanting, and the sac wall is very thin, rupture is pretty certain to take place, either spontaneously or by some accident, in the course of the first few months; death then results from convulsions owing to the rapid draining away of

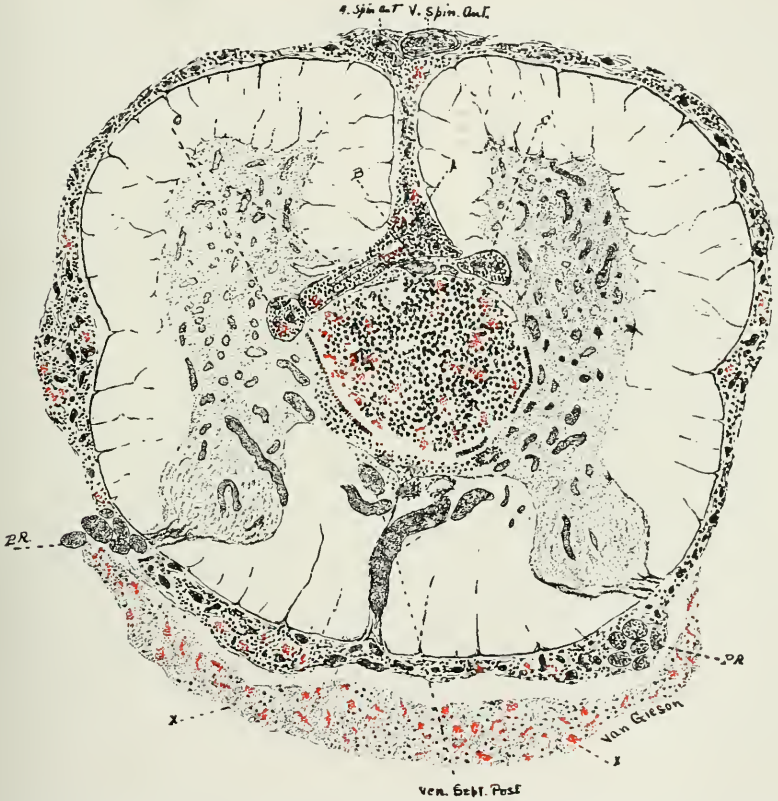

Fig. 134.—Spina bifida, with dilatation of the central canal of the cord, and spinal meningitis. The central canal is filled with round cells, among which are many cocci. *NV* is the pellicle of fibrin upon the posterior surface of the pia mater, also containing many cocci. The pia is everywhere infiltrated with cells, even to the bottom of the anterior fissure. The gray matter of the cord is much congested. *PR* is the posterior nerve root. The section is from the dorsal region of the cord.

the cerebro-spinal fluid, or from secondary infection. In a large number of cases death is due to marasmus dependent upon the associated conditions. Infection of the tumour may take place without rupture, the germs passing through the wall of the sac. If the opening communicating with the spinal canal is small, this infection may excite an inflammation limited to the wall of the sac, and result in a cure of the spina bifida, usually with

sloughing. I have now under observation a girl ten years old in whom this occurred in infancy. The site of the former tumour is marked by a large dense cicatrix, and there still remains partial paralysis of the legs. If the opening into the spinal canal is large, inflammation of the sac is usually followed by spinal meningitis, which may extend upward and involve also the meninges of the brain. In a case published by Van Gieson and myself,* in which there was dilatation of the central canal of the cord and hydrocephalus, bacteria penetrated the wall of the sac and travelled up the central canal of the cord (Fig. 134), finally exciting a suppurative inflammation in the ventricles of the brain, in addition to a spinal meningitis. Sections of the wall of the sac and of the cord at various levels showed the same cocci. The child died at the age of three weeks.

Prognosis.—This depends chiefly upon the anatomical variety and the existence of complications. Simple meningocele, when covered by integument, gives the best prognosis, and complete recovery may occur. In meningo-myelocele, if complete paralysis exists, the prognosis is bad; and if there is hydrocephalus, the case is hopeless. In quite a number of cases in which cure has followed operation, hydrocephalus has subsequently developed. Of fifty-seven cases reported by Demme, twenty-five were operated upon, with seven recoveries and fifteen deaths, while in three there was no result; of the thirty-two cases not operated upon, twenty-eight died within the first month, and not one lived over two years,—the causes of death being marasmus, rupture of the sac, and meningitis.

Diagnosis.—It is usually easy to recognise spina bifida, but it is often difficult to distinguish between the different varieties. The absence of a palpable fissure in the spine, perfect translucency, and a pedunculated tumour, all point strongly to meningocele. Paralysis of the sphincters and lower extremities, umbilication of the centre of the tumour, a sessile tumour, a palpable bony fissure, and a large central cicatrix, point to meningo-myelocele. The coexistence of hydrocephalus points to syringo-myelocele.

Treatment.—In all cases the tumour should be protected from pressure, and care taken where it is not covered by integument, that the surface is kept absolutely clean and aseptic. It should be covered with iodoform and bismuth and surrounded by a large pad of absorbent cotton, or a rubber ring-cushion. Complete paraplegia with involvement of the bladder and rectum, hydrocephalus, or extreme marasmus,—all contra-indicate operative interference. In other cases, operation should be considered. The time of operation will depend somewhat upon the nature of the tumour. If it is covered by integument and growing slowly, it is well to wait until the child is at least six months old. In other cases

* Journal of Nervous and Mental Diseases, December, 1890.

delay is dangerous, because of the liability to spontaneous or accidental rupture.

Nothing is to be expected from simple aspiration and compression. The methods of treatment which have been successfully employed are ligation, aspiration and injection, and excision of the sac. Ligation is admissible only where there is a pedunculated tumour; and even for these cases some surgeons prefer the clamp. Aspiration and injection have been widely used both in Europe and America. The needle should never be inserted near the median line. The tumour having been aspirated and about one half of its contents evacuated, there is injected, without removing the needle, a drachm of Morton's fluid (iodine, gr. x; iodide of potassium, gr. xxx; glycerin, ℥ j). If the tumour is pedunculated, pressure should be made at its neck to prevent the entrance of fluid into the canal. In all cases the child should be kept in a recumbent position for several hours. The operation is not entirely free from danger, as in some cases it has been followed by convulsions and death in a few hours. Considerable inflammatory reaction usually occurs, lasting from two to four days. After this period there is, in a favourable case, a subsidence of the swelling, with a gradual contraction and finally obliteration of the tumour. In some cases two or three injections may be required. The mortality of cases treated by this method is from forty to fifty per cent.* My own experience includes four cases, with two recoveries.

The dangers of this operation and the uncertainty as to its results have led many surgeons to discard it altogether in favour of excision, which with the technique of modern surgery is almost devoid of risk. For a description of this and the various plastic operations that have been proposed in connection with complete or partial excision of the sac, the reader is referred to works upon operative surgery. In operating, it should not be forgotten that in the great proportion of the cases (ninety-five per cent, according to the Clinical Society's Report, which, however, refers only to fatal cases) some part of the cord is in the sac. The cord is often present in tumours situated below the third lumbar vertebræ, owing to its attachment to the sac.

Although recovery may follow operation, in a very large number of cases it is incomplete; some degree of paralysis, with atrophy, contractures, and deformities, remaining because of the implication of cord elements in the sac.

SPINAL MENINGITIS.

In acute meningitis usually only the pia mater is involved. This rarely occurs alone, unless it is due to traumatism. It is most frequently associated with inflammation of the pia of the brain, and may occur either with

* Report of the London Clinical Society.

the simple or the tuberculous variety. A certain amount of acute inflammation of the pia mater accompanies most of the cases of acute myelitis.

Chronic spinal meningitis in children usually involves the dura only. Inflammation of the external layer (external pachymeningitis) is usually secondary to caries of the vertebræ. This is considered in the article on Compression-Myelitis.

Symptoms.—The symptoms of inflammation of the spinal membranes, no matter with what pathological condition it may be associated, are due to irritation of, or pressure upon, the cord or nerve roots. Those which are most common are: pain in the back, which is increased by movement, and usually by pressure upon the spinous processes; radiating pains following the course of the spinal nerves, felt in the extremities or in the trunk; rigidity of the spinal column due to spasm of the spinal muscles, or rigidity of the muscles of the extremities; and hyperæsthesia along the spine, which may be quite acute. When pressure upon the cord is added, there is paralysis or paresis, sometimes muscular atrophy and anæsthesia. Any of the above symptoms may be acute or chronic, according to the nature of the primary disease.

The diagnosis between spinal meningitis and myelitis is often not easy, for except in acute cases the two processes are usually associated; and in a given case it may be difficult to decide whether the lesion of the cord or of the membranes is the more important one. In meningitis, pain, tenderness, spasm, and irritative symptoms are generally more prominent, while loss of power and anæsthesia are usually partial. In myelitis the pain, tenderness, and other irritative symptoms are less marked, while paralysis and anæsthesia may be complete.

Treatment.—This is first of the disease with which it is associated; in addition, counter-irritation by means of the Paquelin cautery, rest in bed, and in severe cases even immobilization of the spine by a mechanical support. Iodide of potassium is often useful.

MYELITIS.

Myelitis is a rare disease in children, with the exception of two varieties, which are discussed under separate heads, viz., compression-myelitis and acute poliomyelitis. Otherwise myelitis usually results from injury, but it may occur as a complication of any of the acute infectious diseases, especially typhoid or scarlet fever, and diphtheria, and even as a primary disease, where it is attributed to exposure or cold, but where it is probably infectious. Chronic myelitis may be due to hereditary syphilis.

Myelitis usually occurs in children over ten years of age. In situation, it may be transverse, diffuse, or disseminated; the process may be acute, subacute, or chronic. The lesions and the symptoms are essentially the same as when the disease occurs in the adult.

Symptoms.—Myelitis usually comes on rather gradually, with only local symptoms; but the onset may be quite acute, with severe general symptoms,—fever, pain, prostration and localized or general convulsions. The local symptoms vary with the seat and the extent of the disease.

In transverse myelitis loss of power and anæsthesia are present below the level of the lesion; either of these may be partial or complete. At the level of the lesion there is a zone of hyperæsthesia and "girdle-pains." All the reflexes below the seat of the lesion are exaggerated. Those at the level of the lesion are lost. There may be loss of control of the sphincters, bed-sores, degenerative changes in the paralyzed muscles, contractures, and vaso-motor disturbances. The paralyzed muscles may be rigid or flaccid according to the seat and extent of the lesion.

When transverse myelitis is situated in the cervical region there are paralysis and anæsthesia of the arms, legs, and trunk. All the reflexes are exaggerated, and there is general rigidity of the paralyzed muscles. There are incontinence of fæces and retention of urine, followed by incontinence from overflow. The pupils are frequently contracted, and there may be optic neuritis. Atrophy, when present, usually affects the muscles of the arms, and indicates that the cord to a considerable extent is involved. There is great danger to life, owing to paralysis of the muscles of respiration.

When the seat of disease is the dorsal region, the symptoms are similar to those above described, with the exception that the arms escape, and that the eye-symptoms are usually wanting. This is the most favourable seat for the disease.

When the disease is situated in the lumbar region, in addition to paraplegia and anæsthesia of the legs, there is, from the beginning, incontinence of urine and fæces. The knee reflexes are lost; the muscles atrophy, and usually give the reaction of degeneration. Bed-sores are frequent.

In diffuse myelitis the symptoms are a combination of the above groups. If a large part of the cord is involved, there are general paralysis and anæsthesia, loss of reflexes, marked trophic disturbances, bed-sores, etc.

The course of myelitis is slow, and it usually progresses steadily from bad to worse. Death is due to exhaustion or complications—cystitis, bed-sores, or hypostatic pneumonia—or to some intercurrent disease. In a small proportion of the cases there may be partial recovery, but very rarely is this complete. The diagnosis is to be made from spinal meningitis, tumours, and hæmorrhage.

Treatment.—The treatment of the early stage consists in the use of ice to the spine, or counter-irritation by means of dry cups, mustard, or the Paquelin cautery. Later, the iodide of potassium should be given in all cases; improvement may follow its use, even when there is no suspicion of syphilis, but large doses are required, and for a long period. Electricity is contra-indicated except in chronic cases, and then but little improvement

is likely to result from its use. In these patients the most important thing is careful attention to cleanliness and to posture, in order to prevent bed-sores, cystitis, and pneumonia.

COMPRESSION-MYELITIS.

Synonyms: Pressure-Paralysis of the Spinal Cord; Pott's Paraplegia.

Compression-myelitis is usually the result of caries of the spine. It most frequently complicates this disease when the cervical or upper dorsal vertebræ are involved, it being quite rare when the lower half of the spinal column is affected. This difference is probably due to the smaller size of the spinal canal in its upper portion. According to Gibney,* paraplegia is seen in fifty per cent of the cases of caries of the upper half of the spine. Essentially the same condition, so far as the cord is concerned, may result from tumours of the spinal cord, or from anything else causing pachymeningitis. These, however, are exceedingly rare in childhood.

Lesions.—In spinal caries there occurs as a result of tuberculous disease a softening of the bodies of the vertebræ, which fall together from the pressure due to the superincumbent weight of the body. This causes a backward projection known as the kyphosis, or angular deformity. The spinal canal is encroached upon by the remains of the vertebral bodies whose ligamentous attachments have been loosened, and also by inflammatory products the result of periostitis, and localized inflammation of the dura mater, chiefly of the external layer, but which sometimes affects the internal layer also. All these conditions lead to the production of a mass of inflammatory material, often containing tuberculous deposits, which is chiefly in front of the cord, but may surround it. The compression takes place slowly in most of the cases, from the gradual progress of the lesions mentioned. In a small number of cases there may be a sudden pressure from the slipping backward of one of the vertebral bodies.

In recent cases the cord at the seat of compression is a little smaller than normal. It is usually involved to the extent of from half an inch to two inches. Paraplegia may have existed where the changes found in the cord are very slight, and sometimes where no changes are visible to the naked eye. In more protracted and more severe cases, the cord is much smaller at the point of disease, and under the microscope shows the changes of interstitial myelitis (Gowers) with meningitis. In old cases there are degeneration of the nerve elements, atrophy, and sometimes disappearance of the ganglion cells, with more or less destruction of the nerve fibres; sometimes all distinction between the gray and white substance is lost. In addition to these marked changes at the point of pressure, there may be ascending or descending degeneration, as from other focal lesions

* Journal of Mental and Nervous Diseases, April, 1878.

There is usually inflammation of the nerve roots, which have also suffered compression. It is in many cases surprising to see to what degree the cord may be compressed and still preserve its functions.

Symptoms.—In caries of the cervical region the symptoms of compression-myelitis not infrequently precede the deformity, and, in fact, the other objective symptoms of bone disease. The earliest symptoms of caries usually arise from irritation of the nerve roots, and consist of acute pains not often referred to the spine, but radiating to the different regions to which these nerves are distributed. They are felt in the neck, in the chest, in the epigastrium, and sometimes in the loins. Such symptoms indicate the presence of pachymeningitis, and may be present whatever the location of the vertebral caries. Accompanying these pains, there is noticed a gradual weakness in the lower extremities, and sometimes also in the arms, according to the location of the disease. This may steadily increase for several weeks until there is complete paralysis. Other symptoms are then commonly present. There is usually some degree of anæsthesia, but in many cases there is none, and there may be numbness, tingling, formication, and pain. The sphincters are not often involved. When the disease is in the upper half of the cord, there are rigidity of the extremities and great exaggeration of all the reflexes, with marked ankle-clonus. In the rare cases in which the lumbar enlargement is involved, there may be loss of reflexes, paralysis of the sphincters and bed-sores.

The distribution of the paralysis will depend upon the point of compression. If this is in the cervical region, all four extremities will be paralyzed; if in the dorsal region, only the legs. In rare cases the paralysis is unilateral, and if there is no spinal deformity the condition may be a most puzzling one. According to the extent of the secondary lesions in the cord, there may occur muscular atrophy and contractures. With disease in the upper cervical region, death may result from sudden pressure upon the cord, owing to a dislocation of the odontoid process, which happened in one of Gibney's cases; or there may be vomiting, pupillary symptoms, irritation of the phrenic nerve causing hiccough, or pressure causing paralysis of the diaphragm.

Course and Prognosis.—These depend much upon the treatment of the case. In many cases of paralysis occurring early in caries, complete recovery takes place in the course of a few weeks, sometimes in a few days, after the application of a proper mechanical support. This may be true even where the paralysis has continued for three or four months. In the cases which have been long neglected, or those in which the paralysis develops while proper mechanical treatment is being carried out, the chances of improvement, or at least of rapid improvement, are not nearly so good. Gibney gives the following statistics of fifty-eight cases under his personal observation: thirteen proved fatal, six dying from myelitis, five from other diseases subsequent to recovery from the paralysis, and two from

tuberculosis before complete recovery; twenty-nine recovered from the paraplegia, but relapses occurred in eight, all but one of these, however, recovering subsequently; fifteen cases were under observation at the time of the report. The usual duration of the disease is from twelve to eighteen months. Complete recovery has often taken place in cases that have persisted for four or five years. No case should be considered hopeless no matter how long the symptoms have lasted, unless there is marked atrophy with loss of electrical reactions, and contractures have taken place.

Diagnosis.—This is rarely difficult. Spinal caries should be suspected in every case where the symptoms point to transverse myelitis coming on without definite cause. The gradual onset, the radiating pains, the stiffness of the spine in walking, the gradual loss of power, the increased reflexes and ankle-clonus,—all are usually present and characteristic. They are sufficient to warrant the diagnosis of spinal caries, even when no deformity exists. When there is deformity, the symptoms are unmistakable.

Treatment.—The most important indications are the removal of pressure and the fixation of the spine by means of a proper mechanical support. If for any reason this is impossible, the patient should be kept in bed. The two other measures which promise most are the use of the Paquelin cautery, and the internal administration of potassium iodide. From his very extensive experience, Gibney has more confidence in this drug than in all else except mechanical treatment. Large doses are required, often from sixty to ninety grains being given daily for months. From personal observation of many of Gibney's cases I can bear testimony both to the beneficial effect of the iodide, and to the ease with which it is generally borne by children in the doses indicated. Very often patients gained steadily in weight while taking the drug, and acne was the exception. The iodide should always be largely diluted. In all cases patients should be carefully watched, kept scrupulously clean, and the position changed frequently to prevent the formation of bed-sores. Electricity is contraindicated. When the paralysis develops rapidly or occurs suddenly, relief may sometimes be obtained by the operation of laminectomy; but little is to be expected from this in the slow cases.

INFANTILE SPINAL PARALYSIS.

Synonyms: Acute Poliomyelitis; Acute Atrophic Paralysis.

This disease is characterized by an acute onset, generally with febrile symptoms, by an early and usually extensive loss of power, and by a considerable degree of spontaneous improvement except in certain groups of muscles which remain permanently paralyzed, and undergo a very rapid and marked atrophy. A chronic form of the disease is described in adults, but this is rarely, if ever, seen in children.

Acute poliomyelitis is the most frequent cause of paralysis in early life and it is often designated simply as *infantile paralysis.*

Etiology.—In 566 * cases the age at which the paralysis developed was as follows:

During the first year	20	per cent.
" " second year	38	"
" " third year	22	"
" " fourth, and fifth years	15	"
After " fifth year	5	"

From this table it will be seen that the great proportion of cases develop before the fifth year, and that eighty per cent of them begin during the first three years, the most frequent period being the second year.

Boys are rather more frequently affected than girls. In the series referred to, fifty-five per cent were males and forty-five per cent were females. Hereditary influences seem to have but little effect in the production of this disease. It is rare to find several cases in the same family, or to trace any relation to nervous antecedents. The onset of the great proportion of the cases is in summer. Of Sinkler's cases, eighty per cent began during the five warm months. This fact is decidedly against the theory so often advanced, that the disease results from exposure to cold. There are, however, a few cases in which the connection between exposure and the disease seems to be a close one. On account of the time of onset—most frequently in the second year—the disease is often ascribed to dentition. In my series this was given as the cause in one fifth of the cases. The connection is at most merely a coincidence. Traumatism is sometimes given as a cause, but the proportion of cases in which the paralysis can be fairly attributed to injury is very small, yet there are a few in which a definite injury of considerable severity has immediately preceded the onset. In about twelve per cent of the cases above mentioned the paralysis came on as a sequel to some other acute disease; this list includes nearly all the diseases of infancy, those most frequently noted being diarrhœa, scarlet fever, and measles; but in the great proportion of the cases the patient was in good health at the time of the attack.

The essential cause of the disease is as yet unknown. On account of the close relation of the lesion to the distribution of the blood-vessels, there has been of late a disposition on the part of many observers to regard it as infectious, the cord changes being the result of infectious embolism or thrombosis.

Lesions.—Infantile spinal paralysis is due to an acute inflammation of the gray matter of the anterior portion of the spinal cord. The late

* These statistics and those which follow in this article are derived from the following sources: Sinkler, in Keating's Cyclopædia, vol. iv, 355 cases; Galbraith, American Journal of Obstetrics, 1894, 75 cases; the remaining 146 are personal cases and others taken from the records of the Hospital for Ruptured and Crippled, New York.

changes which occur in the cord as a result of this process have for many years been well established; but the early changes are even yet a matter of dispute, owing to the lack of opportunities of examining the cord during the stage of acute inflammation.

In autopsies made upon cases of long standing, the part of the cord affected is distinctly smaller than normal. One lateral half is usually involved. The microscope shows that the ganglion cells are few in number or that they have entirely disappeared. Those that remain are shrunken and deformed and scarcely recognisable as ganglion cells. The entire gray horn is much smaller than that of the opposite side, and many of its normal elements have disappeared. The white matter also is smaller than in the sound half of the cord. The anterior nerve-roots of the affected side are smaller than normal, and are degenerated quite to the muscles. The general changes in the cord are of a sclerotic character. The affected muscles are degenerated, and there may be in extreme cases a complete disappearance of muscle fibres, their place being taken by adipose and fibrous tissue. In places where the lesion is less severe the fibres are small. The affected limb is shorter and the bones smaller than upon the sound side. These lesions are all secondary to those of the anterior ganglion-cells.

The most recent observations upon the early stage of the process by Siemerling, Goldscheider, and others, tend to show that primarily the lesion is an interstitial inflammation, and not a parenchymatous one, as was formerly believed. Goldscheider's * theory of the disease is that the first changes are in the blood-vessels, from which the process extends to the neuroglia and produces a proliferation of cells; the changes in the ganglion cells are degenerative in character, and are secondary to those just described; the same is true of the changes in the nerve fibres. Accompanying the process in some cases small hæmorrhages have been observed.

The region of the cord most frequently involved is the lumbar enlargement, but there may be more than one focus of disease. Usually only one lateral half of the cord is affected, but it is not rare for both sides to be involved. In such cases the lesions are generally more advanced upon one side than the other.

Symptoms.—A frequent form of onset is for a child to be taken quite suddenly ill with vomiting, pains in the legs, or general hyperæsthesia, and a temperature of from 101° to 103° F. After these symptoms have lasted a variable time, usually from one to four days, the paralysis is discovered. In a smaller number of cases—about ten per cent of the entire number—the attack is ushered in by more severe constitutional symp-

* Goldscheider, Zeitschrift für klin. Med., 1893, p. 494. See also Sachs, Nervous Diseases of Children, 1895, p. 310.

toms. There are convulsions, delirium, a temperature of 103° or 104° F., marked general prostration, constipation, severe pains in the back and extremities,—in short, all the symptoms of a severe acute inflammation. These symptoms last from two days to a week, often engrossing the attention of the physician, so that the paralysis may not be noticed until the patient has been sick for some time, or possibly not until the beginning of convalescence. In quite a large number of cases the general symptoms are very slight, and they may be absent altogether. A not infrequent history is that the child went to bed apparently well; during the night was noticed only to be a little restless, and that the next morning the paralysis was discovered. In two cases of my series the paralysis came on quite suddenly while the child was walking in the street, and was able to reach home only with considerable difficulty. In such cases it is not improbable that previous symptoms were present, but were so slight as to have escaped notice.

In most of the cases there are pains in the back, in the muscles of the extremities, or along the course of the spinal nerves. With these pains general hyperæsthesia is commonly associated, and there may be other disturbances of sensation such as numbness and tingling. The development of the paralysis is quite rapid, it often attaining its maximum in twenty-four hours; although sometimes it will be two or three days, or even a week, before its full extent is seen.

Extent and distribution of the primary paralysis.—In 560 cases in which this point was noted the distribution was as follows:

One lower extremity	229 cases.
Both lower extremities	176 "
General paralysis of all extremities, and more or less of trunk	79 "
One lower and one upper extremity	36 "
Both lower extremities and one upper extremity	16 "
One upper extremity alone	14 "
Both upper extremities	2 "
All other varieties	8 "

In paralysis of the trunk, the diaphragm and other respiratory muscles are very rarely affected. In combinations of an upper and a lower extremity, the limbs are more frequently affected upon opposite sides than upon the same side. The sphincters almost invariably escape.

Course of the disease.—The rapid development of the paralysis is followed by a period of from one to four weeks' duration in which but little change is seen in the affected muscles. This is followed by spontaneous improvement, which, according to Gowers, begins in the muscles last affected, and generally reaches its limit in about three months. After this time but little spontaneous improvement is to be looked for, and the residual paralysis is likely to be permanent. By the end of two months marked atrophy is present in the paralyzed muscles. The affected limb is distinctly smaller than its fellow, this being quite apparent even in

infants. Except at the onset, sensory disturbances are absent; the knee-jerk is lost in paraplegic cases, and in those in which the extensors of the thigh are paralyzed. There is arrested growth in the whole limb (Fig. 135). It becomes much smaller and shorter than its fellow. The great relaxation of the ligaments at the joints may allow subluxation, especially at the knee and at the shoulder. The circulation in the affected limb is poor; it is often blue and cold, but bed-sores are never seen.

Electrical reactions.—Very early in the disease the atrophied muscles begin to lose their power to respond to faradism. In the muscular groups which are to be permanently paralyzed, the faradic response may be lost in a week. The muscles in which recovery is to take place often preserve a certain degree of contractility, although this is less than normal, and improves later. The response to the galvanic current may be increased for a few months, and then slowly fail as the muscular fibres themselves degenerate, and at the end of two or three years it may disappear altogether. The reaction of degeneration is present with great uniformity in the atrophied muscles, but in them alone.

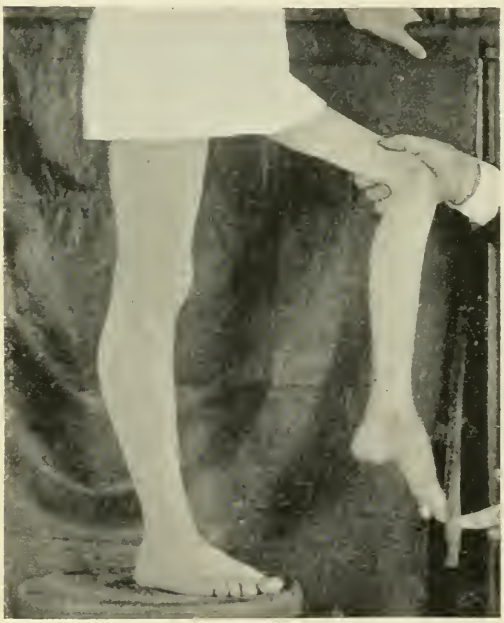

Fig. 135.—An old case of infantile spinal paralysis of the entire left lower extremity, showing extreme atrophy of the thigh and leg, and a very characteristic deformity of the foot.

Residual paralysis and deformity.—Only one lower extremity is involved in half the cases, and the paralysis is usually incomplete and confined to certain groups of muscles. The extensors both of the thigh and of the leg are nearly always involved to a greater degree than the flexors, and in very many cases only the extensor groups are paralyzed. The muscles most frequently affected are the anterior tibial group, and next the peroneal group. The most frequent deformity resulting from this paralysis is talipes valgus, and next to this talipes varus, both of these being usually associated with a certain amount of equinus. In very rare cases there is talipes calcaneus. Most children with paralysis of only one

lower extremity are able to walk alone, or with the assistance of a steel brace.

Paralysis of both lower extremities is the next in frequency. This also is rarely complete. In forty-three cases of my series there was originally complete paraplegia, but it was permanent in only three. The extent of recovery varies much in different cases. Usually one leg re-

FIG. 136.—An old case of infantile spinal paralysis of the left arm and shoulder muscles, with resulting lateral curvature. The spinal deformity is increased by the fact that the patient had also suffered from empyema of the left side.

covers to a much greater degree than the other. Most of these patients are able to walk with the assistance of braces, a few only by the aid of crutches. Some walk while they are young, but are unable to do so when fully grown, because the disproportion between the size of the body and the limbs is then much greater.

Paralysis of one upper extremity rarely occurs alone, but is associated with paralysis of one or both lower extremities. Complete paralysis of an arm is rarely, if ever, seen. The muscular groups affected may be the small muscles of the hand, the muscles of the forearm,—especially the extensors,—or the shoulder group. Of single muscles, the one most frequently involved is the deltoid; this may result in subluxation of the shoulder. From paralysis of the muscles of the trunk or shoulder of one side, lateral curvature may develop (Fig. 136). If the serratus magnus is affected the scapula stands out prominently, giving rise to the so-called "angel-wing" deformity.

Diagnosis.—The general symptoms of the onset have nothing characteristic about them, and no diagnosis can be made until the paralysis has taken place. The acute onset, the rapid wasting, the spontaneous improvement in certain groups of muscles, the absence of sensory symptoms, and finally the reaction of degeneration,—all constitute a type which it is difficult to confound with any other disease.

At the onset this paralysis may resemble that resulting from acute transverse myelitis. In the latter, however, we get anæsthesia, exaggerated knee-jerk, ankle-clonus, generally involvement of the sphincters, a tendency to bed-sores, slight wasting, and no reaction of degeneration. It is, besides, extremely rare.

Multiple neuritis is in most cases easily distinguished from poliomyelitis by its gradual onset, by the presence of pain and other sensory symptoms as well as loss of power, and by the fact that spontaneous recovery generally occurs within two or three months. Besides, there is usually a history of antecedent diphtheria. But multiple neuritis sometimes begins suddenly with febrile symptoms, and paralysis may occur early, precisely as it does in poliomyelitis. Furthermore, in some cases of neuritis, the sensory symptoms are not marked, and they may have entirely disappeared before the patient is seen. In such cases the diagnosis from poliomyelitis may be difficult or even impossible except by the course of the disease; for atrophy is common to both conditions, and even the electrical reactions may be identical. There is no doubt that some cases formerly reported as examples of poliomyelitis terminating in complete recovery were really cases of multiple neuritis.

The diagnosis from acute cerebral palsy is chiefly difficult when the spinal paralysis has been hemiplegic or diplegic in type, or when after cerebral hemiplegia the leg or the arm has recovered so completely that the case resembles monoplegia. In cerebral palsies there is usually rigidity; there is no reaction of degeneration; other cerebral symptoms are commonly present, or there is a history of an onset with cerebral symptoms; and the atrophy is less marked. The most diagnostic point is the electrical reactions.

Infantile spinal paralysis may be mistaken for other than nervous dis-

eases. In the early stage it may be confounded with the pseudo-paralysis of scurvy. I have several times seen the mistake made of diagnosticating paralysis where scurvy was present. In scurvy, however, there are seen excessive tenderness and hyperæsthesia, pain upon motion, especially about the knees, spongy gums, and sometimes ecchymoses about the joints. The muscular weakness of rickets is sometimes mistaken for infantile paralysis. However, in rickets the symptoms are always bilateral, the electrical reactions are normal, and other signs of rickets are present. In all doubtful cases the chief reliance for the diagnosis of paralysis should be placed upon the character of the electrical reactions. The lameness resulting from paralysis may resemble somewhat that due to hip-disease; but with a careful examination there can rarely be any difficulty in making the differential diagnosis.

Prognosis.—Infantile spinal paralysis is accompanied by little, if any, danger to life. It is possible that death may take place during the stage of acute inflammation, but this is certainly extremely rare. The most important question in early prognosis is whether there will be any permanent paralysis, and, if so, what will be its extent. The important symptoms for prognosis are the amount of wasting and the condition of the electrical reactions. Muscles which in ten days have lost completely their faradic contractility are almost certain to waste rapidly and severely. The best indication of coming improvement is the return of faradic contractility. If this is completely lost for six months, recovery is doubtful; if for one year, improvement in these muscles is not to be expected. If faradic contractility has never been lost, very great and early improvement in the paralyzed muscles may be confidently predicted. After three months but little spontaneous improvement is to be looked for, and after two years none at all. Complete recovery is possible only with a lesion of very limited extent; and while it may occur, it is so infrequent that it is never to be expected.

Treatment.—Unfortunately, most of the cases do not come under observation during the acute stage, or the nature of the disease is overlooked until the paralysis has occurred. In the early stage the indications are, to induce free perspiration by hot baths, to keep the patient in a prone or lateral position, and to use counter-irritation to the spine by means of dry cups, mustard, or the Paquelin cautery, or an ice-bag may be placed along the spine. The natural course of the disease is to be kept in mind, for the tendency is to overestimate the effect upon the paralysis of the drugs used in the early stage. On theoretical grounds, ergot is indicated, but it is doubtful whether any drugs have much effect.

After all acute symptoms have subsided, or at the end of two or three weeks, electricity may be used, but its curative effects have been very greatly overestimated. The object in using electricity is to keep up the nutrition of the muscles until the cord has recovered, which it is almost

certain to do to a considerable degree. But no amount of electrization can preserve muscles whose ganglion cells have completely disappeared. These continue to waste and lose their faradic contractility, no matter how early electricity is begun nor how faithfully it is continued. Faradism may be used for such groups as respond to it; otherwise galvanism should be employed. The beneficial results from electricity are to be obtained in the first year, chiefly in the first six months. Too much can not be said against the routine use of electricity in cases which have been paralyzed three or four years, with the vain hope that some good may be done, even though there is no response to either current. Strychnine may be used in conjunction with electricity after all symptoms of central irritation have subsided, but there is still great diversity of opinion regarding its effect.

Friction and massage are of undoubted value in improving the circulation and the nutrition of a limb, and should be continued regularly twice a day for a long period.

Mechanical Treatment.—The first use of mechanical appliances is the prevention of deformity. All cases of paralysis should be carefully watched, and braces applied as soon as any tendency to deformity from muscular contraction shows itself. This is much easier than to overcome deformities which have been allowed to develop, and quite as important for the patient. The second use of apparatus is to furnish support to the limb in order to enable the child to walk. By such means many get about with tolerable comfort, for whom locomotion without apparatus is impossible except with crutches. The third purpose of apparatus is, to overcome existing deformities in neglected cases.* Braces are generally used in conjunction with myotomy or tenotomy of the various shortened tendons, excision of portions of elongated tendons, and the production of artificial anchylosis in cases of "flail joints." By these means the orthopædic surgeon is able to give a great deal of relief to these unfortunate and sometimes helpless patients.

On the whole, the treatment is extremely unsatisfactory, and the result depends upon the severity and extent of the original disease, rather than upon the particular line of treatment adopted or the time at which it is begun.

TUMOURS OF THE SPINAL CORD.

Tumours of the cord are exceedingly rare in childhood, and almost unknown in infancy. The most common varieties seen in early life are glioma, sarcoma, and tuberculous tumours. Eisenschitz has reported a case of tuberculous tumour in the dorsal region occurring in a child of

* See Gibney, New York Medical Journal, April 3, 1886, On the Limitation of Therapeutics in Infantile Paralysis.

three and a half years. There was a similar growth in the cerebellum. The symptoms were essentially those of compression-myelitis.

In my service at the Babies' Hospital I recently had a case of glioma of the cord in a child only one year old, which was in many respects unique. The early symptoms were gradual paralysis of the upper extremities, to which were added later, stiffness of the neck, and finally immobility of the head,—the position being that of typical cervical caries. During the sixteen days of observation there was high fever, from 101° to 104° F. There were no pupillary or vaso-motor symptoms. At the autopsy the cord was found to be the seat of a diffuse gliosis. In the cervical region there was marked enlargement, the cord being fully four times its natural size. A microscopical examination by Dr. C. A. Herter showed that the growth apparently began in the vicinity of the central canal, and that the gliomatous process involved the entire length of the cord.*

A somewhat similar case has been reported by Miura in a boy of eight years.

The diagnosis of tumours of the spinal cord in infancy is practically impossible. In later childhood they are most apt to be mistaken for Pott's disease, but the symptoms are the same as those seen in tumours of adult life.

SYRINGO-MYELIA.

Syringo-myelia, although a rare disease, is sometimes seen in early life. The term is applied to a condition in which there is a cavity in the cord the result of a pathological process, in contradistinction to the cases in which a cavity is the result of a malformation, or *hydromyelus*, although it is not infrequent for the two conditions to be associated. The pathological process which precedes the cavity formation is now thought to be, in most cases at least, an infiltration of the substance of the cord with gliomatous cells. The process is somewhat similar to that just described in the case of tumour of the spinal cord, with the exception that where it results in cavity formation it is slower. The infiltration in these cases usually begins near the central canal. It is followed by a degeneration and breaking down of the infiltrated areas, beginning at the centre. As the cavity forms it extends, and usually first invades the gray matter of the commissure, later the posterior gray horns, the posterior columns, or the anterior horns. The resulting cavity is usually irregular in shape, and may be very small, or may extend through a large part of the length of the cord. It is most frequently situated in the lower cervical and upper dorsal regions. It is filled with fluid, and surrounded by gliomatous tissue.

* For a full report of this case by Dr. Herter and myself, see American Journal of the Medical Sciences, April, 1895. See also Kohts, Beitrag zur Diagnostik der Rückenmarkstumoren im Kindesalter, Dresden, 1886.

According to Starr, the essential symptoms are of three kinds: (1) There is progressive muscular atrophy, with paralysis of some or all the muscles of one limb, usually extending to the opposite limb and to the trunk, sometimes accompanied by the reaction of degeneration; (2) vasomotor and trophic disturbances in the affected limb, such as cyanosis, coldness, bullous eruptions, ulceration, abscesses, atrophy, and sometimes fragility of the bones and diminution of perspiration; (3) sensory disturbances, which are probably the most characteristic symptoms of the disease,—there is loss of the sense of pain and of temperature in the atrophied part, while the sense of touch and of location may be preserved. The extent and distribution of these symptoms will of course depend upon the position of the disease.

The course of syringo-myelia is essentially chronic, the duration being usually several years; and although spontaneous arrest sometimes occurs the disease is in most cases steadily progressive.

The cause is unknown, and it is not influenced by any form of treatment.

FRIEDREICH'S ATAXIA.

This is a chronic disease of the spinal cord and medulla, which begins most frequently in childhood or about puberty. The lesion affects first the posterior columns, afterward the crossed pyramidal tracts, the direct cerebellar tracts in the lateral columns, and Clarke's vesicular columns in the gray matter of the cord. There is probably some disease of the medulla, the pons, and possibly of the cerebellum and the posterior nerve-roots. In advanced cases other parts of the cord may be involved. The disease is seen in certain families, often affecting several members in succession at about the same age. It occurs particularly in families where alcoholism, insanity, and other nervous diseases are frequent.

Bramwell, in his monograph upon this disease, gives the following as the characteristic symptoms: There is ataxia, first of the lower extremities, but gradually extending to the upper extremities and the face. Early in the disease there is some weakness in the legs, especially in the anterior group of muscles. In the late stages this is marked and accompanied by atrophy. The gait is peculiar, like that of ordinary ataxic patients, the difficulty in walking being due to the ataxia and not to the paresis. After a time there is produced a characteristic deformity of the foot,—it is shortened, as if from pressure against the toes and the heel, the instep is high, and the extensor tendon of the great toe stands out prominently. This deformity is seen quite early in the disease. There is often lateral curvature of the spine. The knee-jerk is absent. Unprovoked and uncontrollable laughter is quite a characteristic symptom of the disease. The patient is unable to stand with his eyes closed. There are palpitation, occipital headache, and

sometimes vertigo. In the later stages speech is slow and difficult, and the patient talks like one intoxicated. The expression of the face is vacant, and often nystagmus is present. There may be choreic movements. The symptoms steadily progress until the patient may be helpless, although the general health may remain good for years.

The disease is distinguished from locomotor ataxia by the absence of the "lightning pains," and of the bladder, rectal, or genital symptoms, the pupillary changes, the optic-nerve atrophy, and the trophic changes in the bones and joints. It is distinguished from cerebral tumour by the absence of headache, vomiting, and optic neuritis, and by its longer course. The progress of the disease is slow but steady. It may last from twenty to thirty years. It is incurable.

LANDRY'S PARALYSIS (ACUTE ASCENDING PARALYSIS).

This rare disease is occasionally seen in early life. In regard to its etiology but little is definitely known, the usual causes assigned being the same as those of myelitis.

It is characterized by a paralysis—sometimes preceded by general symptoms of *malaise*, fever, etc.—which begins in the legs and spreads rapidly to the muscles of the trunk and upper extremities; finally it may involve the neck, diaphragm, and muscles of articulation. The paralysis develops quite rapidly, often attaining its height in from twenty-four to forty-eight hours, sometimes even proving fatal within this time. In other cases it comes on gradually, and may be two or three weeks in reaching its maximum. There is dyspnœa from involvement of the muscles of respiration. The paralyzed muscles are flaccid. There is hyperæsthesia, followed by partial or complete anæsthesia and loss of reflexes. There are no changes in the electrical reactions, no atrophy, no bed-sores, and usually no involvement of the sphincters. Occasionally the arms may be affected before the legs, and even the bulbar symptoms may be the first noticed. Death is the most frequent termination, and in fatal cases the disease lasts from two days to a week. If recovery takes place, it is after two or three months of illness.

The pathology of the disease is as yet unknown. The indications for treatment are the same as in acute myelitis, for in the beginning the two diseases can not usually be distinguished from each other.

THE MUSCULAR ATROPHIES.

These cases may be broadly divided into two groups, following in the main the classification of Sachs:* (1) Those dependent upon disease of the spinal cord,—the spinal atrophies; (2) those which are primarily diseases of the muscles themselves,—the idiopathic atrophies.

* New York Medical Journal, December 15, 1888.

In the group of atrophies of spinal origin belong (1) the "hand type" of Aran and Duchenne, which has been shown to be dependent upon a lesion of the spinal cord; (2) the "peroneal type" of Charcot, Marie, and Tooth, which as yet lacks positive pathological proof of its spinal origin, although its etiology, symptoms, and course leave but little doubt that it belongs in the same category with the hand type.

In the second (idiopathic) group are included (1) pseudo-muscular hypertrophy, and (2) the so-called "juvenile atrophy" of Erb, which is a much less frequent condition. These two varieties have the following features in common: There is progressive wasting, beginning early in childhood, and associated at some period with hypertrophy of certain muscles. There are no fibrillary contractions, no reaction of degeneration, and no lesions in the cord. From a pathological point of view these diseases might be more properly considered elsewhere, but they are so closely associated clinically with the spinal atrophies that it has seemed better to describe them in this connection.

Progressive Muscular Atrophy of the Hand Type.—This disease is characterized by a very slow but progressive wasting, which usually begins in the muscles of the ball of the thumb of one or both hands. Then the palmar group of muscles belonging to the little finger are affected, and later the interossei. When the wasting has reached a certain degree, there is produced a peculiar and characteristic deformity of the hand known as *main en griffe*, or "claw-hand." Following these muscles, those of the forearm may be affected. At this point the disease is sometimes arrested, or the atrophy may extend to the muscles of the arm and shoulder, especially the deltoid, and finally to those of the back. Exceptionally, the atrophy begins in the muscles of the shoulder group or even in those of the leg. The wasting takes place very slowly, the muscles disappearing fibre by fibre, but the degree which may be reached is often extreme. The only other characteristic symptoms are fibrillary contractions in the muscles which are soon to atrophy. The patient is not conscious of them, but they are visible. The faradic contractility is preserved just in proportion to the amount of muscle remaining. If the atrophy is complete, it is entirely lost.

The course of the disease is a very chronic one, covering many years. It is incurable. In rare cases the process may extend to the muscles of the tongue, affecting deglutition and articulation, and death may occur from interference with respiration; otherwise the disease does not tend to shorten life.

In this form of atrophy heredity is an important etiological factor. The disease may occur in children, but very often does not begin until after puberty. The lesion consists in an atrophy of the ganglion cells of the anterior horns of the spinal cord, followed by secondary degeneration of the anterior nerve-roots.

Progressive Muscular Atrophy of the Peroneal Type.—This is much less frequent than the variety just described. In this form, the first to waste are the anterior muscles of the leg, especially the extensor longus hallucis and extensor communis digitorum, afterward the peroneal group. The small muscles of the foot are next affected, and the disease may then go on to involve the muscles of the calf. At this point it may be arrested permanently, or for several years, after which the thigh muscles may waste like those of the leg. After many years the hands are in some cases involved as in the type previously described, and even the muscles of the forearm. As a rule, the supinator longus, the muscles of the shoulder, neck, trunk, and face, escape altogether. The atrophy is generally symmetrical, but not invariably so. The cutaneous reflexes are usually present. There is no pain. The reaction of degeneration is present in some of the muscles, and fibrillary contractions are frequent, but not always seen.

In this variety also the influence of heredity may often be traced. It is said that boys usually inherit the disease through the mother. Like the previous type, it begins late in childhood or not until after puberty.

As stated above, positive proof that this disease is due to a central lesion in the cord is as yet lacking. Analogy, however, leads to the belief that it depends upon changes in the ganglion cells of the anterior horns in the lumbar region, similar to those found in the cervical region in the hand type. The course of the disease is very chronic, and it, too, is incurable. The resulting deformity resembles that seen after poliomyelitis, and may require the same mechanical treatment, with similar operations for relieving contractions.

Pseudo-Muscular Hypertrophy (Pseudo-Hypertrophic Paralysis).—This is the most frequent and best-known variety of the idiopathic atrophies. It is a disease of certain families, often three or four children being affected, the boys much more frequently than the girls. The symptoms as a rule come on early in childhood, nearly always before the tenth year. The earlier symptoms relate to a general weakness of the lower extremities, which is accompanied by a marked increase in the size of certain muscular groups, usually those of the calves, but sometimes more of the thighs or the gluteal regions. Children walk late and unsteadily, and fall very easily. They have special difficulty in rising from the floor and in mounting stairs. The method of rising is quite characteristic: the patient lifts his body until he touches the floor only with the hands and feet; then he proceeds to "climb up himself" by putting first one hand upon the knee, and then the other, gradually moving his hands higher and higher up the thighs until the erect position is attained. This is seen in most of the cases, but not in all.

The size attained by the calves is sometimes very great. Gowers mentions a case in which a boy of twelve had calves measuring fourteen and a half inches in circumference. The enlargement may affect almost any

muscular group of the lower extremity. In the upper extremity, the infra-spinatus is most frequently enlarged, next the supra-spinatus and the deltoid. The pectorals and latissimus dorsi are never enlarged, but are generally markedly wasted. Most of these patients exhibit while standing a marked degree of lumbar lordosis, due to the weakness of the extensors of the hip. This is well shown in Fig. 137. The patient may be so weak upon his legs that the slightest touch will cause him to fall, even with his apparently immense muscular development. The small muscles are generally weaker than those which are enlarged.

Fig. 137.—Pseudo-muscular hypertrophy, showing to a moderate degree the large calves and gluteal regions with a marked lordosis. (From a photograph by Dr. M. A. Starr.)

Later in the disease marked atrophy occurs with a corresponding weakness of all the affected groups, and the patient may be unable to walk or even stand. With the exception of the use of his hands, he may be absolutely helpless. The knee-jerk is at first normal, but gradually diminishes until it is finally lost. The electrical reactions are normal until marked wasting occurs, when there is a lessened response to faradism and galvanism, but never the reaction of degeneration. There are no fibrillary contractions, and no sensory disturbances. The progress of the disease is generally slow, and sometimes irregular. It is often more rapid in early childhood, and slower after puberty.

The lesions are confined to the muscles. At autopsy they appear yellow, and microscopically there is found very marked atrophy of the muscle fibres, which in places have been almost entirely replaced by fat; there may be no trace of muscle left, the structure resembling adipose tissue. In other places there is an accumulation of fat between the atrophied muscle fibres, and a very great increase of the interstitial tissue.

The prognosis is grave, most patients dying before adult life is reached. The diagnosis is generally easy from the apparent hypertro-

phy and actual weakness of the muscular groups. The disease is incurable.

The Juvenile Form of Muscular Atrophy.—This is much less frequent than the form just described, but, like it, begins in childhood or early youth. It is characterized by progressive wasting of certain muscular groups, especially those about the shoulders and pelvis, and hypertrophy of others. Of the shoulder and upper extremity, the muscles affected are the pectorals, the trapezius, the latissimus dorsi, the serrati, the rhomboidei, the muscles of the upper arm, and the subscapularis. The deltoid, infra-spinatus and supra-spinatus for a long time escape, and may be hypertrophied. The hand and forearm are not involved. In the lower extremity, the muscles of the pelvis, thighs, and gluteal regions are affected, while those of the leg and foot escape. With this atrophy there may be associated a true or pseudo-hypertrophy of certain muscular groups. In this disease there are no fibrillary contractions, no reaction of degeneration, and no sensory disturbances. The course and result of this form are essentially the same as in the preceding variety. It is now generally regarded as closely allied to it in its pathology, the most important difference being that of localization.

There has been described, chiefly by Landouzy and Déjerine, another form of atrophy known as the *infantile facial type.* In this, wasting begins in the muscles of the face; the lips are thickened, but all the rest of the facial muscles are markedly atrophied, giving a peculiar expression to the mouth known as "the tapir mouth." Later, the atrophy extends to the shoulders and arm, but does not involve the supra-spinatus or infra-spinatus, or the flexors of the hand and forearm. This is sometimes described as beginning in the shoulders, or even in the legs. The description therefore corresponds to the juvenile form of Erb, with the addition of facial symptoms, and it is probably a variety of the same disease.

CHAPTER V.

DISEASES OF THE PERIPHERAL NERVES.

MULTIPLE NEURITIS.

Under the term multiple neuritis are included those cases in which several nerves are involved in an inflammatory process, which may at times be general. In its distribution multiple neuritis is usually symmetrical, but it is not necessarily so.

Etiology.—The chief cause of multiple neuritis in children is diphtheria, although it is occasionally seen after other infectious diseases, especially malaria, typhoid or scarlet fever, and measles. In diphtheria

the inflammation is due to the direct action of the toxines upon the nerve structures, since it can be induced in animals by injecting toxines into the circulation. There is little doubt that in all infectious diseases the inflammation is excited in a similar way. The metallic poisons, lead and arsenic, are rarely the cause of multiple neuritis in early life, and the same is true of alcohol, although a marked case from this cause has recently come under my observation in a child only three years old.* Lastly, there are cases in which the cause assigned is simply exposure to cold,—those classed as rheumatic.

Lesions.—Almost any nerves in the body may be affected, although the distribution varies somewhat with the cause of the disease. The musculo-spiral and the anterior tibial nerves are most frequently involved, but the inflammation may affect any of the spinal nerves, including the phrenic, and occasionally the cranial nerves, especially the pneumogastric hypoglossal, oculomotor, and abducens. Several nerves in different parts of the body are usually affected, the lesion being in most cases symmetrical.

The affected nerve is sometimes red and swollen, owing to acute congestion and œdema or a sero-fibrinous exudation. In other cases the changes are almost entirely degenerative. The microscope shows the changes sometimes to be chiefly interstitial and sometimes chiefly parenchymatous. There is an exudation of cells into the sheath, between the sheath and the nerve fibres, and even between the nerve fibres themselves. The myeline breaks up into granules, and in places may completely disappear.

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* This case was in many respects a remarkable one. The boy completely emptied a decanter containing twelve ounces of whisky, but almost immediately vomited the greater part of it. He soon after showed the symptoms of alcoholic intoxication, and in a few hours became comatose, in which condition he continued for twelve hours. After this he gradually lost power in his legs, and at the end of a week was unable to walk at all. He had convulsions, and after this there developed the usual symptoms of meningitis at the convexity, with which he was admitted to the Babies' Hospital, December 13, 1895, three weeks after drinking the whisky. The child was then unconscious and there was present incomplete paralysis, affecting all four extremities, with anæsthesia of the arms. The active inflammatory symptoms continued for six weeks longer, during which time there were repeated convulsions, continuous stupor, fever, gradually increasing deformities, marked atrophy, loss of reflexes, and great diminution in the faradic contractility of all the paralyzed muscles; in the thighs, left leg, and abdominal muscles there were no responses to a strong current, but there was nowhere the reaction of degeneration. The child was at death's door for three or four weeks. Three months after the attack the first signs of improvement were observed in the cerebral symptoms. Shortly afterward he began to use his hands, and at the end of six weeks he was walking alone and talking freely. The improvement was very rapid, and eight weeks from the date of the first change for the better, and five months from the time of taking the whisky, he was as well as ever. The diagnosis was multiple alcoholic neuritis, with a convexity meningitis. (Fig. 138 is from a photograph taken while the symptoms were at their height.)

The late changes are those of subacute or chronic degeneration of the nerve fibres.*

With these changes in the nerves there are associated, in some cases, inflammatory and degenerative changes in the ganglion cells of the spinal cord, although they are much less severe than are the lesions in the nerves. However, they were once regarded as the explanation of some of these cases, particularly of diphtheritic paralysis.

Symptoms.—The onset of multiple neuritis is in most cases a gradual one, it being usually from two to four weeks before the paralysis reaches its height. Very exceptionally the onset may be abrupt, with fever, and marked paralysis in a few days. It is characteristic of this disease that both motor and sensory symptoms are present, and that they

Fig. 138.—Alcoholic neuritis, showing characteristic dropping of the feet. This position of the lower extremities was maintained for over a month. Boy three years old.

are the same in their distribution. The symptoms are usually symmetrical. There is first noticed a general weakness in the affected muscles, which slowly increases to complete paralysis. As the extensor groups of the hands and feet are apt to be affected, there are wrist-drop and foot-drop (Fig. 138). The paralysis may begin in the feet and hands, and gradually extend until it involves not only the four extremities, but even the muscles of the trunk and the neck, although this is rare. The child may then be absolutely helpless, unable to sit up, or even to support its head. In such cases the head seems loosely attached to the body, and rolls about on the shoulders like a ball. Weakness of the spinal muscles leads to deformities (Fig. 139), which I have seen mistaken for Pott's dis-

* For a full description of the lesions, consult Starr's Middleton-Goldsmith Lectures, New York Medical Record, 1887.

ease, even by experienced observers. In most of the muscular groups the paralysis is incomplete. The symptoms which relate to the phrenic and the cranial nerves will be described with Diphtheritic Paralysis, for they are rarely seen in any other form. It is characteristic of multiple neuritis that the bladder and rectum escape.

The sensory symptoms are marked only in the early stage of the disease, while the paralysis is increasing; they improve so much more rapidly than the motor symptoms, that they may be altogether wanting at the time that the paralysis is at its height. In some cases they are so slight as to be overlooked. There is usually pain along the course of the affected nerves, which is sharp and neuralgic in character, and generally associated with acute tenderness of the nerve trunks and of the muscles. Often there is a general hyperæsthesia in the early part of the attack, followed by partial anæsthesia. The sensations of touch, pain, temperature, and the muscular sense are all about equally affected.

Fig. 139.—Multiple neuritis after diphtheria in a child four years old. The position of the head and spine are due to partial paralysis of the trunk and neck. The legs were also affected.

Ataxia is not uncommon, and may be a more striking symptom than the loss of power. All the reflexes are diminished or lost, especially the knee-jerk, as the legs are usually most affected. Sometimes, particularly after diphtheria, there is loss of the knee-jerk, when there is no other symptom of neuritis. In the severe cases muscular tremor is frequent.

Atrophy is a prominent symptom of neuritis, and it is evident early in the disease, often being quite as rapid as in poliomyelitis. The electrical reactions are altered,—every grade of reduction in the responses being seen, from a slight diminution in the reaction to faradism to the complete reaction of degeneration. Vaso-motor symptoms, such as œdema of the affected parts, glossiness of the skin, etc., are often present. Deformities from muscular contraction occur early; they may be severe, and in some cases, permanent.

Course and Prognosis.—The usual course of the disease is for the symptoms gradually to increase for three or four weeks and then improve,

sometimes rapidly, but more often slowly, the case usually going on to complete recovery in the course of a few months. Exceptionally the paralysis may be permanent. The sensory symptoms always disappear before the motor ones. Multiple neuritis may prove fatal, from paralysis of the heart or the muscles of respiration, or death may be due to asphyxia from the entrance of food or foreign bodies into the air passages, owing to anæsthesia of the epiglottis and paralysis of the muscles of deglutition. Death sometimes follows from complications, especially pneumonia. The electrical reactions are of much prognostic value in regard to the persistence of the paralysis. If the reaction of degeneration is present the paralysis is certain to last many months, and some muscles are sure to be permanently affected. Where there is simply a diminution in the faradic responses, even though accompanied by marked atrophy, complete recovery may be expected, although it is often slow.

Diagnosis.—The diagnostic features of multiple neuritis are the combination of motor and sensory symptoms with the same distribution, the occurrence of atrophy, and the diminution in the electrical responses, even the reaction of degeneration. The gradual onset and the wide-spread distribution of the paralysis are also characteristic. If all four extremities are paralyzed, it is altogether the probable disease; and if to this is added paralysis of the neck and spinal muscles, the diagnosis is almost certain. The facts that the paralysis is often incomplete, and that it involves parts distant from each other, are also important. It may be mistaken for poliomyelitis (page 776), for Landry's paralysis, or for Pott's paraplegia; an important diagnostic point from the last mentioned is the condition of the reflexes,—being greatly exaggerated in Pott's paraplegia, while they are diminished or lost in multiple neuritis.

Treatment.—As this disease tends in the great majority of cases to spontaneous recovery, it is difficult to estimate the value of any method of treatment. Causes, such as lead, arsenic, alcohol, and malaria, are to be sought and removed as the first step. During the acute stage the pain may be so severe as to require relief, which is best accomplished by the application of heat. In using counter-irritation care is necessary, and such active measures as cauterization should not be employed, for troublesome ulceration may follow. After the acute stage has passed, or at the end of three or four weeks, electricity should be begun, faradism being used if the muscles respond to a moderate current, otherwise galvanism. This should be continued daily until recovery. Strychnine is much used in these cases, but it is doubtful whether it has any specific influence, although as a tonic it is valuable. Other tonics, such as iron, quinine, and most of all cod-liver oil, should be given in every case. Massage is also beneficial. The special treatment of cardiac and respiratory paralysis will be discussed in the following article.

DIPHTHERITIC PARALYSIS.

This is not only the most frequent variety of multiple neuritis, but it has some peculiarities which make a separate consideration of it desirable.

Frequency.—According to the statistics of various observers, paralysis including all varieties, occurs after diphtheria in from 5 to 15 per cent of the cases. Sanné gives 11 per cent in 2,448 cases; Lennox Browne, 14 per cent in 1,000 cases; the Report of the Collective Investigation by the American Pædiatric Society, 9·7 per cent of 3,384 cases which were treated by antitoxine.

It is as yet too soon to state to what degree the frequency of paralytic sequelæ after diphtheria is to be affected by the antitoxine treatment; but the figures above given would indicate that the protective power of the serum over the nervous tissues is not so great as is seen elsewhere, and that unless administered very early it may have little or no influence.

Being one of the direct effects of the diphtheria toxine, neuritis is much more likely to follow severe than mild cases; however, its occurrence after some very mild attacks shows how great is the susceptibility of the nervous tissues to the action of this poison. Sometimes the throat symptoms have been entirely overlooked, and the development of paralysis has been the first thing to arouse a suspicion of previous diphtheria.

Time of Occurrence.—During the second week, and sometimes even during the latter part of the first week, the early paralysis occurs, affecting the palate, and in some cases the heart. The most frequent and most characteristic paralysis—that affecting the throat, eyes, extremities, heart, or respiration—begins at a later period, usually from one to three weeks after the throat has cleared off, and sometimes even later than this.

Extent and Distribution of the Paralysis.—Ross * gives the following statistics of 171 collected cases of diphtheritic paralysis: Palate affected in 128; eyes in 77, in 54 of which the muscles of accommodation were involved; lower extremities in 113; upper extremities in 60; trunk or neck in 58; muscles of respiration in 33. I do not think this represents the actual frequency of the different varieties so truly as do the American Pædiatric Society's figures, which give the forms of paralysis noted in a series of cases collected for another purpose. In 328 cases of paralysis, the variety was mentioned in 189: in 124 the throat was affected; in 22 the extremities; in 11 the eyes; in 5 the muscles of respiration; in 32 the heart; in 1 the neck only; in 8 the paralysis was "general."

Symptoms.—In the great majority of cases the throat is affected, and usually the paralysis is first noticed there. It may involve the palate

* The Medical Chronicle, December, 1890.

alone, or the muscles of the pharynx or larynx in addition. The muscles of the extremities or of the eye are often next attacked. In severe cases there may also be involved the muscles of the trunk and neck, and sometimes the diaphragm. Cardiac paralysis not infrequently occurs where no other parts have been previously affected, but in nearly all the other forms, the throat symptoms precede. It is this which distinguishes diphtheritic paralysis from other forms of multiple neuritis. Whatever the extent or situation of the paralysis, the knee-jerk is nearly always lost. The symptoms in the extremities and the trunk do not differ from those of multiple neuritis from other causes. The throat paralysis shows itself by a nasal voice and by regurgitation of fluids through the nose, sometimes by difficulty in swallowing or the entrance of food into the larynx, owing to anæsthesia of the epiglottis and paralysis of the muscles of deglutition. There may be difficulty in protruding the tongue or in articulation. Paralysis of the vocal cords may cause hoarseness, aphonia, or attacks of spasmodic dyspnœa. Facial paralysis is very rare. On the part of the eye there is most frequently seen inability to read, owing to paralysis of the muscles of accommodation; there may be dilatation of the pupils, rarely strabismus or ptosis.

Next to that of the throat, paralysis of the muscles of respiration and the heart are the most characteristic forms of diphtheritic neuritis. Respiratory paralysis may be due to involvement of the phrenic or the intercostal nerves, most frequently the former. Extensive paralysis of other parts—the throat, extremities, or trunk—usually precedes. The first warning is generally in the form of occasional attacks of dyspnœa, sometimes accompanied by cough. Gradually these attacks increase in frequency and severity. The voice is reduced to a whisper. As the diaphragm is usually affected, the breathing is entirely thoracic. The respiratory movements are rapid, but irregular, shallow, and ineffectual. There is cyanosis, also great subjective as well as objective dyspnœa. The anxiety, distress, and apprehension of the patient are sometimes terrible. There is a constant dread of impending suffocation, and the respiratory movements are continued only by the patient's constant efforts, otherwise they may cease altogether. After a few hours these severe symptoms may subside, to return after a short respite. There may be several such attacks during two or three days, in each of which death seems imminent. Unfortunately, this is the most frequent termination. Of thirty-three such cases collected by Ross, only eight recovered. Associated with these respiratory symptoms others may be present, indicating that the pneumogastric is involved. There may be attacks of abdominal pain, vomiting, and disturbance of the heart's action,—usually an irregular or intermittent pulse, which may be either unnaturally slow or very rapid. In many cases the heart continues to beat normally, even though the respiration is so much disturbed.

The premonitory symptoms of cardiac paralysis are an irregular or

intermittent pulse, often slow, but becoming very rapid from even the slightest exertion. It is always weak and compressible. The first sound of the heart is feeble and may be reduplicated. As the symptoms increase there are marked pallor, coldness of the extremities, great restlessness, anxiety, precordial distress, and perhaps orthopnœa. Within twenty-four hours from the beginning of such symptoms death usually occurs. In other cases it may come suddenly without any warning, or with a warning so slight as to be overlooked. At such times it often follows some muscular exertion, such as getting out of bed, walking across the room, or so slight an effort as sitting up suddenly in bed. Fits of temper or other excitement have at times produced it. It is by no means certain that sudden heart paralysis is always due to a lesion of its nerves. A not less important cause is toxic myocarditis. In the cases where death occurs suddenly without premonition after some muscular effort, it is in all probability the heart muscle which is most at fault. However, in many cases the two conditions are associated.

Death from diphtheritic paralysis is usually due either to cardiac or respiratory paralysis. Of one hundred and seventy-one cases of all varieties collected by Ross, forty-five were fatal.

Treatment.—Cases of paralysis of the trunk or extremities are to be managed like others of multiple neuritis. In severe forms of throat paralysis feeding by a stomach tube should always be employed, on account of the danger of the entrance of food into the air passages. It must in most cases be continued for several days. The tube may be passed either through the mouth or the nose.

The great mortality attending paralysis of the heart and respiration shows how unsuccessful is treatment in most of the cases; still, no doubt there are instances where life may be saved by judicious treatment. In cases of threatened heart paralysis, the drug most to be depended upon is morphine, hypodermically; this should be used every two or three hours in sufficient doses to keep the patient under its influence while threatening symptoms are present. In some cases it may be advantageously combined with strychnine. The patient should be kept absolutely quiet, not even being allowed to turn in bed. In respiratory paralysis the general reliance is upon strychnine used hypodermically in doses sufficient to produce its physiological effects, and upon faradization of the respiratory muscles, particularly the diaphragm. Faradism is to be used in the attacks of respiratory failure and continued while they last. In some cases patients may by these means be tided over the dangerous stage of the disease.

FACIAL PARALYSIS.

Peripheral paralysis of the face occurring as a result of injury inflicted during delivery has already been described (page 108). There remain to

be considered here cases which arise from causes that operate at a later period. The facial nerve may be affected in any one of three situations,—after its exit from the cranium, in the bony canal, and within the cranium.

In the first situation, the principal cause of neuritis is exposure to cold (the "rheumatic" cases), but it occasionally occurs as a complication of mumps and disease of the lymph glands of this region. The nerve is affected just after it has escaped from the stylo-mastoid foramen, and all the branches given off beyond its exit are involved. There is paralysis of the muscles of the forehead, those about the eye, the cheek, nose, and mouth. The affected side of the face is smooth, there is inability to wrinkle the forehead, contract the eyebrows, close the eye completely, raise the nostril, whistle or blow. The mouth is drawn to the affected side (Fig. 140). If the paralysis is complete, there may be difficulty in drinking or in articulation. In partial paralysis the symptoms may not be noticeable while the face is at rest. There are no sensory symptoms. The electrical reactions resemble those of other forms of neuritis; there is diminution in the response to the faradic current, which is more or less marked according to the severity of the lesion, and there may be the reaction of degeneration.

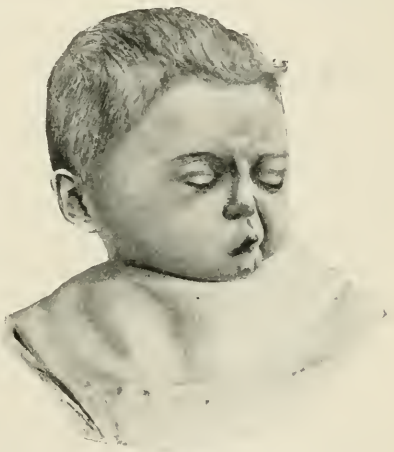

Fig. 140.—Facial paralysis from middle-ear disease in a child two and a half years old.

In the bony canal, the facial nerve is usually inflamed as a result of disease of the ear. In children this is much more frequent than from the causes just mentioned. While it is possible for it to occur in acute cases, it generally accompanies chronic otitis, especially where there is caries of the petrous bone. In addition to the paralysis there is present or there is a history of a discharge from the ear, and generally there is some deafness upon the side affected. The facial symptoms are usually the same as in the cases first described. However, when the nerve is affected between the stapedius and the geniculate ganglion, there is a disturbance of the sense of taste, and of the secretion of the saliva.

At the base of the brain the trunk of the nerve may be involved in cerebral tumour, basilar meningitis, and in fracture of the skull. In any of these conditions the auditory nerve also is likely to be affected.

Prognosis.—The result is greatly modified by the cause in the different cases. In those which are due to cold, spontaneous recovery usually occurs in the course of a few weeks or months. In those depend-

ing upon disease of the ear, the outlook is not so favourable, and though there may be improvement, it is not rare for some paralysis to be permanent. In the third group of cases, facial paralysis is only one of the symptoms, and the result depends entirely upon the nature of the cause.

Diagnosis.—Facial paralysis is easily recognised. It is important to separate the peripheral paralysis from that due to a lesion above the pons, as in cases of ordinary hemiplegia. In the latter group only the lower half of the face is affected, the muscles of the forehead and those about the eye escaping, and the electrical reactions are unchanged.

Treatment.—This is essentially the same as in other cases of neuritis. In cases due to ear disease the primary lesion should receive appropriate treatment.

SECTION VIII.

DISEASES OF THE BLOOD, LYMPH NODES, BONES, ETC.

CHAPTER I.

DISEASES OF THE BLOOD.

In general, the blood in infancy and childhood, as compared with that of adult life, is thinner and contains a larger proportion of water; it is also poorer in solids and has a lower specific gravity.

Specific Gravity.—This has no constant relation to the number of white or red corpuscles, but varies with the amount of hæmoglobin. The highest specific gravity is seen in the blood of the newly born, when, according to Lloyd-Jones, it is 1·066. During the first two weeks of life it sinks rapidly to its lowest point—1·048 to 1·052—where it remains until about the end of the second year; after this time it rises gradually until about puberty. The average specific gravity during childhood is 1·052 to 1·055 (Hock and Schlesinger).

Hæmoglobin.—The percentage of hæmoglobin is highest in the blood of the newly born, and falls rapidly during the first few days after birth. Throughout childhood it is considerably lower than in adult life. The hæmoglobin is lowest between the third month and the fifth year; after the fifth year it gradually increases up to puberty. According to Wydowitz, the usual range in infants and young children, as measured by the adult standard, is between 60 and 80 per cent, 60 per cent being the lowest limit in healthy children.

The cells of the blood are the red corpuscles or erythrocytes, and the white corpuscles or leucocytes.

Red Corpuscles.—The number of red corpuscles is highest in the newly born. At this time it is from 4,350,000 to 6,500,000 in each cubic millimetre. In infancy it is from 4,000,000 to 5,500,000; in later childhood, from 4,000,000 to 4,500,000 (Hayem). In size a much greater variation is seen in the red cells of the newly born than in those of older children and adults. In the blood of the fœtus there are present nucleated red corpuscles or erythroblasts (Plate XVI, A, 5, and B, 2). These diminish in number toward the end of pregnancy. They are always found in the blood of premature infants, but in infants born at term they are seen only

in small numbers and disappear after a few days. In later infancy their presence is always pathological.

White Corpuscles.—Of these, five different varieties are distinguished by Ehrlich:

1. *Lymphocytes or small mononuclear cells* (Plate XVI, A, 6). These resemble the red blood-cells in size, and have a single deeply staining nucleus, which is so large as nearly to fill the cell body; the protoplasm is non-granular. The source of these cells is believed to be the lymph glands.

2. *Large mononuclear cells.* These are much larger than the preceding variety, and have a single large ovoid nucleus with quite a broad margin of protoplasm surrounding it. They are not numerous in normal blood; they are derived from bone-marrow and the spleen.

3. *Mononuclear transition forms.* These are derived from the variety last mentioned, being similar in size and colour. The nucleus shows an indentation on one side—the beginning of a nuclear division. When further developed, these cells show traces of neutrophile granulations in the protoplasm, usually between the horns of the nucleus.

4. *Polynuclear cells with neutrophile granulations* (Plate XVI, A, 3). The nucleus is long, irregular, and twisted in various shapes or divided into several parts. The protoplasm contains fine granulations affected only by stains of neutral reaction. These cells are smaller than the mononuclear forms from which they are derived, although somewhat larger than the red cells. They constitute the largest proportion of the leucocytes in normal blood, and they are the only forms increased in ordinary leucocytosis. Forms 2, 3, and 4 probably represent different degrees of development of the same cells.*

5. *Eosinophile cells* (Plate XVI, A, 1). These are not related to any of the preceding forms. The protoplasm contains large fat-like granulations, which can be seen even before staining. They stain readily with acid colors, especially with eosin, from which peculiarity their name is derived. The granulations of these cells are much coarser than those of the polynuclear neutrophile cells, while their nuclei, of which there are generally two or three, do not stain so darkly. After the eosinophile cells have broken down, the resulting granulations somewhat resemble groups of cocci. In normal blood these cells form but a small proportion of the leucocytes.

The number of leucocytes in the blood of the newly born is three or four times that of the adult, being on the average 18,000 per cubic millimetre (Hayem). The variations during later childhood are from 6,000 to 12,000.

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* In Uskow's classification these are derived as "ripe" and "over-ripe" cells from the lymphocyte, which is regarded as the young or "unripe" cell.

PLATE XVI.

Fig. A.

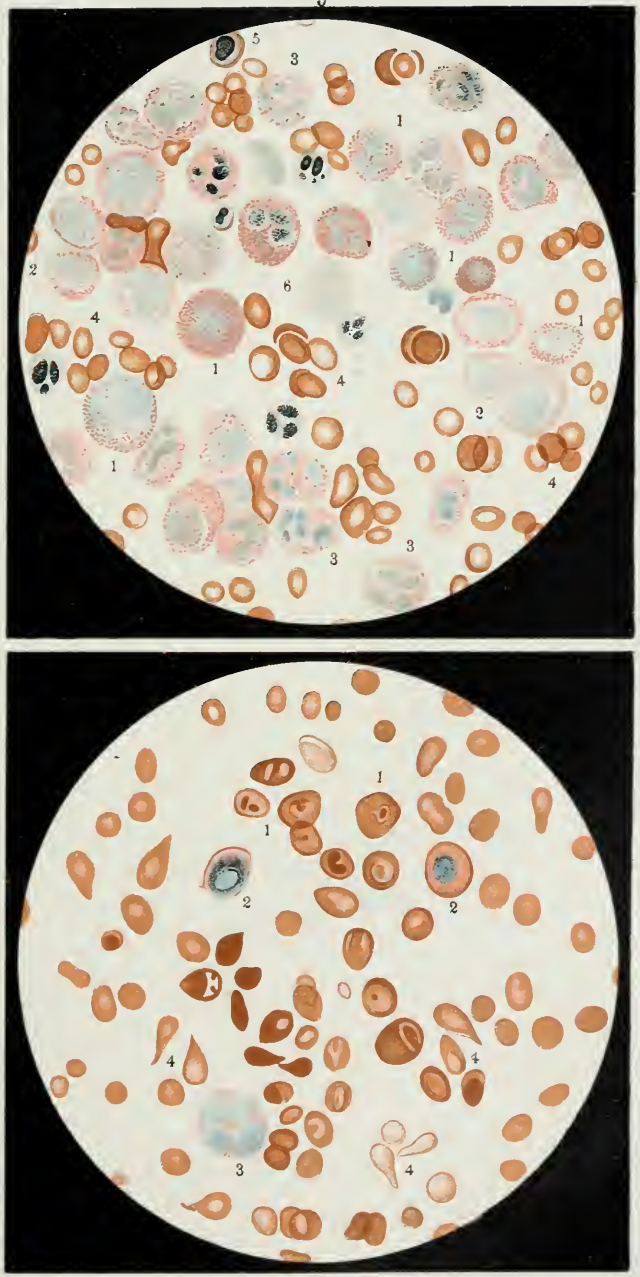

Fig. B.

A. The Blood in Leucæmia.

1, Eosinophile cells; 2, myelocytes; 3, polynuclear neutrophile cells; 4, red cells; 5, nucleated red cells; 6, lymphocytes.

B. Pernicious Anæmia.

1, Megaloblasts; 2, nucleated megaloblasts; 3, a polynuclear neutrophile cell; 4, poikilocytes. (After Monti and Berggrün.)

The white cells may be said to be increased—i. e., leucocytosis exists—when their proportion to the red cells is greater than 1 to 200. It is not yet possible to state the exact percentages of the different varieties of white cells in normal blood. The polynuclear cells are, however, the most numerous, the lymphocytes next, and the eosinophile cells least frequent.

Before leaving the subject of the cells of the blood the so-called *blood-shadows* deserve a brief mention. These, according to Silbermann, are common in the blood of the newly born, but diminish with the age of the child. They contain no hæmoglobin. The existence of such cells is denied by some observers, who regard the appearance as due to the preparation of the specimen.

The following are the principal peculiarities in the blood of the newly born: The specific gravity and the hæmoglobin are high. The number of red cells is considerably higher than the average during childhood, and the same is true to a less degree of the leucocytes. The red cells vary much in size. They show less tendency to form rouleaux, although this is denied by some observers. Nucleated red cells, erythroblasts, are found for a day or two in small numbers, and the blood-shadows of Silbermann may be present.

It is only within the last few years that the diseases of the blood have been studied with anything like scientific accuracy. With our present knowledge it is difficult to classify accurately the various forms of anæmia. The essential character and the relation of the different forms to one another, are matters upon which there is still much difference of opinion among good observers. The classification here presented is that which has received the most general adoption, and may be accepted as a provisional one. With reference to the nicer points, most of the observations made prior to 1885 must be taken with considerable allowance.

SIMPLE ANÆMIA.

This consists in an impoverishment of the blood, especially the red cells, and a corresponding diminution in the specific gravity and in the amount of hæmoglobin. It is essentially a secondary anæmia, and occurs apart from disease of the blood-making organs. The important factors in its etiology are, first, an insufficient production of blood in consequence of deficient food or interference with the absorption of food, and, second, an increased drain or destruction of blood, as in exhausting diseases. Infancy and childhood are themselves strong predisposing causes of anæmia, on account of the great demands made upon the blood in the rapid growth of the body.

Etiology.—In certain cases anæmia may be congenital, as in infants born of delicate or anæmic parents, or where the mother during pregnancy has suffered from some serious disease, such as syphilis or nephritis. Acquired anæmia may come on at any period in infancy or childhood. The

cause may be loss of blood, as in hæmorrhages of the newly born, epistaxis, purpura, scurvy, or hæmophilia. None of these are very common etiological factors. More frequently anæmia depends upon a loss of albumin of the blood, as in prolonged suppuration, chronic nephritis, large serous effusions occurring in the course of cardiac disease, certain forms of diarrhœa, and in malignant disease. Very frequently also it depends upon improper food, or disease of the organs of digestion or assimilation, as in the various forms of chronic diarrhœa, ileo-colitis, or chronic indigestion. These cases form a group sometimes classed as anæmia from inanition. In infancy, unhygienic surroundings, bad air, and close confinement to unhealthy apartments, are important factors in producing anæmia. In a large number of cases the anæmia is of toxic origin. In this group may be classed not only cases in which anæmia depends upon mineral poisons introduced into the body, such as mercury or chlorate of potassium, but also the poisons of all the infectious diseases, notably diphtheria. Febrile anæmia is not entirely due to toxic causes. It depends in part, no doubt, upon interference with digestion and assimilation. Anæmia may be due to parasites in the blood, the most striking illustration being the plasmodium malariæ, and it may occasionally arise from some forms of intestinal worms. The etiology of the anæmia accompanying certain constitutional diseases, such as rickets, tuberculosis, or rheumatism, is of a complex character.

Symptoms.—One of the most striking symptoms is the pallor of the skin and mucous membranes, although this is by no means an infallible guide to the degree of anæmia. Such children usually exhibit also symptoms of malnutrition: their muscles are soft and flabby; they are frequently thin and poorly nourished, but occasionally have an unusual amount of fat. They almost invariably suffer from digestive disturbances, such as coated tongue, poor appetite, and constipated bowels. The extremities are often cold, the pulse is rather weak and often slightly irregular. The heart-sounds are feeble, and anæmic murmurs may be heard either over the heart or the large vessels even in infancy, and occasionally a venous hum may be heard in the neck. In a certain number of cases of moderate severity there is found enlargement of the spleen, but rarely to the degree seen in leucæmia, or in the pseudo-leucæmia of infants. These cases were formerly classed separately as "splenic anæmia."

Nervous symptoms are frequent. Anæmic children are fretful, irritable, and often exhibit a degree of nervousness amounting almost to chorea. Others complain of headache and indefinite pains. Sleep is restless and disturbed, and often there is insomnia. The urine is scanty, frequently pale, and in many cases contains an excess of uric acid; there may be enuresis. Such children are easily fatigued, they frequently suffer from shortness of breath upon exercise, and occasionally have fainting attacks. They are especially prone to chronic catarrhal inflammations of the nose,

pharynx, and bronchi. Epistaxis is not an uncommon symptom. Leucorrhœa may be present even in girls of three or four years. Dropsy is not infrequent in infants, but is rather more common in older children. In infancy, if anæmia comes on rapidly, as in the course of diarrhœal diseases, cerebral symptoms may be present.

The blood.—The changes in the blood depend much upon the grade of anæmia. In the milder forms there is only a moderate diminution in the specific gravity (1·042 to 1·046), in the hæmoglobin (50 to 55 per cent), and in the number of red cells, with very slight changes in their form or size. There is no increase in the leucocytes, although they are relatively more numerous on account of the reduction in the number of red corpuscles.

In more severe cases the hæmoglobin may be reduced to 30 or even 20 per cent, the specific gravity to 1·038 or lower, and the number of red cells to less than half the normal. In cases of such severity quite marked changes are usually present in their size and form. Microcytes, megalocytes, poikilocytes, and nuclear red cells (Plate XVI) may be present. The leucocytes in many cases show only a relative increase; in others they are actually increased, and may be twice as numerous as normal. Cases of this severity are to be considered, according to Monti and Berggrün, as intermediate between simple and pseudo-leucæmic anæmia.

Prognosis.—The course and termination of anæmia depend upon its cause. If this can be removed, steady improvement and recovery may be expected. In extreme cases death may take place, but rarely from the anæmia, usually from some complicating disease.

In making a prognosis there must be considered not only the general symptoms and the cause of the anæmia, but also the condition of the blood. If there is only a moderate reduction in the hæmoglobin and in the number of the red cells, with slight changes in their form and with no increase in the leucocytes, the prognosis is good. If the hæmoglobin is reduced below 30 per cent, if the number of red cells is less than half the normal, and marked changes in form are present, with or without great increase in the actual number of leucocytes, the prognosis is less favourable.

The treatment of all the forms of anæmia will be considered together at the close of the chapter.

CHLOROSIS.

Chlorosis is a primary or essential anæmia which usually occurs in young girls about the time of puberty. It is characterized by a peculiar greenish-yellow tint of the skin, and is not accompanied by emaciation. The changes in the blood consist in a very great reduction in the hæmoglobin without a corresponding diminution in the red corpuscles.

Etiology.—The exact cause of chlorosis is not yet fully understood.

The disease rarely occurs in males, the great majority of the cases being in girls between the fourteenth and seventeenth years, and more often in blondes than in brunettes. Heredity appears to be a factor in a considerable number of the cases. Among the other causes may be mentioned occupations deleterious to health, such as employment in factories or confinement in ill-ventilated rooms; insufficient food or clothing; psychical disturbances, like grief, care, or fright; excessive mental or physical strain; and disorders of menstruation—although the latter are perhaps more frequently a result than a cause of the disease. Virchow first called attention to the fact that chlorosis might depend upon a congenital narrowing of the aorta, sometimes associated with a small heart. It is difficult to reconcile this etiology with the rapid recovery under appropriate treatment which is seen in most of the cases. Andrew Clark has advanced the view that the chief cause of chlorosis is constipation and the resulting absorption of toxic materials from the intestine. The intestinal origin of the disease has been lately urged with a good deal of force by Forchheimer.

Lesions.—Chlorosis is rarely fatal. In the few fatal cases the lesions noted have been dilatation of the right heart with hypertrophy of the left ventricle, a small aorta, small uterus and ovaries, and occasionally round ulcer of the stomach. Under the microscope there may be found a very marked degree of fatty degeneration of the heart muscle, and sometimes of the inner coat of the blood-vessels.

Symptoms.—The general symptoms of chlorosis are very like those of simple anæmia. There are observed shortness of breath upon exercise, palpitation, syncope, attacks of vertigo, disturbances of digestion, amenorrhœa, and almost invariably constipation. The appetite is capricious, it being a peculiarity of these patients to crave all sorts of indigestible articles. Instead of the usual pallor of anæmia, the skin has a yellowish-green tint, from which the term "green-sickness" has arisen. Occasionally patches of pigmentation are seen. Anæmic cardiac murmurs may be heard in various situations, most frequently a systolic murmur at the base of the heart, and usually loudest over the pulmonic area. There may be a venous hum in the neck. In some marked cases there is evidence of slight cardiac dilatation, especially of the right heart, and there may be hypertrophy of the left ventricle. The pulse is weak and soft, œdema of the feet is frequent, and sometimes there is slight albuminuria. In some cases there is fever. Nervous disturbances, such as vague, indefinite pains, attacks of migraine, supra-orbital neuralgia, various hysterical manifestations, and chorea, are common. Ulcer of the stomach is sometimes seen as a complication.

The blood.—The blood changes in chlorosis are quite constant. The red corpuscles may be normal or but slightly diminished in number. In many cases but little variation from the normal size is seen; in others there are microcytes, megalocytes, and poikilocytes. The red corpuscles

have an unusually pale colour. The number of leucocytes is normal or very slightly increased. The hæmoglobin is uniformly reduced, usually to a great degree. Osler gives 44·1 per cent as the average in forty cases.

Prognosis.—The course of the disease is essentially a chronic one, often lasting for a year. Relapses are quite frequent. Except when dependent upon congenital malformations of the heart and blood-vessels, these cases regularly recover when proper treatment can be carried out. A small number prove fatal by the development of tuberculosis or the occurrence of gastric ulcer.

Diagnosis.—The diagnosis is in most cases easily made from the etiology, the functional derangement of the heart, the colour of the skin, and a positive diagnosis always by an examination of the blood.

PSEUDO-LEUCÆMIC ANÆMIA OF INFANCY.

This form of anæmia was first described by Von Jaksch in 1889, and is believed to be peculiar to infants and young children. It is characterized by marked leucocytosis, marked reduction in the number of red corpuscles and in the hæmoglobin, a great enlargement of the spleen, and sometimes a moderate enlargement of the liver and the lymphatic glands. This disease is not to be confounded with the pseudo-leucæmia of adults, or Hodgkin's disease, which is purely a disease of the lymphatic glands with secondary anæmia, but without any leucocytosis.

Etiology.—Of the cases thus far recorded the majority have been between the ages of seven and twelve months, the oldest being at three and a half years. Of twenty cases collected by Monti and Berggrün,* sixteen showed evidences of rickets and one was syphilitic. Pseudo-leucæmia, however, appears to occur in this disease only when the splenic enlargement has reached a certain grade. The exact cause of the disease is still unknown, and its essential nature is a matter of some doubt. Monti believes that it may develop from the more severe cases of anæmia which are accompanied by leucocytosis, as he has observed this condition before the development of pseudo-leucæmia and during its subsidence. The disease may terminate in ordinary leucæmia, and possibly in pernicious anæmia.

Lesions.—The most characteristic change is found in the spleen. This organ is very much enlarged, often forming an abdominal tumour, which extends as low as the crest of the ilium and as far forward as the umbilicus. It is firm, hard, the surface appears somewhat wrinkled, and there may be evidences of perisplenitis. The microscope shows an increase of cellular elements, a few cells containing hæmoglobin (Luzet).† Enlargement of the liver is less constant, it being normal in more than half the cases. There is no relation between the size of the spleen and that of the liver.

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* Die chronische Anämie im Kindesalter, Leipsic, 1892. † Thèse, Paris, 1891.

The hepatic cells are unchanged. Enlargement of the lymph glands has been noted in about half the reported cases, the swelling affecting the cervical, axillary, or inguinal glands; but it is rarely great. A moist appearance and a diffuse redness of the bone-marrow have been described by Luzet, the changes being usually most marked about the epiphyses.

Symptoms.—*The blood.*—The number of reported cases is as yet too small to make positive statements possible upon all points. The most constant features noted thus far are the following:

The specific gravity is lowered, the usual range being between 1·035 and 1·044. The reduction of the hæmoglobin is very great; in many of the cases it has been as low as 30 per cent, and in a few below 25 per cent. The leucocytes are increased in number, this being one of the striking features of the disease. In ordinary cases the proportion of leucocytes to red corpuscles is 1 to 100 or 1 to 75. In severe cases the proportion may be as high as 1 to 20 or even as 1 to 12. All the usual varieties of leucocytes are seen, the proportions of these varying much in the different cases. The red corpuscles are reduced in number in proportion to the severity of the disease, usually to from 65 to 75 per cent, but they may be as low as 35 or even 25 per cent. In six of twenty cases the actual number was below 1,600,000 (Monti and Berggrün). More characteristic than any of the above features are the changes in the appearance of the red cells. Very marked inequality in their size and shape is seen in most of the cases. Many microcytes are present; also great numbers of nuclear red blood-cells (erythroblasts), normoblasts, and megaloblasts with dividing nuclei. These are seen to some degree in other forms of anæmia, particularly in the pernicious variety and in the severe types of simple anæmia, but they are more abundant in pseudo-leucæmia. The larger the proportion in which they are present the worse the prognosis. Finally, there is occasionally seen a division of the nuclei of the red cells (karyokinesis), regarded by some as characteristic of the disease, although this is not admitted by all.

The general symptoms of the disease develop slowly and with the usual signs of anæmia. In some cases the infants continue to be plump and well nourished. Pallor is usually very marked. Enlargement of the spleen is so great that it can hardly be overlooked if the abdomen is examined. The glandular enlargements are not marked, and in many cases are wanting altogether.

The course of the disease is essentially chronic. Cases have been seen in which pseudo-leucæmia developed from an ordinary severe simple anæmia in the course of a few weeks. The symptoms and blood changes generally come on slowly in the course of weeks or months, and sometimes remain nearly stationary for as long a period as several months, and then slowly improve. In other cases they grow gradually worse, and the changes in the blood come to be the same as in ordinary leucæmia. Some

observers are inclined to believe that the disease is really an early stage of leucæmia, which does not reach its full development because the children succumb too early. In the cases going on to recovery, there is noticed improvement in the general symptoms coincident with a diminution in the size of the spleen, a reduction in the number of leucocytes, an increase in the red corpuscles, the hæmoglobin, and the specific gravity, and a gradual disappearance of the erythroblasts.

Prognosis.—In Monti's list of twenty cases four proved fatal; one recovered, in which the proportion of leucocytes to the red corpuscles had been 1 to 12. The prognosis should always be guarded, for, although improvement may take place, patients are very apt to be carried off by intercurrent disease.

Diagnosis.—The diagnosis is to be made from simple anæmia with leucocytosis, and from leucæmia. In simple anæmia the leucocytosis is not so great, and it is not accompanied by such a degree of splenic enlargement. In leucæmia the reduction in the red cells and in the hæmoglobin is very rarely as great as in pseudo-leucæmia.

PERNICIOUS ANÆMIA.

This is the most severe form of anæmia known. Its cause and essential nature are as yet very imperfectly understood. It is characterized by quite uniform blood changes and by the general symptoms of a very marked anæmia, and it tends to go on from bad to worse, terminating fatally in the great proportion of cases.

Etiology.—Pernicious anæmia is a rare disease in childhood, and especially rare in infancy. In the cases which have been observed in early life the following etiological factors have been noted: It has been associated with hereditary syphilis and with severe rickets, especially when accompanied by a marked enlargement of the spleen. It has followed other diseases, especially grave disturbances of nutrition. Sometimes simple anæmia, when severe and of long standing, has gradually developed into the pernicious type. In a few instances parasites, particularly tapeworms, have been the cause. Pernicious anæmia has in some instances occurred in patients where no cause whatever could be assigned.

Many theories have been advanced in explanation of pernicious anæmia. The one which at present appears to have most in its favour is that the disease consists in a great destruction of the red blood-cells, particularly in the liver, and that this is brought about through the agency of some poison or poisons taken up from the intestine by the portal circulation.* This has been advanced by Hunter and others in explanation of the peculiar deposit of iron found in the hepatic cells.

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* For fuller discussion of this theory of pernicious anæmia, see Griffith and Burr, the Medical News, October 17, 1891.

Lesions.—There is found a very high grade of anæmia in all the internal organs, fatty degeneration of the heart and blood-vessels, and sometimes also of the liver and kidneys, with numerous capillary hæmorrhages in the various organs. The most characteristic post-mortem change, however, according to Hunter, consists in the deposit of iron in the hepatic cells. Its distribution is peculiar and unlike that seen in any other disease.

Symptoms.—*The blood.*—Both the specific gravity and hæmoglobin are much reduced, the latter usually below 25 per cent, and in several instances below 15 per cent, but the percentage is still distinctly greater than that of the red cells. One of the most striking changes is the great reduction in the number of the red blood-corpuscles, the number of which is lower than in any other form of anæmia, the reduction being greater than in the hæmoglobin. Very often the number has been reduced below 500,000 in a cubic millimetre. Marked inequality is seen in the distribution of the hæmoglobin in the red corpuscles, some being almost colourless while others are deeply stained. There is great variety in the size and form of the red cells, this generally being proportionate to the severity of the disease. There are found microcytes and poikilocytes, but especially characteristic is the large number of macrocytes. There are many nuclear red blood-corspucles, both normoblasts and megaloblasts (Plate XVI, B). The reduction in the number of the leucocytes is usually in proportion to that of the red corpuscles. This is a peculiar feature of this disease (Monti and Berggrün). In most of the other conditions attended by reduction in the number of red cells the leucocytes are relatively increased.

The general symptoms are those of a most intense anæmia. There is marked pallor of the skin and mucous membranes, with great weakness and prostration. Various anæmic heart murmurs are heard. There is dyspnœa, and usually the urine is scanty and of low specific gravity. There may or may not be emaciation. The late symptoms are hæmorrhages from the nose and other mucous membranes, subcutaneous ecchymoses with dropsy of the feet and ankles, and sometimes of the large serous cavities of the body, but without albuminuria. In many cases fever is present. This may be so high as to lead to the suspicion of some acute infectious process.

The course of the disease is chronic, it being in most cases several months. In some, however, the progress is so rapid that death may occur within two or three months from the beginning of marked symptoms. As a rule, the symptoms are steadily progressive until death occurs; the only exceptions being the cases in which the disease depends upon some intestinal parasite; here improvement and even recovery may occur.

Diagnosis.—This is to be made from other forms of anæmia only by the blood examination; the most important points with reference to red

corpuscles are the great reduction in their number, the unequal distribution of hæmoglobin, the marked irregularities in form and shape, and the presence of many large nuclear forms; with reference to the leucocytes, a reduction in number proportionate to that of the red cells.

Treatment of the Different Forms of Anæmia.—In *secondary anæmia* the thing of the first importance is to discover and treat the primary condition upon which the anæmia depends. In infancy, special attention should be given to diet and hygiene, particularly with reference to an abundant supply of fresh air. The whole manner of life of these patients must be carefully studied and managed according to the directions laid down in the chapter upon Malnutrition, with which condition, especially in infancy, a very large number of these cases are associated. The general treatment referred to is often more important than the administration of the preparations of iron, which, however, should never be omitted.

The preparations of iron available for infants are the Drees's albuminate, the pepto-manganate (Gude), the bitter wine, the malate and the citrate. The dose should be regulated according to the age of the child. Older children may take the same preparations as adults, especially Blaud's pills. Much benefit is seen from combining arsenic with iron, or from alternating the two. Arsenic should be used in conjunction with iron in every anæmia in which there is enlargement of the spleen or lymphatic glands. In addition to these remedies, cod-liver oil should be given throughout the entire cold season.

In *chlorosis* more decided results are seen from the use of iron than in any other form of anæmia. Blaud's pills are here the favourite method of administration, and are advantageously combined with small doses of nux vomica and aloin to overcome the tendency to constipation. Arsenic is useful in these cases also. Great benefit in chlorosis results from change of air and change of scene, thus removing the patient from all sources of nervous excitement or disturbance. The general condition, diet, and habits of life should also receive careful attention, particularly the condition of the bowels. The use of oxygen is a valuable adjuvant in the treatment of cases not yielding to iron alone. It is important that the administration of iron should be continued for several months after the disappearance of all symptoms, on account of the tendency to relapses.

In the *pseudo-leucæmic anæmia* of infants, arsenic is decidedly the most valuable drug, but should be given in combination with iron. Fowler's solution is the best preparation for infants; the dose should rarely be more than one drop, which should be repeated four or five times daily after feeding, and continued for a long time. The general treatment of these patients is the same as in cases of simple anæmia. When rickets is present cod-liver oil and phosphorus should be added.

In *pernicious anæmia*, arsenic offers a much better prospect of improvement than iron. Beginning with small doses, the amount should be

gradually increased up to the point of tolerance, very much as in cases of chorea.

In every case of anæmia the most careful attention should be given to the general condition, particularly guarding against exposure to cold and dampness. The feeble circulation of these patients renders them peculiarly susceptible. Caution should also be given against much muscular exercise. With a severe grade of anæmia very active exercise should be prohibited, and many of these patients do best when complete rest in bed, either for the entire time or for a considerable part of each day, is insisted upon. This applies to children of all ages.

LEUCÆMIA.

This is a disease in which the essential feature is a great increase in the number of leucocytes, with a moderate reduction in the number of red corpuscles, and the presence in the blood of cellular forms not found in other diseases.

Etiology.—Leucæmia is a rare disease in childhood, but has been seen even in early infancy. Its greater frequency in males holds good even in childhood. In a small number of cases heredity seems of some importance as an etiological factor. Leucæmia may follow syphilis, rickets, malaria, or even simple anæmia, or it may occur as a primary disease in children previously healthy. In the great majority of cases the cause is unknown.

Lesions.—The essential lesions of leucæmia are found in the spleen, the lymphatic glands, and the bone-marrow. In rare cases the most important changes are in the lymphatic glands, giving rise to the *lymphatic* form of leucæmia. In such cases the changes in the spleen or marrow may be slight or absent. Changes in the spleen and marrow are, however, usually associated, giving rise to what is known as the *spleno-myelogenous* form of the disease, which is the most frequent variety. The spleen is usually enormously enlarged, sometimes filling half the abdominal cavity. In the early stage it is soft, vascular, and of a dark-red colour; in the late stages it is firm and hard, and usually deeply fissured at its margin. There may be perisplenitis. On section, light-gray patches of lymphoid tissue may be seen scattered throughout the organ, and in some instances there may be wedge-shaped infarctions. The microscope shows thickening of the trabeculæ and deposits of lymphoid tissue, especially about the arteries. The bone-marrow is of a yellowish-green or dark-brown colour, and shows immense numbers of nuclear red corpuscles in all stages of development, and many cells corresponding to the myelocytes found in the blood. The lymphatic glands, when they are involved, are not so uniformly enlarged as is the spleen. Any of the external glands of the body may be affected, the cervical, axillary, and the inguinal, or the mesenteric, tracheo-bronchial, the tonsils, and even the lymph nodules of the small and large intestines. The changes in the glands are gen-

erally those of a simple hyperplasia. The liver is enlarged in very many of the cases, chiefly from an infiltration with lymphoid tissue, which may be diffuse or may occur in patches. Less frequently similar lymphoid masses are seen in other organs.

Symptoms.—*The blood* (Plate XVI, A).—In gross appearance the blood is paler than normal, and the clot of a yellowish-green colour. The fibrin is usually increased. Both the specific gravity and the hæmoglobin are diminished, the latter often being reduced to 25 per cent. The most important change is in the leucocytes. These are enormously increased, the proportion often being one to five of the red, and sometimes even one to two.

In the spleno-myelogenous variety the predominant form is the large mononuclear cells with neutrophile granules, and are known as *myelocytes* (A, 2). The presence of the neutrophile granules distinguishes them from other mononuclear cells. The source of these is the bone-marrow, and they are not found in the lymphatic variety of the disease. In addition there is often an increase in the eosinophile cells (A, 1). The lymphocytes are relatively diminished; the percentage of the polynuclear neutrophile cells (A, 3) is normal or diminished. The red corpuscles are moderately reduced in number, usually to from 30 to 50 per cent, and exhibit the irregularities in form and shape seen in other varieties of anæmia. There are also nuclear red-corpuscles present whose nuclei are sometimes undergoing division.

In the lymphatic form of the disease, the blood shows quite marked differences. The increase in the leucocytes is not so great, and is due solely to the increase in the number of lymphocytes, the myelocytes being absent. Occasionally both forms of the disease may be combined.

The other symptoms of leucæmia in children resemble those in adults, with the difference that, as a rule, the progress of the disease is much more rapid in early life. In most of the cases the early symptoms are latent. A sudden and alarming hæmorrhage is sometimes the first thing to call attention to the serious condition. In other cases there are only the symptoms of general weakness and anæmia. Sometimes the splenic tumour or the enlargement of the lymphatic glands is first noticed. In the early part of the disease, the usual symptoms of anæmia are present,—digestive disturbances, shortness of breath, weak and rapid pulse. Hæmorrhages may occur as an early or late symptom; they are most frequently from the nose, but severe hæmorrhages may occur from the stomach, the mouth, the intestines, or there may be ecchymoses upon the skin. The enlargement of the spleen may be sufficiently marked to form an abdominal tumour, so as to attract the attention even of the parents. The swelling of the liver is not so great. The lymphatic glands are enlarged only to a moderate degree, and in many cases this symptom is

absent altogether. They are painless, movable, and usually several groups are affected.

The late symptoms are dropsy of the feet or general anasarca, hæmorrhages, diarrhœa, headaches, general weakness, and attacks of fainting. Fever is quite constant in the late stages of the disease, and the temperature may be from 101° to 103° F. The urine may contain albumin and casts. Vision is sometimes disturbed by the formation of leucæmic plaques in the retina. It is rare that there are any symptoms referable to the bones, although expansion and tenderness of the flat bones have been observed.

Course and Prognosis.—The course of leucæmia is chronic, and in most cases slowly progressive, but not always so. The prognosis is very bad, the great proportion of the cases in children proving fatal within a year from the first symptoms, in infancy sometimes in two or three months. There has been described by Epstein and others an acute form of the disease, proving fatal in a few weeks. The usual causes of death are exhaustion, hæmorrhages, and broncho-pneumonia.

Diagnosis.—This, in children, has to be made chiefly from simple anæmia with leucocytosis, and pseudo-leucæmic anæmia. Without a blood examination this is impossible. Reliance is to be placed upon the enormous increase in the leucocytes, and especially upon the presence of myelocytes. In the other diseases mentioned there is simply an increase in the usual varieties of leucocytes; different forms may predominate in different cases, but no new ones are present.

Treatment.—The general treatment of leucæmia should be the same as that of anæmia. Of the drugs now in use, arsenic has altogether the most testimony in its favour. It must be given in large doses and for a long period. Next to this in value come iron and cod-liver oil. Leucæmia, however, is in most instances very little influenced by treatment. The reported cures must be taken with some allowance, for most of these were published before the time when leucæmia was sharply differentiated from simple anæmia with leucocytosis and from the pseudo-leucæmic anæmia of infancy.

HÆMOPHILIA.

Hæmophilia is an hereditary disease, in which there is a tendency to profuse or even uncontrollable bleeding from slight wounds, or sometimes even spontaneously. In many cases there is associated an inflammation of the joints. Persons so affected are known as "bleeders."

Etiology.—The hereditary tendency of the disease is very strongly marked, and it has often been traced through seven or eight generations. Males are much more frequently affected than females, the proportion being about twelve to one. In the matter of inheritance, the disease is most often transmitted through the mother, who may, however, herself escape.

Patients suffering from hæmophilia have nothing else about them that is abnormal. The exact nature of the disease is unknown. It has no connection with either purpura or scurvy. Although generally classed among the diseases of the blood, it has not been established that there are any constant changes either in the blood or in the blood-vessels.

Symptoms.—The first manifestations of hæmophilia are not often seen before the second year. The hæmorrhages of the newly born have no relation to this condition. The discovery of the disease is generally quite accidental. The first hæmorrhage may be traumatic or spontaneous. In traumatic hæmorrhages there may be very severe bleeding after so slight a wound as the drawing of a tooth; sometimes a large hæmatoma forms between the muscles as the result of a moderate contusion.

The following is the relative frequency of spontaneous hæmorrhages in 334 cases collected by Grandidier: bleeding from the nose in 169, mouth in 43, intestines in 36, stomach in 15, urethra in 16, lungs in 17. There may be hæmorrhage from the skin or from any mucous membrane of the body. The attacks of spontaneous hæmorrhage are often periodical, and may be accompanied by arthritic symptoms resembling rheumatism. The severity of the hæmorrhages varies much in the different cases. From a slight wound a patient may bleed until he is exsanguinated, and even until death occurs. Such a result from the first hæmorrhage, however, is rare. In some cases the disposition to bleed is outgrown in later life. Grandidier states that, of 152 boys, over one half died before reaching the seventh year. It is striking that when the disease affects females there is no tendency to excessive bleeding at menstruation or parturition.

Treatment.—The indications at the time of bleeding are, to arrest the hæmorrhage by the use of the ordinary surgical means—compression, styptics, etc.—and the nares should be plugged for severe epistaxis. Little benefit is to be expected from drugs. In convalescence after attacks of hæmorrhage, iron and general tonics should be given. In all patients who are bleeders everything which might by any means excite hæmorrhage should be avoided. Marriage should be discouraged in girls who inherit the disease.

PURPURA.

The term purpura is used to designate a condition in which there is a tendency to spontaneous hæmorrhages beneath the skin, from the various mucous membranes, and in some cases into the internal organs. The term *purpura simplex* is applied to those cases in which the hæmorrhages are limited to the skin; *purpura hæmorrhagica* to those in which there is in addition bleeding from the mucous membranes or visceral hæmorrhages. It is impossible to draw a line sharply between these two classes of cases, as the chief difference between them seems to be one of

degree. Purpura is sometimes known as *morbus maculosus* or as *Werlhof's disease.*

Symptomatic Purpura.—This occurs in quite a variety of conditions, the hæmorrhages generally being limited to the skin, but not always so. These cases may be grouped in the following classes:

1. *Infectious.*—This form of purpura is very constantly seen in malignant endocarditis, in the hæmorrhagic forms of the various eruptive fevers—measles, scarlet fever, variola, vaccinia, and typhus—also in epidemic meningitis and occasionally in diphtheria, pyæmia, and septicæmia. The occurrence of hæmorrhages in these cases appears to depend upon an altered condition of the blood, which is a direct result of the infection. In most of the diseases mentioned it is a bad prognostic sign, as it indicates a severe form of the disease, but it requires no special treatment.

2. *Cachectic.*—Purpura occurs late in the course of many protracted and exhausting diseases, especially in infancy. It is most frequently met with in broncho-pneumonia, empyema, tuberculosis, ileo-colitis, in both the tuberculous and the simple forms of meningitis, and in malignant disease. It also occurs from apparently similar causes in several of the diseases of the blood, particularly in leucæmia and pernicious anæmia, and occasionally it is seen in chronic nephritis and in cardiac disease. In most cases of cachectic purpura the hæmorrhagic spots are not very abundant, and occur either upon the abdomen or the lower extremities. They are usually small, but when once they have appeared new spots usually continue to come until death. This form is quite common in hospital practice, and when occurring in the course of the diseases mentioned it is almost invariably indicative of a fatal result. Cachectic purpura is usually limited to the skin, hæmorrhages from the mucous membranes being infrequent and visceral hæmorrhages very rare. The condition is undoubtedly dependent upon a deterioration in the blood possibly also upon the condition of the minute blood-vessels themselves. Purpura adds nothing to the severity of the original disease, but is an indication of how extensive the blood changes are. It requires no special treatment.

3. *Toxic.*—Certain drugs, such as phosphorus, quinine, potassium chlorate and sometimes others, may produce hæmorrhages when long continued or in large doses. The hæmorrhage of jaundice may also be considered in this group. All these conditions are extremely rare in childhood.

4. *Mechanical* hæmorrhages, such as those occurring in pertussis or epilepsy, are sometimes classed with purpura. In convalescence from protracted illness there are sometimes seen, when patients first stand or walk, purpuric spots on the lower extremities. I have seen it after diphtheria. It may occur after prolonged confinement of a limb in bandages or splints.

In both these cases the cause is partly mechanical and partly due to the weakened condition of the blood-vessels.

5. *Neurotic.*—These cases are occasionally seen in diseases of the spinal cord and sometimes in hysteria in young adults, but very rarely in children.

Primary Purpura.—This occurs in children of all ages, being not uncommon in infancy. Hæmorrhages of the newly born have not generally been included in this class, although there are some reasons why they might well be. The age at which primary purpura is most frequently seen is from two to ten years. The sexes are about equally affected; of Steffen's 56 cases, 27 were males and 29 females. The disease may occur in children who are cachectic, rachitic, or anæmic, and in those whose surroundings are poor, but it has not, like scurvy, any close relation to diet. It may follow any acute disease, being associated most frequently with derangements of the stomach and bowels. Quite frequently the disease develops abruptly, without any assignable cause, in children previously healthy. It is not contagious. Epidemics of purpura have been reported, but these are somewhat doubtful, as they were recorded before this disease was sharply differentiated from scurvy. The association of purpura with rheumatism will be considered later.

Lesions.—The external hæmorrhages may occur upon any part of the body. There are smaller or larger ecchymoses or an infiltration of the tissues with blood, which undergoes gradual absorption with the usual changes. With the hæmorrhages, various forms of inflammation of the skin may be associated, especially erythema and urticaria, with sometimes more or less œdema. Free bleeding from the skin is very rare. Hæmorrhages from the mucous membranes are more frequent, because of the feebler resistance of the tissues. There are seen ecchymoses upon the visible mucous membranes which resemble those upon the skin. At autopsy they are occasionally seen in the trachea or bronchi, but more often in the digestive tract. The stomach and intestines may contain dark, clotted blood, bloody mucus, or even fluid blood. In the colon, and occasionally in the small intestine, ulcers may be found; but they are rarely if ever seen in the stomach. They may be superficial or deep, and have even been known to cause perforation. The deep ulcers have generally been attributed to thrombosis. Ulcers are often absent where intestinal hæmorrhage has been severe. Associated with these lesions there may be inflammatory changes in the mucous membrane of the stomach and intestines.

Intracranial hæmorrhages are rare, and those which occur are usually meningeal. These may be extensive and sufficient to cause severe symptoms. In 1893 a case occurred in the New York Infant Asylum in an infant six months old, with an extensive meningeal hæmorrhage covering a large part of the brain. In Steffen's paper several such cases are mentioned.

Pulmonary hæmorrhages are not frequent. They generally occur as small ecchymoses just beneath the pleura. In one of my own cases, a hæmorrhagic area as large as a walnut was found in the lung at one apex. Ecchymoses are found beneath the pericardium; but endocarditis and pericarditis are extremely rare, probably occurring only in the rheumatic cases. Fatty degeneration, with some degree of dilatation of the heart, has been seen in some of the most protracted severe cases. The spleen is occasionally enlarged, but by no means uniformly so, and it may be the seat of hæmorrhages. The liver is normal, or the hepatic cells may be the seat of fatty degeneration.

While hæmaturia is one of the most frequent of the visceral hæmorrhages, severe nephritis is rare. Acute degeneration of the renal epithelium of the tubes is quite common. There may be punctiform hæmorrhages, and occasionally larger ones beneath the renal capsule. Ecchymoses may be found on the mucous membrane of the pelvis of the kidney. The suprarenal capsules may be the seat of extensive and even fatal hæmorrhage, as in Wolff's case in a child two and a half years old. In addition to these lesions, there may be effusions of a sero-sanguineous fluid into any of the large serous cavities, most frequently into the peritonæum. The articular lesions of purpura may be of a rheumatic character, with which purpura occurs as a complication; or there may be hæmorrhages into the tissues about the joint, or even into the joint itself,—usually the knee or elbow.

Thus far no constant or essential changes have been demonstrated in the blood, other than those which are due to hæmorrhages—viz., a moderate reduction in the hæmoglobin and the red corpuscles, with occasional irregularities in size and the appearance of erythroblasts. In the most severe cases there is a moderate degree of leucocytosis.

Pathology.—Why it is that under certain circumstances the blood-vessels will not hold their contents, it is difficult to understand. There have been described by Cassel, Riehl, Wilson, and others, changes in the small blood-vessels, usually a form of endarteritis. These changes are in all probability dependent upon some alteration in the blood itself. It is not necessary to assume a lesion in the blood-vessels, since we know that diseased blood may pass through even normal vessels. Henoch has suggested the vaso-motor origin of purpura, in which there is first a paralytic distention of the small vessels, followed by stasis, hæmorrhage, or œdema. In certain forms, as in malignant endocarditis, it is well established that the cause is an infectious thrombosis. Although the bacteriological examinations made thus far in purpura are not numerous enough to settle the question positively, there is little doubt that infection is the essential factor in other forms of the disease, particularly in the cases characterized by sudden onset, high temperature, and cerebral symptoms, and which run a rapidly fatal course. This may possibly be

true of most of the primary cases. At the present time the exact pathology of purpura is unknown. There are, no doubt, now included under this term, several diseases quite distinct from one another.

The clinical types.—1. The ordinary form.—In the mild cases the hæmorrhage is confined to the skin (purpura simplex), or it is accompanied by slight bleeding from the mucous membranes. There is usually some general indisposition of an indefinite character for a day or two before the purpuric spots are noticed; most frequently a disturbance of digestion with vomiting, diarrhœa, and sometimes slight fever. The hæmorrhages appear as small petechiæ, varying in size from a pin's head to a pea; usually first upon the lower extremities, but sometimes first upon the trunk, the face, or the upper extremities. There may be only a few widely scattered spots or the body may be covered. The colour is first a bright red, then purple, gradually fading in the course of a few days. New spots come as the old ones disappear, so that the amount of eruption may not diminish; often the spots come out in distinct crops. They do not disappear upon pressure.

The course of these cases is generally favourable, recovery taking place in from one to four weeks under the influence of general tonic treatment. Relapses are, however, very frequent, and such attacks may come at intervals of a few weeks or months for a considerable period. One must be guarded in giving an absolutely favourable prognosis even in cases of such severity, for it occasionally happens that in a patient, who for several days has had symptoms of mild purpura, there suddenly develop those of the most severe type with a rapidly fatal termination.

2. The severe form.—Such cases are characterized by hæmorrhages from the mucous membranes (purpura hæmorrhagica) from the outset. These may even appear before the spots upon the skin. The relative intensity of the two varies much in different cases. In severe attacks the petechial spots are more likely to appear suddenly, and large ecchymoses, varying in size from a pea to the palm of the hand, are more frequent. There may be bleeding from the nose, gums, mouth, or pharynx, and ecchymoses may be seen upon these mucous membranes, also upon the conjunctivæ. Vomiting of blood and bloody discharges from the bowels are quite frequent symptoms. The urine may contain enough blood to give it a bright-red colour. Less frequently there are seen hæmorrhages of the retina or choroid and from the female genitals. In one of my own cases there was almost continuous bleeding from one ear. Hæmoptysis and free bleeding from the skin are both rare. Cutaneous ecchymoses are increased by slight injuries, such as the pressure from a bandage or from scratching. Epistaxis may be copious enough to necessitate plugging of the nares. The amount of blood vomited is not often large; its source may be the stomach, the mouth or the pharynx. The blood in the stools is usually dark coloured, but there may be some bright-red blood even when there

are no ulcers present. In one of my cases so much blood was lost by the bowels as to produce the symptoms of a very marked cerebral anæmia. In certain cases the gastro-intestinal symptoms are very prominent, and there may be slight icterus. The discharge of blood from the stomach or intestine may be accompanied by very severe attacks of colic and tenesmus. In some of these cases there are pains and slight swelling of the joints. Renal symptoms are generally present. These attacks of pain with purpura and the discharge of blood, may come on paroxysmally every few days for a period of several weeks. They have been ascribed to thrombosis of the intestinal vessels. This is sometimes known as "Henoch's purpura."

Constitutional symptoms are present in most of the severe cases. There is usually fever, from 101° to 103° F., and sufficient prostration to keep the patient in bed. If the amount of blood lost is large, there are the usual symptoms of severe anæmia,—pallor, weak pulse, cold extremities, fainting attacks, and functional heart murmurs. The loss of blood may be sufficient to cause death, particularly in infants. Cerebral symptoms may depend upon anæmia or upon meningeal hæmorrhage. They are not frequent in this form of the disease. Œdema, especially of the face and feet, may exist without albuminuria, and albuminuria may be present in cases in which there is no renal hæmorrhage. The amount of albumin is generally small, and casts are rare.

In some of the cases beginning with severe general symptoms, and occasionally when the onset is mild, the patients after a few days pass into a typhoid condition with low delirium, great prostration, weak and irregular pulse, dry, cracked tongue, and high temperature. Such cases are almost always fatal. They are not to be confounded with ordinary typhoid fever complicated by purpura.

The course varies much in the different cases. It lasts from one to six weeks, the symptoms slowly subsiding, but often showing a strong tendency to recurrence. The prognosis depends upon the age of the patient, the extent of the hæmorrhage, and the presence or absence of septic symptoms.

3. The hyper-acute form (purpura fulminans).—This is a rare form, especially in young children. Its development is usually sudden with a chill, vomiting, marked prostration, and high temperature. The purpuric spots come out with great rapidity, and in the course of a few hours or a day they may be very extensive. In addition to the ordinary subcutaneous hæmorrhages, bloody vesicles may form upon the skin. In many cases the hæmorrhages are limited to the skin, the mucous membrane and the viscera escaping altogether. There is no tendency to gangrene. Cerebral symptoms are invariably present and usually prominent; there may be delirium, dulness, stupor, and finally coma. The spleen is apt to be enlarged. The urine is nearly always albuminous. This form of purpura

has all the characteristics of a general infectious disease, and it is almost invariably fatal. But little is as yet definitely known regarding its cause or its relation to the other forms.

4. The gangrenous form.—Sloughing is not common in purpura, but it is most often seen in the mucous membranes. Osler refers to two cases affecting the uvula. I once saw a slough which caused perforation of the soft palate. Wickham Legg reports a case with gangrene of the prepuce. The deep ulcers of the intestine which are seen in some of the severe cases are apparently necrotic rather than inflammatory. Gangrene of the skin is even less frequent, although cases have been reported even in young children. Charron's case was only three years old, and several others in children are collected in Gimard's monograph upon this subject. The gangrene may involve the skin only, or the subcutaneous tissues and even the muscles. It has been seen upon the upper and lower extremities and even upon the face, and may extend over quite a large surface. In some of the milder forms of purpura, gangrene results from some slight injury, such as a blow, the pressure from a bandage, or in the nose, from the pressure of a tampon. In the gangrenous cases, all the symptoms are usually severe and indicate extensive blood alteration. They are almost invariably fatal. Those in which the sloughing is confined to small areas of the mucous membrane of the mouth often recover.

5. The rheumatic form.—Rheumatic purpura (peliosis rheumatica) is applied to cases, not so common in children as in older patients, in which subcutaneous hæmorrhages, and sometimes bleeding from the mucous membranes, are associated with painful joint swellings. These are to be regarded as cases of rheumatism complicated by purpura. The joints most frequently affected are the knee and the ankle. The arthritic symptoms are usually less severe than in attacks of acute rheumatism. There may be present erythema exudativa or erythema nodosum or urticaria. Usually there are throat symptoms and fever, and frequently œdema of the face and eyelids with albuminuria. The spleen may be enlarged. The usual duration is from one to three weeks, and although relapses may occur, the cases usually recover.

Joint symptoms, particularly articular pains, are not infrequent in the course of milder attacks of purpura without the febrile symptoms mentioned. In severe cases extravasations of blood have been reported as occurring in the tissues about the joints, and even in the joints themselves, these being cases of true arthritic purpura. It is probable that, in the past, some cases of scurvy have been included in this category.

Diagnosis.—The rapid acute cases may be confounded with the hæmorrhagic forms of the various eruptive fevers. The ordinary subacute or passive forms are chiefly to be differentiated from scurvy. The diagnosis is not difficult and the mistake need not be made if the essential features of scurvy are borne in mind,—its dietetic cause, bleeding gums, hyperæs-

thesia, and deep rather than subcutaneous hæmorrhages which are usually near the joints.

Prognosis.—This depends very much upon the form of the disease. Of 128 cases of all varieties occurring in children in Steffen's collection, there were 40 deaths. In 12 cases of severe primary purpura reported by Gimard, there were 3 deaths and 9 recoveries. Purpura simplex is rarely fatal; cases of purpura hæmorrhagica usually recover unless marked febrile symptoms are present. The forms classed as typhoid, gangrenous, and purpura fulminans are almost invariably fatal. The tendency to relapses exists in all varieties.

Treatment.—The treatment of symptomatic purpura should have reference to the cause of the disease. The mild cases of primary purpura usually recover promptly under a tonic plan of treatment. The more severe cases require confinement in bed, absolute quiet, and care to avoid exposure and even the slightest injury or extra pressure upon any part. Drugs do not seem to influence the course of the disease in any constant and uniform way. Those most frequently employed are hydrastis, hamamelis, aromatic sulphuric acid, the vegetable acids, ergot, and gallic acid. Iron should be deferred until active hæmorrhage has ceased. Whether or not it is true, as claimed by some, that all hæmorrhagic diseases are related to scurvy, the striking improvement seen in this disease from the use of fresh fruit and vegetables, suggests their employment in purpura. In some cases very decided benefit seems to follow their use in the acute stage, but more particularly in convalescence. For hyperacute and gangrenous cases, little can be done except to treat the symptoms. Surgical means of arresting the hæmorrhage are rarely successful. Iron and arsenic and alcoholic stimulants should be used in all cases during convalescence.

CHAPTER II.

DISEASES OF THE LYMPH NODES (LYMPHATIC GLANDS).

LYMPHATISM.

It is characteristic of infancy and childhood that the lymphatic glands, or the lymph nodes, as they are now coming to be generally called, throughout the body are prone to swelling and hyperplasia. While this tendency belongs to all children, in certain individuals it is so marked as to deserve a place as a distinct diathesis. It was formerly classed as one of the manifestations of "scrofula" or "struma"; but the proof that most of the manifestations formerly classed as "scrofulous" are really forms of local tuberculosis, makes it undesirable to use that term any longer as descrip-

tive of conditions now known to be often due to other causes besides inherited tuberculosis. The term *lymphatism* has been used by Potain and other French writers, and in this country by Bosworth, to designate this condition.

In stout, robust children, infectious processes of the nose, pharynx, or bronchi, cause acute swelling of the lymph nodes in the neighbourhood, but these rapidly subside when the cause is removed. In others, in whom a certain constitutional condition exists, the process in the mucous membrane is likely to be protracted, and the enlargement of the lymphatic glands once started continues even after the primary cause has subsided; or, diminishing for a time, it increases again with every new exciting cause until permanent enlargement may be produced.

I shall use the term lymphatism in the sense indicated,—viz., to designate an exaggerated susceptibility of the lymphoid tissue, a constitutional condition in which any inflammation of the mucous membranes or skin sets up hyperplasia in the lymph nodes with which these parts are connected, which is out of proportion to the exciting cause and which continues after the cause has ceased to operate. Besides, there must be included in this category, children who at birth have an excessive development of lymphoid tissue, seen particularly in the region of the throat in the form of enlarged tonsils, adenoid vegetations of the pharynx, etc.

Lymphatism may be inherited or acquired. The influence of heredity is too often seen to be passed over as a coincidence. Frequently the parents, when children, suffered from the same condition, and very often every member of a large family of children is affected. This may be the case in those who are in other respects healthy, who have been reared amid good surroundings, and in whom no evidence of any other constitutional disease can be found. Any disease in the parents in consequence of which children are born with tissues having less than normal resistance, may be regarded in the light of a remote cause. As such may be mentioned gout, rheumatism, alcoholism, syphilis, or tuberculosis, the child under these conditions inheriting not the disease, but, so to speak, its consequences.

Among the causes operating after birth to produce lymphatism, the surroundings of the child are of the first importance. It is seen to perfection in children reared in institutions; it is also frequent in crowded tenements and in cities rather than in the country. Anything which produces malnutrition or lowers the general vitality of the tissues may be ranked as a cause. Rickets and lymphatism are very frequently associated; sometimes rickets is to be reckoned as a cause, and sometimes both conditions depend upon the same causes.

The local manifestations of lymphatism are modified by the age of the child. During infancy, the glands which are most frequently affected are those connected with the gastro-enteric and the bronchial mucous membranes; in childhood it is those which are connected with the pharynx

and tonsils. This localization, of course, depends largely upon the fact that the susceptibility of the different mucous membranes is greatly influenced by age.

The degree of enlargement of the lymph nodes which is sometimes found in the different situations has often led to a misinterpretation of them, particularly by those who only seldom see autopsies upon infants or young children. They have often been connected with pathological conditions or clinical symptoms with which they have really nothing to do. One or two examples will suffice:

Enlargement of the mesenteric glands and of the solitary follicles of the large and small intestine, are very frequently seen in infants who have died of marasmus, and have been regarded as the cause of the wasting, while in reality they were only the consequence of the chronic indigestion which is an almost constant accompaniment of that condition. The finding of swollen Peyer's patches in cases of acute diarrhœa, with some other symptoms during life suggestive of typhoid fever, have often been looked upon as a confirmation of that diagnosis, as in a recent case reported by Northrup, in which cultures showed that the disease was not typhoid.

The condition under consideration relates not only to the larger lymph nodes, but to the smaller ones discernible only by the microscope. Where the larger ones exist, immense numbers of the small ones are sure to be present.

Lymphatism is essentially a condition of childhood. As time passes we see a regular succession of retrograde changes in the different series of glands unless they become the seat of tuberculous infection. Those connected with the digestive tract begin to diminish after the second year, and by the fifth or sixth year the enlargement has almost disappeared; while the tonsils, adenoid growths of the pharynx, and enlarged cervical glands are usually stationary after the seventh or eighth year and undergo quite a marked atrophy about the time of puberty. The presence of these enlarged lymph nodes, the catarrhal condition of the mucous membranes with which they are associated, and the constitutional condition upon which both depend, are important in relation to all acute infectious diseases which affect these mucous membranes. They bring about an increased susceptibility to scarlet fever, measles, diphtheria, diarrhœal diseases, and most of all to tuberculosis.

*Table showing the Situation and the Drainage-Areas of the Various Groups of Lymph Nodes of the Head and Neck.**

	Name of the group.	Number and situation.	Organs or areas from which they receive lymphatics.
1	Sub-occipital	One or two; at nape of neck.	Scalp, posterior portion.
2	Mastoid.	Four or five small ones; in mastoid region.	Receive efferent vessels from group 1, and through them from part of scalp.
3	Parotid.	Five to ten; on the surface and in the substance of the parotid gland.	Scalp, frontal and parietal portions; orbit, posterior part of nasal fossa, upper jaw, posterior and upper part of pharynx.
4	Submaxillary.	Twelve to fifteen; along base of jaw, beneath cervical fascia.	Mouth, lower lip, gums.
5	Supra-hyoid.	One or two; median line between chin and hyoid bone.	Chin and middle portion of lower lip.
6	Superficial cervical.	Five or more; along external jugular vein, beneath platysma, but superficial to the sterno-mastoid.	Auricle, part of scalp, skin of face and neck, and some efferent vessels from groups 1 and 2.
7	Deep cervical, upper set.	Ten to sixteen; about bifurcation of common carotid and along internal jugular vein. They are just above upper border of thyroid cartilage and on a level with hyoid bone.	Lower part of pharynx, larynx, palate, tonsils and part of tongue, part of nasal fossa, deep muscles of head and neck, and from inside the cranium. Receive also efferent vessels from groups 3 and 4.
8	Deep cervical, lower set.	A chain in the supra-clavicular fossa.	Connect with axillary group by a chain along axillary artery; also with glands of mediastinum and with groups 7 and 9.
9	Sub-hyoid.	A few small glands below hyoid bone and near median line.	Communicate with group 8, and may connect below with chain of bronchial glands.
10	Retro-pharyngeal.	Two small glands in front of spine and upon prevertebral muscles.	Pharynx and part of nasal fossa.

SIMPLE ACUTE ADENITIS.

This is an acute inflammation of the lymph nodes which in infancy frequently terminates in suppuration. A certain amount of inflammation of the lymph nodes occurs in children in all acute processes affecting the mucous membranes, especially when they are severe or prolonged. Those in connection with the various internal organs are considered with the diseases of the organs. Acute inflammation of the external nodes is of sufficient frequency to require separate consideration. While this is probably always secondary to some pathological process in the skin or mucous membranes, the primary condition may be so slight as to be overlooked, and the adenitis may be the more important condition or may even assume the appearance of a primary disease. It is particularly in

* Modified from Treves after Curnow in the Lancet, 1879, vol. i, p. 397.

infants that this is seen, and it depends upon the unusually active absorption and upon the susceptibility of the lymphoid tissues at this age. The cervical glands are frequently affected, and occasionally those of the axillary and inguinal regions.

Etiology.—Acute adenitis occurs in children of all ages in connection with diphtheria, scarlet fever, measles, and influenza. In such cases it is often severe, and, particularly with scarlet fever, not infrequently ends in suppuration. With the simple acute catarrhal processes of the pharynx and rhino-pharynx adenitis also occurs, but it is usually mild and rarely suppurates. In infancy, on the other hand, acute adenitis is not only very common from simple catarrh, but often severe, and frequently terminates in suppuration. Ulcerative stomatitis, carious teeth, eczema of the scalp or traumatism, may excite adenitis in children of all ages. Axillary adenitis may result from vaccination; inguinal adenitis, from vaginitis.

Of 109 cases of acute adenitis, not including those associated with diphtheria, measles, or scarlet fever, more than three fourths occurred in the first two years, and half of them in the first year of life. This susceptibility of infants is very striking. The disease occurs frequently in those who are in other respects perfectly healthy, and often when the evidences of disease of the mucous membrane are slight. This is true not only of the cases of cervical adenitis, but also of others in which the inguinal glands are involved. The inflammation is excited in most of these cases by the absorption of pyogenic germs from the mucous membranes or skin; in some cases, as in diphtheria, probably by the action of toxines.

Lesions.—The changes taking place in the glands are acute congestion, with swelling, œdema, and active hyperplasia of the lymphoid elements. The process may terminate in resolution or in suppuration according to the intensity of the infection and the susceptibility of the tissues. When severe enough to cause suppuration, the adenitis is accompanied by considerable inflammation of the surrounding cellular tissue.

In a series of 109 acute cases of which I have notes, not including the specific infectious diseases, 96 were cervical, 9 were inguinal, and 4 axillary; 62 per cent terminated in suppuration, the latter being nearly all in infancy. Suppurative otitis was present in 16 per cent of the cases. Suppurative retro-pharyngeal adenitis (retro-pharyngeal abscess) was seen in several cases.

In infancy the disease is usually unilateral, or, if bilateral, the glands of one side are much more severely affected than those of the other. Suppuration is nearly always of one side, and usually the abscess starts from a single gland.

Symptoms.—The symptoms and course of the adenitis of the specific infectious diseases belong to their clinical history. Suppuration is infrequent, except after scarlet fever. It is very rare after diphtheria, and

when present usually signifies mixed infection; I have seen it occur but twice.

The typical cases of acute adenitis are those which occur in infancy. There are present the symptoms of the original disease,—usually catarrh of the nose or rhino-pharynx, mouth, or ear, which may not be very severe, and sometimes is overlooked. The glands most frequently affected are the deep cervical group. The tumour appears just below the angle of the jaw at the anterior border of the sterno-mastoid muscle (Fig. 141). The swelling during the acute catarrh is not rapid or great, but continues after the original process has subsided until it reaches the size of a walnut or even a pigeon's egg. In the most acute cases there is marked inflammation of the periglandular cellular tissue, with pain, tenderness, and extra heat. If suppuration occurs, it is generally evident in the latter part of the second week, but sometimes it may be as late as the third or even the fourth week. In the axillary or inguinal region (Fig. 142) the symptoms of adenitis are essentially the same as in the neck. In the inguinal cases the degree of catarrh of the mucous membrane is often very slight.

Fig. 141.—Acute suppurative adenitis in an infant one year old, showing the most frequent situation of the tumour in the cervical region.

Fig. 142.—Acute suppurative adenitis (inguinal) in an infant three months old.

Most cases run their course with slight fever and few general symptoms; but in young infants the constitutional symptoms are often severe and the physician may be in doubt whether the local process is sufficient to explain them. The temperature may be from 102° to 104° F. for several days, with considerable prostration, which is much increased if there is complicating otitis. After suppuration, if freely opened at the proper time, the abscess heals rapidly and permanently, a sinus being rare. Occasionally infection extends from one gland to another, and a succession of these glandular abscesses occurs.

In the non-suppurative cases the swelling may be even greater than in those which suppurate; but it is less diffuse and apparently limited to the gland. It subsides slowly in the course of from four to eight weeks, often leaving a small tumour which may be apparent for several months. In susceptible children recurrent attacks of acute inflammation may lead to chronic enlargement which may last indefinitely. These glands do not become cheesy, except from subsequent tuberculous infection.

The acute cases in infancy in which suppuration occurs, appear to recover about as promptly and quite as completely as those terminating in resolution, although in the former the constitutional symptoms are more severe.

Diagnosis.—This is usually easy if it is remembered that, with the exception of the specific infectious diseases, and occasionally local causes like eczema of the scalp, carious teeth, etc., acute adenitis is essentially a disease of infancy. I have often seen it mistaken for mumps when the swelling was severe, but on close examination there is but little resemblance between the conditions. The disease is essentially acute, and has nothing in common with the slow suppuration seen in later childhood from the breaking down of tuberculous glands.

Treatment.—Prophylaxis requires that in all acute catarrhs, the mucous membrane should be kept as clean as possible by the use of nasal or pharyngeal sprays, or by syringing with simple solutions like Dobell's or Seiler's (page 56), or one of common salt.

In the stage of acute inflammation very hot applications or an ice-bag may be used for the relief of pain. It is very doubtful whether either of these means has much influence in preventing suppuration. If abscess forms, incision had best be deferred until pointing has taken place. If this plan is followed, refilling is rare. A simple free incision with proper antiseptic treatment is all that is required. Curetting may be done if there is much broken-down tissue present, but it is not usually necessary. In most of the cases the abscess promptly heals and a perfect cure takes place. In cases which do not suppurate, absorption may be promoted by the internal use of the iodide of potassium in full doses,—gr. x to xv daily to an infant of one year. I confess rarely to have seen any benefit from painting with iodine or from inunctions of iodine ointment or the oleate of mercury. If adenitis is secondary to carious teeth, eczema, or ulcerative stomatitis, these conditions should receive appropriate treatment. Such cases do not usually suppurate, but subside rapidly when the primary cause is removed.

SIMPLE CHRONIC ADENITIS.

This consists in a simple hyperplasia of the lymph nodes. There are considered here only the external glands, but those of the cavities of the

body are affected in a similar way, in diseases of the mucous membranes with which they are connected.

Simple chronic adenitis is not nearly so frequent as the acute form even in infants and young children, and it is rare after the fifth year. It may follow one or more attacks of acute adenitis, or it may result from subacute or chronic inflammations of the skin or of the various mucous membranes, infection from which causes the acute form. The same groups of glands are affected in both varieties. The most frequent subjects are children who have the diathesis described as lymphatism.

Symptoms.—The glands upon both sides of the neck are usually involved, and more often a group than a single gland. The degree of swelling is not generally great, being much less than in acute adenitis, and usually less than in the tuberculous form. There are no constitutional symptoms. Hypertrophy of the tonsils and adenoid growths of the pharynx are frequently present. There is seen no tendency to suppuration or caseation. The swelling usually increases slowly for one or two months, then remains stationary for about the same length of time, after which it slowly subsides, although it may not entirely disappear for years. A subacute course is more frequent than a very chronic one.

Diagnosis.—These cases are especially to be distinguished from those of tuberculous adenitis. The most important points for differentiation are: that they occur, as a rule, in children under five, and most frequently under three years, a period when tuberculous disease is not very common; that some definite exciting cause is usually present; that caseation and suppuration do not occur; that the glands do not become adherent to the skin or to the deeper tissues; that they enlarge much more rapidly than do the non-caseating tuberculous glands; and that they are influenced to a much greater degree by constitutional treatment. There are, however, some cases in which a differential diagnosis is impossible. Glands in which there was originally only a simple hyperplasia may undoubtedly become tuberculous by subsequent infection.

Treatment.—Operative measures are not called for. The local cause usually to be found in the pharynx, nose, or mouth—hypertrophied tonsils, adenoid vegetation of the pharynx, decayed teeth, etc.—should be removed whenever possible. Little benefit is seen from local applications. The syrup of the iodide of iron (twenty drops three times a day to a child of four years) or potassium iodide (five grains three times a day) should be given for a long period. In some cases more decided benefit is seen from arsenic (four drops of Fowler's solution in a glass of water three times a day). In all cases cod-liver oil should be given except during warm weather.

SYPHILITIC ADENITIS.

It is quite rare that a marked degree of glandular enlargement is seen as a symptom of hereditary syphilis; indeed, so rare that it is often for-

gotten that chronic multiple glandular enlargements are ever due to this disease. In the few examples that have come under my observation, this has been a late symptom of hereditary syphilis. The glandular enlargements have been cervical and multiple, and the degree of swelling has often been marked. They may be associated with disease of the bones or mucous membrane of the throat or of the nose, or without signs of such disease. The diagnosis of syphilis rests upon the association of other late manifestations of the disease—keratitis, periostitis, deformities of the teeth—and the prompt improvement under anti-syphilitic treatment. In their local appearance they resemble tuberculous glands.

TUBERCULOUS ADENITIS.

Synonym: Scrofula.

Tuberculous disease of the lymph glands of the cavities of the body is discussed elsewhere; only that of the external glands is here considered. These present some striking peculiarities,—they are relatively rare in infancy, although a frequent form of tuberculosis in older children; it is exceptional to find them associated with general tuberculosis, and then they more often follow than precede the general disease. In the great majority of cases it is the cervical glands which are affected.

Etiology.—The age at which tuberculosis of the cervical lymph glands is usually seen is from three to ten years. In my experience with tuberculosis in infancy, the external glands are rarely involved, this being in striking contrast to the regularity, almost uniformity, with which the bronchial glands are the seat of infection.

In addition to infection with the tubercle bacillus, local causes are usually present; the most important are adenoid growths of the pharynx, chronic pharyngitis, and hypertrophied tonsils; less frequently there are chronic otitis, chronic conjunctivitis, and pathological processes of the skin or the mouth, such as eczema of the face or scalp, ulcerative stomatitis, carious teeth, etc. For the production of the disease, therefore, there appear to be necessary, first, favourable local conditions, and, secondly, exposure to infection. That the pharynx is the most frequent seat of primary infection, is shown by the fact that the deep cervical glands are generally first affected. The question often arises whether the process in the glands is at first simple, and later becomes tuberculous, or whether it is tuberculous from the outset. No doubt there are many examples of both conditions; however, my own conviction is that in the majority of cases the process is a tuberculous one from the beginning.

Children who are by inheritance predisposed to tuberculosis and those also who are prone to glandular enlargements—two conditions which are by no means identical—are the ones most liable to be affected. Attacks of acute infectious diseases, particularly measles, scarlet fever, and influenza, frequently play the rôle of exciting causes.

The age of those affected corresponds very closely with that at which most children are seen with hypertrophied tonsils and adenoid growths of the pharynx. The subsidence of symptoms about the time of puberty, is also characteristic of both conditions.

Lesions.—It has been already stated that in the great majority of cases the cervical glands are involved, and generally they are the only ones affected. In 155 cases of tuberculous glands in the series reported by Treves,* those of the neck were the seat of disease in 145 and the only seat in 131; those of the axilla were involved in 17, but alone only in 4; the groin in 8, and alone in 6. This indicates the close association of the disease with infection through the upper respiratory tract. The glands first affected are most frequently the upper set of the deep cervical group; sometimes, however, it is the superficial glands of the submaxillary, or the parotid group, and occasionally the submental or the pre-auricular.† The chain of deep cervical glands which is involved, follows the carotid artery, and often extends some distance below the clavicle. These deep glands are sometimes connected with the bronchial group.

The process in all tuberculous glands is essentially a chronic one, but pathologically the cases may be divided into two groups, corresponding somewhat to the forms of disease seen in the lungs. In the first group the process is more rapid, and tends to early caseation and softening; the products of inflammation are mainly cellular, and the amount of fibrous tissue is small. In the second group the course is much slower, and fibrous tissue predominates, the cells being fewer, and caseation and softening infrequent.

In the first group the glands in the early stage are swollen, of a pale pink colour, and homogeneous; later they become more firm, and show, as the first gross evidence of tuberculous deposits, small grayish-white spots, which are generally numerous and scattered through the affected gland; these spots enlarge, and may coalesce to form one large gray mass, involving nearly the whole gland. Subsequently there is caseation and then softening, usually beginning in the centre of the caseous area. Inflammation within the gland is followed by that of the surrounding tissues, which may result in adhesions or in the formation of a periglandular abscess. The first change in the gland is the production of epithelioid and giant cells, about which there is a zone of small round cells; cheesy degeneration then begins in the centre. The caseous masses may become encapsulated by the production about them of fibrous tissue; or softening may occur at one or more foci, and an abscess form. Such an abscess contains curdy materials but very little true pus, the contents being

* Scrofula and its Gland Diseases. Smith, Elder & Co., London, 1882.

† Nicoll, Glasgow Medical Journal, January, 1896.

chiefly parts of the gland not completely broken down. Caseation may be followed by calcareous degeneration, although this is rare, much more so than in the mesenteric or bronchial glands. Tubercle bacilli are usually more numerous in the early stages of the process, but are often difficult of detection in late cases in broken-down tissues, and the curdy pus is sometimes sterile. As the glands soften, the process gradually extends from the centre to the surface, and they become adherent to the surrounding structures—blood-vessels, nerves, organs, or the cellular tissue—they fuse together and form large knotty masses, and when they ultimately break down they lead to the formation of abscesses in the cellular tissue, finally involving the skin. In the form of suppuration which occurs in and about tuberculous glands, an important part is often played by other bacteria, usually the staphylococcus or the streptococcus.

In the second group of cases, where the process goes forward more slowly, the changes are not quite the same, the essential difference being that the amount of fibrous tissue is much greater. These glands are not so vascular; they are tough and hard, appearing like small fibrous tumours. The capsules are greatly thickened, and under the microscope is seen fibrous tissue arranged in concentric layers, often inclosing small caseous masses. These glands less frequently form adhesions to the surrounding tissues, and consequently are freely movable, while suppuration is quite exceptional. Although the separate tumours are much smaller than in the first group, the glandular mass is often a large one, because of the number of glands involved.

Treves gives some interesting observations in regard to the spreading of the process from one gland to another. He states that while it often takes place along the direct line of the lymph current, this is not always the case, and sometimes it spreads in exactly the opposite direction. This he believes to be due to an extension of disease from the gland to the afferent lymphatics, these vessels themselves becoming the seat of disease, with changes similar to those taking place in the glands. In consequence of this many more tuberculous nodes may be found than there were originally lymph glands,—a point which has often been noticed, but for which there is no other satisfactory explanation.

Symptoms.—In the early part of the disease there are no symptoms but glandular swelling, and this begins very gradually, often insidiously. In the majority of the cases both sides are involved, although one frequently begins before the other and advances more rapidly. The enlargement is not always continuous; it may increase for a time and then remain stationary or even diminish, to take a fresh start under the stimulus of some new process in the mucous membrane with which the glands are associated, such as an attack of measles or scarlet fever, or simply from a depreciation of the patient's general health. During exacerbations, the glands may be painful and tender, and show the usual signs of local inflam-

mation. The whole course of the disease varies from several months to as many years. Treves gives three and a half years as the average duration where suppuration occurs. The glands first affected are usually those situated near the bifurcation of the common carotid artery. Such tumours usually make their appearance just in front of the sterno-mastoid muscle—sometimes behind it—and at the level of the upper border of the larynx or the hyoid bone. In the more rapid cases the tumours usually attain a considerable size in three or four months, sometimes in half that time. The usual size reached is from that of an almond to an English walnut. At first the tumours are movable and preserve their distinct outline; later they become adherent, first to the deeper tissues and to each other, finally to the skin, and there is formed an irregular nodular mass in which it is sometimes difficult to make out the individual glands. As they approach the surface there are small spots of softening; then there is distinct fluctuation; the skin becomes discoloured and finally gives way, and there is a discharge of thick, curdy pus, which may continue for an indefinite time, until the whole of the broken-down gland has been thrown off.

In the cases which progress more slowly, a chain of glands is usually involved which individually are smaller than the preceding, and yet together they may form quite a large mass. These rarely become adherent, except to each other, and suppuration is very infrequent; the skin over them therefore is generally healthy. In most of the cases where suppuration has not occurred an improvement takes place about the time of puberty. In what proportion of these glands there is suppuration it is impossible to say. Like other tuberculous lesions in the body, these glands are much more often the seat of infection than was formerly supposed, and in many cases the diagnosis is not made. Of those recognised clinically as tuberculous adenitis, from one half to two thirds suppurate, provided they are allowed to run their natural course. Resolution is more likely to occur where the progress is slow, and where there are many small tumours than with one or two large ones. If softening has occurred, resolution is not to be expected, although even in such cases encapsulation of the cheesy foci may take place. Occasionally cases are cured by intercurrent acute disease. A cure has been known to follow an attack of scarlet fever, and erysipelas of the face (Treves). The usual effect of the eruptive fevers, however, is to accelerate the process.

Two forms of suppuration occur in connection with tuberculous glands,—one an abscess of the gland proper, the other outside of and usually over it. In a typical case of the first variety, the gland is distinctly outlined and often superficial, there is very little inflammation, the spot of softening and fluctuation is small, and the pus discharged is always curdy. In the second variety the abscess is preceded by a more diffuse swelling, and the outline of the gland may not be made out; the signs of inflammation are more marked, the area of fluctuation is larger, and the pus is

more like that of any ordinary abscess. Often the two varieties are combined; as when a gland beneath the deep fascia breaks down and there is formed directly over it an abscess in the cellular tissue, which communicates through a narrow opening with the gland beneath. In such cases the discharge may continue for a very long time, until the whole of the gland has been removed. If healing occurs before this, the cicatrix soon breaks down.

Where abscesses are allowed to open spontaneously, large, irregular, and usually very intractable ulcers often form. The skin is undermined for a considerable distance, and it has an unhealthy appearance.

Fig. 143.—Cicatrices following a neglected case of tuberculous adenitis, in a girl seven years old. There is also a tuberculous patch upon the skin of the cheek in a very frequent location.

Such ulcers sometimes continue for many months in spite of all treatment, particularly if the patient's general health is poor. The scars left after them are large and unsightly, and sometimes positively deforming (Fig. 143). Their appearance is quite characteristic. They often have many tabs of skin attached to them; they may form prominent ridges which may undergo contraction like those after burns; they are of a purplish-red colour, and adherent to the deeper tissues. They are often sensitive and painful. As time passes they atrophy and become less conspicuous, though they remain through life.

The general health of children with tuberculous glands may be much or little affected, and not a few remain in good condition throughout the whole course of the disease, particularly when suppuration does not occur, but sometimes even when it is protracted.

Prognosis.—In no case, I think, does tuberculosis of the external lymph glands cause death. Though the course is often protracted, lasting in some cases for eight or ten years, ultimate recovery may be confidently predicted in the great majority of cases. As stated at the beginning of this article, it is a matter of surprise that so few of these children ultimately develop general tuberculosis. Treves * says, "The percentage of those who fall victims to diffused tubercular disease is so small that the probability of that disease may be put out of the question," and that to urge the prevention of phthisis as an argument for operation "is unworthy of consideration." Poore † states that of fifty-eight cases, only two were known to have died of tuberculosis. Nordan on the other hand reports that of 149 cases that were followed, eighteen per cent were known to have died from tuberculosis, and nine per cent, though living, were suffering from that disease. Although it is certainly infrequent, I can not believe such a sequel to be quite so rare as do the two authors quoted.

Diagnosis.—Tuberculous adenitis is to be distinguished from simple chronic enlargement, from that due to syphilis, from Hodgkin's disease, and from malignant disease. The diagnostic features of tuberculous glands are the age of the patient—usually from three to ten years—the site of the primary swelling, the indolent course, the trifling original cause, and most of all the disposition to slow caseation, softening, and abscess. The cases of simple hyperplasia are usually in children under five years, their progress is much more rapid, there is often some definite cause, and they have in most cases nearly or quite disappeared in the course of three or four months. They suppurate, if at all, during the first month. Syphilitic disease is to be recognised mainly by discovering the evidence of syphilis elsewhere, and by the effect of treatment. In Hodgkin's disease, glandular groups in other parts of the body are involved simultaneously or in rapid succession. There are no signs of inflammation or caseation; and the swellings are accompanied by very marked and definite constitutional symptoms,—anæmia, emaciation, and general prostration. Malignant growths are very rare, they increase rapidly, often attaining a great size in a few months.

Treatment.—The general treatment of tuberculous glands is to put the child under the very best surroundings possible. The seaside has a great reputation for such cases, and no doubt the majority do very well there; but some are benefited even more by a dry, mountain climate. At all events, a child from the city should be sent into the country whenever

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* *Loc. cit.*, p. 188. † New York Medical Journal, June 23, 1892.

this is possible. Internally the only remedies which have any special virtues are cod-liver oil and the syrup of the iodide of iron. The latter should be given in full doses—i. e., twenty or thirty drops, three times a day, to a child of six years. Arsenic and iron are useful as general tonics. Local applications are of little value and most of them positively harmful; painting with iodine and poulticing should be discarded altogether. The parts should be protected against cold, and should be rubbed or handled as little as possible.

It is important in every case to remove from the nose and throat all sources of local irritation. Hypertrophied tonsils should be excised, and the adenoid tissue of the pharynx scraped out, even when not very extensive, since these are the two regions which most frequently harbour the tubercle bacilli. Any pathological conditions in the nose, such as hypertrophy of the turbinated bodies, should receive attention; so also should chronic otitis, chronic conjunctivitis, carious teeth or ulcers in the mouth. All these, if they do no more, keep up a constant glandular irritation, and produce conditions which are most favourable for the activity of the tubercle bacillus.

Operative measures.—These are indicated if, after two or three months of constitutional treatment, the glands affected continue to increase in size and number. The advantages of operation over leaving the case to Nature are, that it leaves a clean scar instead of a large, irregular one; that it shortens the disease and prevents the long, tedious suppuration of cases left to themselves; that it is a radical measure; and that it avoids the danger of general infection by removing the tuberculous focus.

With reference to the choice of operations, surgeons are by no means agreed. The indications for the different operations laid down by Treves, seem to me to be the best that have been formulated:

1. Excision and enucleation.—Adapted to cases where there is no active inflammation and no softening; where the process is very slow and indolent; where there are one or two large, hard glands, or a chain of smaller ones, all freely movable and all clearly defined, or where there is a single large tumour causing pressure symptoms.

2. Scooping.—Adapted to glands which have softened and are adherent, especially to the skin; also where the capsules are thickened. This operation should not be done during a period of acute inflammation.

3. Cautery puncture.—Useful both in hard, movable glands and in those which are soft and adherent; particularly adapted to those adherent to the skin, and for these it is better than the scoop. It is not applicable to glands smaller than a cherry. This operation is done with a small cautery point, which is thrust through the skin into the gland, and then in two or three directions through it, after which some soothing dressing is applied. Although widely used in Europe, this operation is but little

employed in America,—not so often, it would appear, as it should be, from the advantages claimed for it.

All surgeons agree that in operating, violent tearing out of the glands should be avoided; that as little injury as possible should be done to the tissues; that the capsules should not be torn nor the tuberculous materials allowed to escape into the healthy tissues. All agree also that prolonged dissections are to be avoided, and that in removing deeply-seated glands there is great danger of injuring vessels and nerves and the dome of the pleura.

Glandular abscesses should in all cases be opened as soon as pus forms, to prevent the extensive undermining of the skin, which is so likely to occur. The opening should be a small one, and all squeezing of the gland or surrounding tissues avoided.

HODGKIN'S DISEASE (ADÉNIE).

This is a rare disease in which there is a general hyperplasia of the lymphatic glands throughout the body, with growths of lymphoid tissue in the spleen, liver, and other internal organs. It is accompanied by marked anæmia, is progressive in its course, and usually terminates fatally. The cause is unknown. It is much more common in males than in females. Its occurrence in childhood is exceedingly rare.

The changes in the glands consist in a simple hyperplasia, which may be extreme. Suppuration and caseation are very rare, if indeed they ever occur. Any of the external or internal groups of lymph glands may be affected, and in severe cases the disease may involve almost every chain of glands in the body. Of the external groups, the cervical and the axillary are usually most affected; of the internal groups, those of the mediastinum and the retro-peritoneal region. The spleen and the liver are moderately enlarged, and lymphoid growths, varying in size from a pin's head to a grape, are usually scattered throughout their substance. There may be changes in the bone-marrow.

Symptoms.—These come on very gradually, often insidiously. The external glandular swellings are usually the first noticed, but sometimes it is the anæmia which first attracts attention; occasionally it is the local symptoms resulting from the pressure of internal glands, which may give rise to œdema, pain, cough, or dyspnœa. The progress is generally slow but steady, and the glands may reach an immense size. The blood shows a moderate reduction of the red and an increase in the white cells, particularly the lymphocytes (Osler).

Treatment.—The only remedy which is of much avail in this disease is arsenic, which must be given in full doses and for a long time. The general treatment should be tonic.

CHAPTER III.

DISEASES OF THE SPLEEN.

Weight.—From one hundred and forty observations made at the New York Infant Asylum the following were the weights recorded at the different ages:

Weight of the Spleen in Infancy and Early Childhood.

Age.	Ounces.	Grammes.
Birth	$\frac{1}{4}$	7·7
Three months	$\frac{1}{2}$	15·5
Twelve "	$\frac{3}{4}$	23·2
Two years	$1\frac{1}{4}$	38·5
Three "	$1\frac{1}{2}$	46·4

Position and Methods of Examination.—The normal position of the spleen is close against the diaphragm, its external surface being opposite the ninth, tenth, and eleventh ribs. Its anterior border comes as far forward as the middle axillary line, its posterior border being usually near the vertebral column. In infancy it is practically impossible to outline the spleen by percussion, unless it is enlarged. During full inspiration the spleen is often depressed enough to be felt at the free border of the ribs, but at other times it can not be felt unless it is enlarged or pushed downward by some pathological condition in the chest. Normally, the long axis of the spleen is nearly parallel with the ribs, but when the organ is much enlarged, its axis corresponds nearly with a line drawn from the axillary line at the border of the ribs to the middle of Poupart's ligament.

The thin abdominal walls of young children render palpation of the spleen much easier than in adults; and this is a much more satisfactory method of examination than is percussion. In fact, the results from percussion are so uncertain and misleading that in most cases one may dispense with it, and rely on palpation to determine the size of the spleen. For satisfactory palpation it is necessary that the abdominal walls should not be tense. It is therefore important that the child should be quiet, and that the examination be made as gently as possible, and no force or undue pressure used. The child should lie upon its back with the thighs flexed and the skin, of course, bared. The physician, always having taken the trouble to warm his hands, should stand upon the left side of the patient and make pressure with the tips of the fingers, which are semiflexed. The pressure should be at first light and gradually increased, the fingers being then held stationary during two or three respiratory movements. It is sometimes better to use the fingers of one

hand for palpation, and make pressure with the other directly over the first. Palpation should be made in the axillary line. If the examination is satisfactory, and in the great majority of cases it is so if the child is quiet, the spleen can easily be felt when it is sufficiently enlarged to be of any diagnostic importance. With a little practice one can readily detect even slight degrees of enlargement.

When moderately enlarged, the lower border of the spleen is an inch or so below the free border of the ribs; when greatly enlarged, it forms a tumour which may nearly fill the left half of the abdomen. A tumour in the left hypochondriac region is recognised to be the spleen, by the fact that it is freely movable laterally and at its lower border or extremity, while it is attached above; also its inner border can usually be felt to be thin and sharp, and marked about its middle by quite a deep notch.

ENLARGEMENT OF THE SPLEEN.

In Acute Disease.—The spleen is most frequently and most constantly enlarged in malarial and typhoid fevers, but it is occasionally so in all the acute infectious diseases.

In most of these cases the enlargement is chiefly from congestion, but there may be acute hyperplasia and an increase in size of the Malpighian bodies. It may contain small hæmorrhages, and in extremely rare cases the spleen may rupture. In appearance it is generally dark-coloured, soft, and somewhat friable. In the cases which recover, the splenic swelling subsides with the original disease.

In Chronic Disease.—Like the lymph nodes, the spleen is much more often enlarged in children, particularly young children, than in adults. Enlargement is seen at times in almost all the chronic diseases of early life; but it occurs most frequently in rickets, syphilis, malaria, tuberculosis, the blood diseases, and in amyloid degeneration. Besides, it may be the seat of primary disease, either simple or malignant.

Rickets.—The splenic enlargement which accompanies rickets is generally seen during the first year; at this period it is very frequent. The swelling is usually moderate, but occasionally it is so great that the lower border is three or four inches below the ribs. It belongs to the most severe forms of the disease.

Syphilis.—Enlargement of the spleen is one of the most constant lesions in congenital syphilis. It is present with great uniformity in children born with syphilitic lesions, and very frequently during the active period of the disease in early infancy. It is seen at a later period during infancy or childhood, associated with other late symptoms. The degree of enlargement is often great. In several cases I have seen it sufficient to form a large abdominal tumour. The liver also is increased in size, but not to such a degree. The pathological changes in the spleen in syphilis are considered with that disease.

Küttner * has made a study of the blood in cases of hereditary syphilis and rickets that were accompanied by splenic enlargement. The number of red cells was found to vary greatly, as did also their ratio to the white cells.

Malaria.—The swelling in these cases may be very great. The liver is not so often enlarged as in syphilis. There is usually a history of exposure in a malarial district.

Tuberculosis.—It is rare to find anything more than a moderate swelling of the spleen in tuberculosis. In the most acute cases this may be due to the fever and general infection; in those which are less rapid, it depends either upon tuberculous deposits or passive congestion from venous obstruction, the result of the pulmonary disease.

The blood diseases.—Marked enlargement of the spleen is found in many cases of simple anæmia accompanied by moderate leucocytosis. This is quite peculiar to infancy and early childhood. The spleen is constantly swollen, and usually greatly so, in the pseudo-leucæmic anæmia of infants, in leucæmia, and in Hodgkin's disease. In the last two diseases the liver is also enlarged, but to a much less degree than the spleen; in the others it is but slightly changed.

Amyloid degeneration.—The causes of this condition and its general symptoms are mentioned in connection with amyloid disease of the liver (page 413). The spleen is constantly involved, and the enlargement of this organ, as well as that of the liver, may be very great. The changes resemble those found in the liver.

Cardiac disease.—In all forms of cardiac disease, and in other conditions in which there is obstruction to the systemic venous circulation, the spleen is enlarged. It is seen in congenital as well as in acquired cases. The liver is usually enlarged to about the same degree as the spleen, and there may also be dropsy of the feet.

New-growths, tumours, etc.—In rare cases in early life, the spleen is the seat of new-growths; these are usually varieties of sarcoma, but carcinoma has also been reported. Lymphoma, or, as it is more properly called, simple hyperplasia of the spleen, has occasionally been observed in early life, apart from any of the constitutional diseases above mentioned.

Acker (Washington) has reported a remarkable case in a coloured boy of eight years, who died of scarlet fever a year after the splenic tumour was first noticed. At the autopsy the spleen weighed fifty-two ounces. There was found a very great degree of hyperplasia, but nothing indicating malignant disease.

Echinococcus of the spleen has been reported in Europe, but none, so far as I am aware, in America, among children.

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* Jahrbuch für Kinderheilkunde, Bd. xxxv, H. 2.

CHAPTER IV.

DISEASES OF THE BONES AND JOINTS.

ACUTE ARTHRITIS OF INFANTS.

The term *acute arthritis of infants* has been given by Thomas Smith, Townsend,* and others, to a form of joint inflammation which is peculiar to infancy and not very rare at this time. It has been described under the names of *acute purulent synovitis of infants*, *acute epiphysitis*, *pyæmia of bone*, *acute osteo-myelitis*, etc. The disease is essentially a form of pyæmia, and is a suppurative process almost from the outset. It may begin at the epiphyseal junction, in the medullary canal, or in the joint; usually, however, the joint is invaded secondarily, the disease sometimes spreading to it with great rapidity from the bone. It may also result in a diffuse osteo-myelitis or in a subperiosteal abscess. Secondary abscesses may form in the viscera or in distant articulations. As a consequence of the disease, there may be separation of the epiphysis from the shaft, sometimes entire destruction of the articular extremities of the bone or articular cartilages. As late results there may be a pathological dislocation, or a "flail joint"; less frequently there may be anchylosis. The extent of the ravages in the joint structures depends chiefly upon the duration of the process. Where the pus is evacuated early, recovery may take place with very little permanent damage; but in neglected cases complete destruction of the joint often occurs.

Etiology.—Of 73 cases collected by Townsend, all but four occurred during the first year of life, and over half of them during the first three months. These early cases have already been mentioned among the Pyogenic Diseases of the Newly Born (page 82). So far as is known, the disease has no relation either to syphilis or tuberculosis. There is in some cases a history of traumatism, but this can only play the rôle of an exciting cause. The essential cause of the disease is the entrance of pyogenic germs into the circulation. They may gain admission through the umbilicus, some abrasion of the skin, or the conjunctiva (pages 79, 80). Very often the source of infection cannot be discovered. Cases occurring later than the first few months of life have sometimes followed measles, scarlet fever, or empyema.

Symptoms.—The onset is often sudden, with well-marked local and constitutional symptoms. The disease may be ushered in with a chill, followed by a fever, which is frequently high, fluctuates widely, and is accompanied by general prostration, restlessness, and other signs of pain.

* W. R. Townsend, M. D., American Journal of the Medical Sciences, January, 1890. Here will be found a full discussion of the subject, and the bibliography.

There is rapid swelling about the affected joint, which is usually diffuse, as the lesion is deep-seated. There is also acute tenderness, and usually deformity. Later there are redness, œdema, a glazed skin, and deep fluctuation. In some cases the constitutional symptoms are slight or wanting. After pus forms, it may lead to rupture of the capsule and infiltration of all the tissues about the joint, often burrowing for a considerable distance before it reaches the surface.

When its progress is most rapid, death may occur in two or three days, from exhaustion or general pyæmia. The lesions in such cases are usually multiple. The usual duration is from one to two weeks, suppuration generally being evident in four or five days. In Townsend's collection of cases the joints were affected in the following order: hip, in 38 cases; knee, in 27; shoulder, in 12; wrist, in 5; elbow, in 4; ankle, in 4; fingers, in 2; toes, in 1; sterno-clavicular, in 1. I have met with one case in which suppuration occurred in the temporo-maxillary and the medio-sternal joints; in another, in the temporo-maxillary and shoulder. In 75 per cent of the cases collected by Townsend only one joint was involved, and of these two thirds recovered; in the remaining 25 per cent, with multiple joint lesions, only one fourth of the cases recovered. Of those who survive the acute period, the number who recover with perfect joints is small.

Diagnosis.—The disease is not usually difficult of recognition, from the constitutional symptoms, the marked swelling, tenderness, and deformity, and the rapidity with which suppuration occurs. It has been mistaken for rheumatism, although rheumatism is so rare in infancy that it may be practically ignored. Syphilitic epiphysitis resembles it in the localized pain, tenderness, and general immobility, but lacks the rapid swelling, fever, and severe constitutional symptoms, and its course is more prolonged. Acute cellulitis in the neighbourhood of the joints may resemble it, but this is rare except from traumatism. The disease has little in common with tuberculous bone disease of later childhood.

Treatment.—The general treatment is to be directed toward the patient's condition, and the purpose of it should be to relieve pain and support the general strength. Suppuration occurs very early, and no time should be wasted in trying to allay the inflammation by local applications. The best results are obtained by early incision, free drainage, and thorough antiseptic treatment. Fixation of the joint should follow operation, in order to prevent deformity.

THE TUBERCULOUS DISEASES OF THE BONES AND JOINTS.

The chronic forms of tuberculous bone-disease, on account of their insidious onset and the frequency with which they simulate other diseases, more frequently fall, in the early stage at least, into the hands of the physician than into those of the general or orthopædic surgeon. All that will be attempted in this chapter will be to outline in a general way

the most important forms—viz., disease of the vertebræ, hip, and knee—dwelling particularly upon the early symptoms and diagnosis. For their fuller discussion, particularly as to the details of treatment, the reader is referred to text-books on general or orthopædic surgery. The causes are the same, and the lesions are very similar in all forms, and will therefore be considered together.

Etiology.—The age at which tuberculosis of the bones most frequently begins, is from the third to the eighth year, it being comparatively rare before the end of the second year. The sexes are affected with about equal frequency. Tuberculous bone disease may occur in a child who has previously been in apparent health, but more often in one who has been reduced by some previous illness, especially the infectious diseases of childhood; of these, it most frequently follows measles and whooping-cough.

A history of inherited tuberculosis is present in a large number, but by no means in a majority of the cases. Like tuberculosis of the cervical glands, it is rarely preceded by other tuberculous processes, although it may be followed by them. It usually appears as an example of primary infection; but it seems very improbable that such should actually be the case. It is more likely that there has previously been a latent focus of tuberculosis elsewhere in the body. In many cases, antecedent disease of the bronchial glands has been demonstrated by autopsy. Infection from these or from other tuberculous lymph glands, is the most probable explanation of the origin of infection in cases of bone disease. However, by some writers, notably Baumgarten, tuberculous disease of bone is regarded as due to direct inheritance, and is to be compared to the bone lesions which occur as late manifestations of hereditary syphilis.

Traumatism is often an exciting cause, and it may determine the site of the disease.

Lesions.—The tuberculous joint diseases of childhood are, as a rule, secondary to disease of the bones. Hip-joint disease usually begins in the head of the femur, and knee-joint disease in one of the condyles; ankle-joint disease in the lower epiphysis of the tibia, etc.

The frequency with which disease is seen in the different locations is shown by the following table, which gives the number of cases of each form applying for treatment at the Hospital for Ruptured and Crippled, New York, during the years 1884 to 1893 inclusive:

Spine	2,145	cases, or	37·5 per cent.
Hip	1,937	" "	34·0 "
Knee	1,222	" "	21·5 "
Ankle or tarsus	255	" "	4·5 "
Elbow	71	" "	1·2 "
Wrist	50	" "	0·9 "
Shoulder	24	" "	0·4 "
Total	5,704		100·0

The character of the bone disease upon which chronic joint disease depends is generally a primary ostitis, which affects the articular extremities of the long bones usually beginning near the epiphyseal line; in the short bones it is a central ostitis. The stages in the process are first congestion, swelling, and cell infiltration, followed by caseation, and frequently by softening and suppuration. In the early stage, the bone is slightly enlarged, and on section one or more yellowish foci of disease are seen. The disease may be arrested in this stage, encapsulation of the inflammatory products taking place; or it may continue until there is a more or less extensive breaking down or disintegration of the affected bone. As the disease extends there are involved, the periosteum, the articular cartilage, and finally the joint itself. Abscess may form in the joint or in the soft parts surrounding the bone. The process is quite analogous to tuberculous disease of the lung. As the disease advances ligamentous attachments are loosened, and displacement of the parts occurs with the production of deformity, due partly to muscular contraction and partly to the weight of the body. The inflammatory process with its resulting disintegration generally goes on to a certain point, where it is arrested. Gradually the broken-down bone substance is separated and thrown off in small particles in the discharge, and a reparative process begins, with the formation of healthy bone. Where joint structures have been destroyed, cure takes place by bony anchylosis. Sometimes the disease finds its way to the surface without involving the joint; at other times the disease may be arrested, and its products become encapsulated within the bone. Inflammation of the joint may occur by a gradual extension of the inflammatory process, or by a sudden perforation of the articular lamella. As a result of extensive disease, all the joint structures may be affected,—the synovial membrane, ligaments, articular cartilages, and the cellular tissue surrounding the joint. The process of disintegration and that of repair are both very chronic and measured by months or years. The entire course of the disease is from one to ten years, three years being about the average duration. In the great proportion of cases but one joint is involved, although it is not infrequent in hospitals to see two, three, and sometimes four of the large joints affected in the same patient.

Secondary lesions.—Abscesses form in a considerable proportion of the cases, and often burrow a long distance before they reach the surface. Amyloid degeneration of the liver, spleen, and kidney, and sometimes of the villi of the intestines, occurs as the result of the prolonged suppuration, chiefly in connection with disease of the hip or spine, occasionally with that of the knee. General or localized tuberculosis, particularly tuberculous meningitis, may develop at any time and prove fatal.

Caries of the Spine—Pott's Disease.—This consists in a chronic inflammation of the bodies of the vertebræ, usually beginning in the central portion and extending to the periosteum, ligaments, cartilages, and,

in fact, to all the contiguous structures. It frequently involves the membranes of the cord, the roots of the spinal nerves, and even the cord itself. The number of vertebræ usually affected is from two to five. The gross appearance of the lesion in a well-marked case is shown in the accompanying cut (Fig. 144). After the bodies of the vertebræ have become softened and partially broken down by disease, the pressure from the superincumbent weight of the body causes them to fall together and produces a backward displacement of the spinous processes, giving rise to the deformity known as kyphosis, which in its extreme form is popularly known as "hunchback."

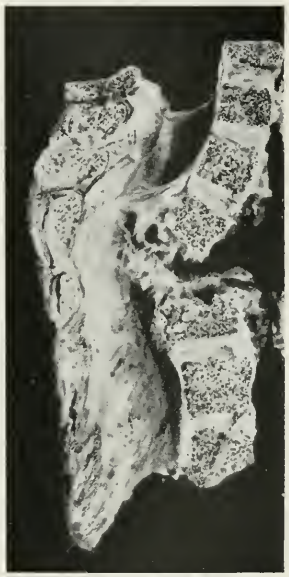

Fig. 144.—Pott's disease of the upper dorsal region; a vertical section of the spine, showing disintegration of the bodies of the vertebræ and encroachment upon the spinal canal. (From a patient dying in the Hospital for Ruptured and Crippled.)

Any part of the vertebral column may be affected; but the disease is most frequent in the dorsal region, as shown by the following statistics from the Hospital for Ruptured and Crippled: of 2,143 cases, 72·5 per cent affected the dorsal region, 15·3 per cent the lumbar region, and 12·2 per cent the cervical region.

Symptoms.—The onset is gradual, often insidious, and the early symptoms are frequently overlooked or misinterpreted. The case may go on for weeks or even months before the true nature of the disease is recognised, which is often not until deformity has occurred. In nearly all cases, however, the early symptoms are sufficiently characteristic to enable a careful observer to make a diagnosis before the stage of deformity.

The most constant early symptoms are: (1) pains caused by the irritation of the nerve roots and referred to various parts of the body, following the distribution of the spinal nerves; (2) rigidity of the spine from muscular spasm, this being an attempt to prevent motion at the seat of disease; and (3) the assumption of various postures calculated to relieve pressure upon the diseased vertebral bodies. Sometimes the first symptoms are those of pressure-paralysis (page 768); at others they are the local signs of abscess. In addition to the local symptoms mentioned, there is usually disturbed sleep, often accompanied by moaning.

Cervical disease.—The pains are often felt above the point of disease, frequently in the form of occipital neuralgia; sometimes they are referred to the front or the side of the neck. They may be so frequent and so severe that the face assumes a constant expression of anxiety or distress. In other cases pain is excited only by an attempt at movement. The

muscular spasm most frequently takes the form of slight torticollis, sometimes of slight opisthotonus; sometimes there is simply a fixation of the head by a tonic spasm of all the muscles of the neck; both active and passive motion is resisted, and any movement may be so painful that the child involuntarily steadies its head with its hands. These symptoms come on gradually and are persistent. Sometimes they are overlooked, and the first thing to attract attention is a progressive weakness in the lower extremities, which proves the beginning of paraplegia. Occasionally the first marked symptoms are those due to the formation of a retro-pharyngeal or a retro-œsophageal abscess (page 276).

The deformity from cervical disease develops much later than when the disease is located elsewhere. Usually the neck appears broadened or thickened in a nearly uniform way, and often the head seems to have settled downward upon the shoulders. In the lower cervical region, a kyphosis is not infrequent; but in the middle and upper regions there is more often an anterior prominence, which may be felt in the posterior wall of the pharynx.

Dorsal disease.—The referred pains are now below the seat of disease, and take the form of intercostal neuralgia or pain in the epigastrium or the abdomen. They are often ascribed to cold, malaria, indigestion, or worms. There is a disposition to assume the prone position while sleeping, and also to lean across a chair or the lap of the nurse. The child walks carefully, holding the spine erect and very stiffly, and exhibits great caution in getting into or out of bed, or in rising from a recumbent position. In the beginning there may be a slight lordosis, or forward curve at the seat of disease, instead of the usual kyphosis or backward projection, but the latter soon takes its place, and with it is seen the compensatory lordosis in the lumbar region.

Lumbar disease.—The first symptoms here are often pain and lameness, referred to one of the lower extremities. This frequently leads to the suspicion that the hip is the seat of disease. In addition to the lameness there may be a tilting of the pelvis to one side, and sometimes quite a distinct lateral curvature of the spine. Referred pains are not so frequent nor so severe as when the upper part of the spine is affected; they may be felt in the groin, in the loin, in the thigh, in the buttock, or in the hypogastrium. The gait and attitude are very characteristic: throwing the shoulders well back, the patient walks stiffly with short steps, holding the spine with the greatest care. He rises from the floor awkwardly and with difficulty. Deformity is not usually so early or so marked as when the disease is dorsal, and often before it is visible there are symptoms due to the formation of psoas abscess,—lameness, flexion of one thigh, and a tumour may be found deep in the iliac fossa or at the upper and inner aspect of the thigh; in both locations it has often been mistaken for hernia.

Physical examination.—Whenever any of the above symptoms are present, the child should be stripped and submitted to a thorough examination, the purpose of which should be to determine, first, the existence of any deformity; secondly, the mobility of the spine; thirdly, the presence of any secondary lesions, such as abscesses or paralysis. The mobility of the spine is best determined by studying the attitude, gait, and posture of the child, and the manner of stooping or rising from the floor. The gait has already been described with the symptoms of lumbar disease. As it has been tersely put, "the child walks with its legs but not with its back." In stooping, the same disinclination to bend or move the spine is seen. It is often impossible to induce the child to stoop at all, and when it does so, to pick up some object, there is acute flexion at the knee and hip, but as little bending of the spine as possible. In rising from the recumbent position the same thing is seen. The posture and attitude of the child will be modified by the position of the disease, and somewhat by the activity of the process at the time; however, by comparing the movements referred to with those of a healthy child, the great difference will at once be apparent. If the symptoms point to cervical disease, a digital exploration of the pharynx for deformity or abscess should be made, and the extremities should be examined for paralysis. If the disease is in the lumbar region, deep palpation of the iliac fossa should be made to discover a psoas abscess, and the passive movements of the thigh should be carefully tested to determine whether there is any resistance to extreme extension, this often being present before the psoas tumour. No matter how clearly the lameness may be at the hip, it should be remembered that this often results from disease of the lumbar spine. If the thigh is flexed and freely movable except in extension, the symptoms are probably the result of psoas irritation, for in hip-joint disease the other movements of the joint are also resisted.

The deformity of Pott's disease is often spoken of as "angular" curvature of the spine. While this is a true description of the disease at an advanced stage, there is often in the early stage only a general curve. Later a slight knuckle is seen from the unnatural projection of a single spinous process. This deformity may increase and finally involve five or six vertebræ. It is usually greatest in the upper dorsal region. A slight prominence, which does not disappear on suspending the patient, is always suspicious.

Tenderness upon pressure over the spinous processes and increased sensitiveness to heat and cold, are rarely present. Pain may sometimes be produced by downward pressure upon the head or shoulders in the axis of the spine. This symptom is not necessary for diagnosis, and the attempt to elicit it is strongly condemned by Gibney, who has seen serious harm follow such a test.

Course of the disease.—Caries of the spine is a very chronic disease, its

course being measured by months or years, but marked, as in all chronic diseases, by periods of remission and exacerbation. An exacerbation may follow traumatism, and is often accompanied by the formation of an abscess. After the disease has lasted from one to three years, the destructive inflammation ceases and repair begins, a cure being finally effected by a process of consolidation of the fragments of the diseased vertebræ, and the production of anchylosis. Relapses are easily excited by traumatism, by improper treatment or by discontinuing the use of mechanical supports before the disease is arrested.

Abscesses.—The frequency with which abscesses occur depends somewhat upon the treatment. Townsend states that of 380 cases, abscess was present in 20 per cent. They are rarely seen earlier than three or four months from the beginning of symptoms, and usually belong to the second year of the disease. They sometimes form with acute symptoms, but more frequently they appear as typical cold abscesses. Those connected with cervical disease are retro-pharyngeal or retro-œsophageal, or they may open externally, usually just above the clavicle, in front of the sterno-mastoid muscle. Those with disease of the lower cervical and upper dorsal vertebræ, are apt to burrow along the spine, appearing in the lumbar region; rarely they may rupture into the œsophagus or the pleural cavity. Those with disease of the lower dorsal or lumbar vertebræ, may open just above the iliac crest posteriorly, or burrow anteriorly between the abdominal muscles, but the usual course is for them to follow the psoas muscle, appearing in the groin just above Poupart's ligament or at the upper and inner aspect of the thigh.

Paralysis occurs in about one half the cases in which the disease affects the lower cervical and upper dorsal vertebræ, but it is rare when the disease is below the middle dorsal region (see Compression Myelitis, page 768).

Prognosis.—The actual mortality of Pott's disease is difficult to state, so many of the consequences of the disease being remote and not fully appreciated until adult life is reached. The general mortality from all causes is from ten to twenty per cent. The causes of death are exhaustion from prolonged suppuration, amyloid degeneration, myelitis, general tuberculosis, and tuberculous meningitis. Sudden death occasionally occurs from pressure upon the cord in the upper cervical region, or from the pressure effects of abscesses in the posterior pharynx or in the posterior mediastinum.

The prognosis as to the amount of permanent deformity, will depend upon the seat of the disease, the time at which treatment is begun, and upon the thoroughness with which it is carried out. The best results as to deformity are obtained when the disease is below the middle dorsal region. With improved methods of treatment begun early, a large number of these patients recover with an insignificant amount of deformity, and some with none whatever.

Diagnosis.—The spinal deformity resulting from Pott's disease may be confounded with rachitic kyphosis or with rotary lateral curvature. Rachitic curvatures (page 225) are usually seen in children under eighteen months of age, a time when Pott's disease is rare; there are other signs of rickets present, and instead of rigidity there is usually undue mobility of the spine. What is true of rickets may be said of all curvatures depending upon malnutrition. Rotary lateral curvature is seen about puberty, rarely in young children except in connection with rickets. A slight lateral deviation of the spine, sometimes seen in the early stage of caries, may resemble a case of incipient rotary curvature. The latter is not attended by pain or rigidity, and is most frequent in young girls from eleven to fourteen years of age.

Other abscesses may be mistaken for those dependent upon vertebral caries. This difficulty is likely to exist in the cases attended by very little spinal deformity. These abscesses are most frequently in the iliac fossa or in the lumbar region, and may be due to perinephritis or appendicitis. The latter are more acute than those depending upon bone disease and usually accompanied by fever. Tumours of the vertebræ or of the spinal cord may give rise to symptoms almost identical with those resulting from compression myelitis due to Pott's disease, but both of these are extremely rare.

Treatment.—The treatment of Pott's disease is both general and local, and neither should be neglected. The constitutional treatment should be similar to that employed in other forms of tuberculosis.

The indications for local treatment are to put the diseased parts at rest, by immobilizing the spine and removing the superincumbent weight of the body. With the great advances made in orthopædic surgery it is no longer necessary to confine these patients in bed, as was formerly practised, to secure this result. It may be accomplished either by plaster-of-Paris, or some other form of jacket, or a properly fitting steel brace. A head-support should be attached to all forms of apparatus, if the disease is above the middle dorsal region. The closest attention to details and much experience in the use of apparatus are required to secure the best results. In perhaps no class of cases has the beneficial results of modern scientific treatment been more apparent than in those of Pott's disease. For the details in regard to the mechanical treatment and the different forms of apparatus, the reader is referred to works on general or orthopædic surgery.

Articular Ostitis of the Hip—Hip-Joint Disease—Morbus Coxarius.—In early childhood this generally begins as a chronic ostitis in the head of the femur, starting near the epiphyseal line. Exceptionally, and according to Gibney, oftener in older children, it begins in the acetabulum. The pathological process, as well as the clinical history, is generally described as consisting of three stages. In the first stage—that of ostitis—the lesions are limited to the bone; in the second stage—that

of arthritis—all the joint structures are involved, and in this stage suppuration usually occurs; in the third stage there are breaking down and absorption of the head and sometimes of the neck of the femur, which, with destruction of the ligaments, lead to marked displacement of the parts from muscular contraction. The disease may be arrested in the first or in the second stage, or it may continue through all three stages.

Symptoms.—Clinically, the usual duration of *the first stage* is three or four months; it may last only for a few weeks, it may extend over two or three years, and the disease may be arrested in this stage. The onset is usually very gradual, and the symptoms are often considered of trivial importance until they have continued for some weeks. Generally the first thing noticed is slight lameness, due to stiffness of the joint. In the beginning this may be seen only in the morning, wearing off during the day. It may be accompanied by some tenderness about the hip and a disinclination to walk. A little later the child complains of pain, which is most frequently referred to the front of the knee or the inner aspect of the thigh, but only in rare cases to the hip itself. This is slight at first, but gradually increases in frequency and severity, and soon there are added the "starting pains" at night, which are one of the most characteristic features of early hip-disease. These pains are produced by a sudden spasm of the muscles during sleep. The child often cries out sharply without waking, sometimes wakes with a cry; this is often repeated several times during the night. Soon restlessness and fretfulness during the day are present. The lameness, which at first was slight and occasional, or noticed only in the morning, comes to be a constant symptom, and week by week increases in severity. The evolution of these symptoms may take only a few weeks, but sometimes they come and go in the most inexplicable manner during a period of several months, or even one to two years, before they are fully developed.

Physical examination.—Every child with a suspicious lameness, or with pains like those mentioned, should be stripped and submitted to a thorough examination. The first points to be observed on inspection relate to the general contour of the hip; every prominence and depression should be carefully noted. Then the attitude and gait should be studied; and finally all the functions of the joint should be carefully tested, and the limbs measured, to determine the existence of shortening or atrophy. At every step a comparison should be made with the sound limb. The contour of the hip is changed quite uniformly: there are broadening and flattening of the whole gluteal region; the trochanter is unnaturally prominent; the gluteal fold is shortened, and often single instead of double. There is no characteristic position of the limb in this stage. There is atrophy of the thigh and often of the calf. In Fig. 145 is shown the appearance of a typical case in the full development of the first stage. In walking, the child favours the diseased side, throwing the weight as

much as possible upon the sound limb; but all these symptoms are of much less importance for diagnosis than is an examination of the functions of the joint.

For this purpose the child should be placed upon a table upon its back, and the various movements of the hip—abduction, adduction, flexion, extension, and rotation—should be executed, first with the sound limb and then with the suspected one, the two being carefully compared at every point to determine the degree of motion allowed. It is not necessary that force should be employed or pain inflicted. If the symptoms have existed for some weeks, there is generally a limitation of motion at the hip in all directions, but first usually in abduction, rotation, or extension. In more advanced cases, no motion whatever may be permitted at the joint, the pelvis tilting with the slightest movement of the femur. This fixation of the hip is due to tonic muscular spasm. Crowding the articular surfaces together, by pressure upon the heel or trochanter, produces pain, which is usually referred to the joint. This test should be carefully made, lest injury be inflicted. Gibney cautions against examinations under ether, since in this way serious injury may be done unconsciously.

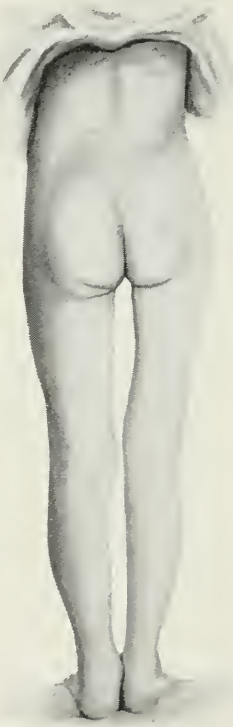

Fig. 145.—Hip-joint disease, at the end of the first stage, showing muscular atrophy, prominence of the trochanter, flattening of the gluteal region, and a single gluteal fold.

Second stage.—This has been called the stage of arthritis. Its existence may be assumed when the limb takes the position of marked permanent deformity, which is due at this period to muscular action, not to destructive bone changes. The transition from the first to the second stage is in most cases a gradual one, and the line between the two can not be sharply drawn. Sometimes, however, it is rapid, and marked by a sharp exacerbation of all the symptoms. This may indicate a sudden perforation of the joint, and the rapid development of suppurative arthritis. Such is the usual result when an abscess which has been slowly forming in the bone, opens into the joint; or acute joint inflammation may be lighted up without so evident a cause. Sometimes the pus reaches the surface below the capsular ligament, and the joint remains intact. An acute exacerbation is indicated by increased pain, excessive tenderness about the hip, often by inability to walk, or even to bear any weight upon the limb, and frequently by fever. The position assumed by the limb is now fairly

characteristic. The foot is generally everted, the thigh slightly flexed and rotated outward, and the limb apparently lengthened. There may be infiltration anywhere about the hip, due to the formation of an abscess. The muscular spasm is so great that the joint is locked,—no motion whatever being allowed. Abscesses may form at any point about the hip; they are especially frequent at the upper and outer aspect of the thigh, and may burrow long distances before reaching the surface. The duration of the second stage also is indefinite, but it usually lasts from a few months to a year, or the disease may be arrested in this stage.

Third stage.—There is now marked deformity, which is the result of muscular contraction after absorption of the head and sometimes the neck of the femur, and destruction of the ligaments. The position of the limb is a very constant one, and resembles that present in dislocation upon the dorsum of the ilium. There is shortening of from one to four inches; the thigh is strongly flexed, adducted, and rotated inward, and the foot is inverted; the trochanter lies against the outer surface of the ilium, and is above Nélaton's line. In this position the joint may become anchylosed. The displacement usually comes on gradually, but it is sometimes so sudden as to be mistaken for a true dislocation, although the latter is exceedingly rare in the course of hip-disease.

There is now marked atrophy of all the muscles of the limb, and the thigh may be two or three inches smaller than its fellow. No motion at all is usually allowed at the hip, but this is compensated for to some degree, by the exaggerated mobility of the lumbar spine. The spinal curvature—lordosis—is very marked both upon standing and walking. The duration of this stage may be several years. From time to time exacerbations occur, often excited by falls, and accompanied by the formation of new abscesses. In protracted cases, all the soft parts about the hip may be seamed with cicatrices from old sinuses. After the disease has gone on to the third stage, cure can take place only by anchylosis.

Diagnosis.—The important point in the early diagnosis of ostitis of the hip, is the gradual evolution of the symptoms, the most characteristic of which are lameness, starting pains at night, and impairment of all the functions of the joint. Mistakes in diagnosis most frequently arise from a failure to obtain a careful history, and from relying too much upon the symptoms of lameness and deformity. The essentially chronic character of the disease should constantly be borne in mind. In the vast majority of cases, with a careful history, and a thorough examination, there can be but little doubt as to the diagnosis except at the very outset. The proportion of obscure and irregular cases to those following the regular course, is small.

In the early stage, hip-joint disease may be confounded with a strain of the joint, with muscular rheumatism, poliomyelitis, periostitis of the shaft of the femur, phlegmonous inflammation in the neighbourhood of the

joint, or with caries of the lumbar spine. In the second stage there is even less difficulty in diagnosis, although abscesses resulting from perinephritis or appendicitis have been mistaken for those arising from hip-disease. In the third stage, a mistake is almost impossible.

Prognosis.—This is to be considered both with reference to life and limb. The records of the Hospital for Ruptured and Crippled show the mortality of hospital patients with hip-disease to be nearly 25 per cent. This includes deaths directly or indirectly traceable to the disease. The causes are nearly the same as in caries of the spine,—exhaustion from prolonged suppuration, amyloid degeneration, and general tuberculosis or tuberculous meningitis.

Under the most favourable conditions, the disease may be arrested in the first stage, and recovery occur without lameness or any noticeable impairment of the joint functions. This result, however, is not often obtained, because the disease is usually well advanced before it is recognised, or because of the difficulty in the way of carrying out all the details of treatment in the best possible manner. If the disease has advanced to the second stage, and suppuration has occurred, there always results some impairment of the joint functions; usually there are decided lameness and marked muscular atrophy, but very little shortening or deformity, provided the limb has been kept in the proper position. If the disease has advanced to the third stage, there are always marked shortening, deformity, and lameness.

Treatment.—The indications for constitutional treatment are the same as in caries of the spine. The purpose of local treatment is to secure constant and complete rest for the diseased parts, and to prevent deformity. Rest is secured by overcoming the muscular spasm by means of extension, by immobilizing the joint, and by transferring the weight of the body, in walking, from the hip to the perinæum. All these indications are now met, while the patient is up and about, by the use of the most approved apparatus. Formerly, rest and immobilization could be secured only by keeping the patient in bed, with the use of the weight and pulley. The general opinion of orthopædic surgeons at the present day is against excision, except in cases where, in spite of treatment by apparatus, the disease has advanced to the third stage, and in cases where life is threatened from prolonged suppuration and exhaustion. Under these conditions, excision should be performed; but early excision gives results very much inferior to those obtained by mechanical and constitutional treatment.

Articular Ostitis of the Knee—Knee-Joint Disease—White Swelling.—Ostitis of the knee usually begins in one of the condyles of the femur, the inner much oftener than the outer one; less frequently it begins in the head of the tibia. The pathological process is very much like that at the hip. There is in the first stage a central ostitis accom-

panied by infiltration and expansion of the part of the bone affected. The disease may remain limited to the bone, the inflammatory products becoming encapsulated, or softening and breaking down may occur, with the formation of an abscess. Gradually the process extends outward, and the periosteum and the soft parts are involved. The disease may invade the joint itself in a destructive inflammation, or pus may escape externally without seriously involving the joint structures. The degree to which the joint is involved, varies much in different cases; there may be only a simple synovitis, a suppurative arthritis, or a destruction of the cartilages and articular ends of the bones, synovial membrane, and ligaments, so that in the advanced stage all traces of a joint structure are lost.

If the process remains limited to the bone, recovery may take place with very little impairment of the joint functions. If suppuration in the joint has taken place, there will be more or less stiffness and fibrous or bony anchylosis. When there is destruction of the ligaments and articular ends of the bones, the limb assumes a characteristic position,—the joint is flexed, the tibia is displaced backward and rotated outward, and there is marked over-riding of the femur. Bony anchylosis in this position is often seen.

Symptoms.—The earliest symptoms of disease at the knee are usually a slight stiffness of the joint, with a disposition to flexion and slight lameness. At first these symptoms are noticed only occasionally; finally they become constant and there is pain, which is usually referred to the knee. In some cases there are "starting pains" at night, although these are less constant and less severe than in hip-disease. Swelling is noticed early, as the diseased parts are so superficial. At first this is chiefly of the bone itself; the condyle, usually the inner one, is enlarged and elongated, often to a marked degree, before there is any infiltration of the soft parts. Later there is a general fusiform swelling, involving the entire joint and effacing all the normal outlines. Some tenderness upon pressure over the bone affected is present quite early, and there may be atrophy of the muscles of the thigh and calf. The knee is flexed and slightly rotated outward, the position which secures the most complete relaxation of the joint structures. In some cases there is seen the characteristic swelling due to distention of the synovial membrane. Abscesses may form anywhere about the joint; very frequently they burrow beneath the tendon of the quadriceps extensor as far as the middle of the thigh. Gradually the deformity increases until the leg may be flexed at a right angle, and rotated outward over an arc of twenty or thirty degrees.

The course of the disease resembles that of ostitis of the hip and the spine. During periods of remission, pain and tenderness often subside for several months so completely as to lead to the supposition that the disease has been arrested. An exacerbation is often excited by a fall or a strain of the joint, or it may follow an attack of acute illness. The disease may

then progress rapidly and abscess after abscess form, with extensive destruction of all the joint structures and the production of permanent deformity.

Prognosis.—The danger to life is considerably less than in disease of the hip or spine. Death, however, results from the same causes—exhaustion, amyloid degeneration, and general tuberculosis or tuberculous meningitis.

With an early diagnosis and proper treatment the disease may, in a considerable proportion of cases, remain limited to the bone, and the resulting lameness and deformity be very slight; but otherwise a certain amount of lameness results from the stiffness of the joint. This may be due either to fibrous thickening or to bony anchylosis. Nearly all patients are able to walk without crutches, and if proper treatment has been carried out there is neither marked shortening nor deformity, although there is always great muscular atrophy.

Diagnosis.—The important symptoms for diagnosis, are the gradual onset, the early swelling which is due to enlargement of the bone, and the constant lameness and deformity. The disease may be confounded with rheumatism, with synovitis, and even with scurvy. In all these cases the resemblance exists only during the period of exacerbation. A careful history, however, will usually clear up the diagnosis.

Treatment.—The general treatment is the same as in other forms of joint disease. The indications for local treatment are the same as in hip-disease,—viz., to immobilize the affected limb and prevent deformity. This is accomplished by a form of apparatus which transfers the weight of the body from the joint to the perinæum, and which overcomes the muscular spasm which produces flexion and inward rotation of the joint. As in hip-disease, the results of mechanical and constitutional treatment are decidedly better than early operative measures; but late operations are indicated under the same conditions.

Tuberculous Osteo-Myelitis.—This disease is rarely seen except in the short tubular bones, most frequently those of the hand and fingers. From this fact it is often called *scrofulous* or *tuberculous dactylitis*. It is described by many writers under the name of *spina ventosa*. Unger* gives the following figures showing the frequency with which the different bones were affected: fingers in 43, toes in 3, metacarpus in 41, metatarsus in 14, radius in 2, ulna in 2, tibia in 3, jaw in 3. The first phalanx of the index finger is the bone which is most frequently the seat of disease. In the majority of cases the process is confined to a single bone, although it is not rare to see five or six affected. In such cases the disease is seldom symmetrical. The process is a chronic inflammation, beginning in the centre of the bone with the deposit of tuberculous material. The swelling

* Archiv für Kinderheilkunde, Bd. ii, 233.

which follows causes an expansion of the bone and thinning of the shaft, until a mere shell may remain. The later changes are, inflammation of the periosteum and the soft parts, the formation of abscesses and sinuses, necrosis, the exfoliation of sequestra, etc. The entire disease lasts from one to three years, and causes in most cases marked deformity.

Tuberculous dactylitis is essentially a disease of early childhood, being seen most frequently during the second and third years. In a considerable proportion of the cases there is a history of inherited tuberculosis. It usually exists as the only tuberculous lesion in the body, but occasionally it is associated with tuberculosis of the hip, knee, ankle, or spine.

Symptoms.—Tuberculous dactylitis usually begins as a painless enlargement of one of the phalanges, most frequently the first one of the index finger. It may be two or three months before it is of sufficient size to

Fig. 146.—Tuberculous dactylitis of the first phalanx of the index finger.

attract much attention. Exceptionally the inflammation is a more active one, and is accompanied by both pain and tenderness. The swelling is quite characteristic; it is smooth, hard, uniform, and generally spindle-shaped, involving the entire phalanx of the affected finger. The appearance of a severe typical case is shown in Fig. 146. Later there is discolouration of the skin, and usually there is suppuration. The abscess generally opens at the side of the finger, and a curdy pus is evacuated. If the opening is enlarged by an incision there is found a cavity partly filled with caseous matter, and dead bone is felt, and perhaps a loose sequestrum. The cavity is surrounded by a thin shell of new bone, which is formed from the periosteum. If no operation is done the discharge continues for weeks or months, other abscesses often form, and finally several small

sequestra are exfoliated,—sometimes a single large one, which is the shell of the diseased phalanx almost entire.

In some cases the disease is arrested before necrosis occurs, but in the majority this is not so. After the wounds have all healed the finger remains shortened, deformed, and often useless. In some cases the disorganization is so extensive that amputation is necessary.

Diagnosis.—The recognition of dactylitis is usually easy, but as symptoms identical in almost every particular may be seen in a syphilitic inflammation, it is often difficult to tell with which of the two forms one has to deal. The tuberculous form is very much more frequent; it may occur in a patient with tuberculous antecedents, or it may be associated with other tuberculous lesions. Syphilitic cases are distinguished by the fact that the lesion is more frequently multiple, that it is often symmetrical, and that other manifestations of syphilis are generally present. It is affected by anti-syphilitic remedies, which is not the case in the tuberculous variety.

Treatment.—Painting with iodine and like measures are useless. The diseased part should be kept at rest,—if a finger, by the application of a splint. Every means should be taken to build up the patient's general health, as this is the most effective way to influence the local process. The general verdict of surgeons is against early excision as a means of arresting the disease. Abscesses should be opened early and freely, all diseased bone removed, the finger kept in proper position, and the wound treated according to general surgical principles. Under almost any treatment the disease is a protracted one, and rarely lasts less than a year.

THE SYPHILITIC DISEASES OF BONE.

The bone lesions of hereditary syphilis are not infrequent, but were long unrecognised, and have only within comparatively recent times been fully understood.* They may be divided into two groups,—those occurring with the early symptoms, and those which belong to the late manifestations of the disease.

Acute Epiphysitis.—This is the most frequent variety of bone disease in early hereditary syphilis. It may begin even in intra-uterine life, and it forms one of the most characteristic lesions of the disease. To some degree it is almost invariably present in syphilitic fœtuses and in syphilitic infants who are still-born.

In the early stage, there is an increase in the cartilage cells and delayed ossification. Later, a line of softening forms at the epiphyseal junction, which may cause loosening of the cartilages and ultimately complete separation of the epiphysis from the shaft, by the formation of granula-

* See Taylor, Bone Syphilis in Children, New York, 1875; also G. Wegner, Virchow's Archives, Bd. 1, Heft 3.

tion tissue between them. In cases receiving proper treatment, recovery may take place with good union, perfect function, and without any deformity. In other cases degenerative changes continue, and infection with pyogenic germs may be added. The periosteum and the soft parts in the neighbourhood are now involved, with the formation of external abscesses; or the disease extends to the medullary cavity, giving rise to acute osteo-myelitis, which may lead to necrosis; or the contiguous joint may be invaded, causing an acute suppurative arthritis (page 835). This last result is more likely to occur where the epiphysis joins the shaft within the joint cavity. The large joints are usually affected, and the

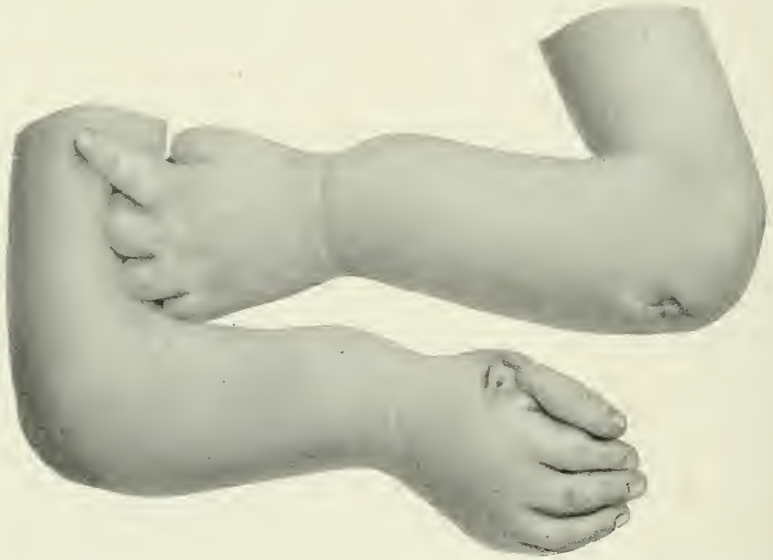

Fig. 147.—Syphilitic bone disease in a boy four years old. The lower end of the radius of both arms is enlarged as a result of former epiphysitis: there are sinuses leading to dead bone over the metacarpal bone of the right thumb, and over the upper extremity of the left ulna. The last two are recent lesions.

lesions are frequently symmetrical. Acute suppurative arthritis may occur independently of changes at the epiphysis; but even when these are seen in syphilitic infants they are to be regarded as of pyæmic rather than of syphilitic origin. Secondary to the changes at the epiphysis, there are periostitis and inflammation of the soft parts. Periostitis of the shaft is rare in early infancy,

The bones most frequently the seat of acute epiphysitis are the humerus, radius and ulna, although any of the long bones may be affected.

Symptoms.—The early symptoms are usually quite acute, and appear during the first six weeks of life; they may precede any other manifestations of syphilis. In some cases there is first noticed an inability on

the part of the child to move the limb, which may easily be mistaken for paralysis. It is, in fact, often described as "syphilitic pseudo-paralysis." The limb lies perfectly motionless, and any attempt at passive movement causes evident pain. There is tenderness on pressure and soon swelling is seen, both being most marked at the epiphyseal line. If the bone affected is superficially situated, as the lower epiphysis of the humerus, radius, or tibia, swelling is very apparent, while it may be scarcely perceptible at the upper epiphysis of the humerus. The swelling is usually cylindrical and moderate in degree, being limited to the extremity of the bone. In the more severe cases it may involve a great part of the limb. Abscess may form and separation of the epiphysis take place, so that crepitation may be obtained by moving the limb. Separation of the epiphysis not infrequently occurs even when there has been no suppuration.

In the milder cases, or those which have been subjected to active treatment, both the swelling and the tenderness subside rapidly without suppuration; and even though the epiphysis has separated from the shaft, it speedily unites. Where pseudo-paralysis has been the chief symptom, very rapid improvement occurs under treatment, and usually complete recovery of function in two or three weeks. If the disease extends to the joint, or if osteo-myelitis develops, the case is almost certainly fatal.

Diagnosis.—This is usually easy, from the age of the patient—generally under three months—the early prominence of pain and apparent loss of power, with the later appearance of swelling and signs of inflammation at the epiphyseal junction. In all these respects the disease closely resembles scurvy; but the latter is rare before the eighth or tenth month, there is usually a history of the long-continued use of some proprietary infant food, and it is cured by dietetic treatment alone.

The apparent loss of power may lead to the diagnosis of birth palsy, especially of the upper-arm type (page 110). The presence of acute pain and tenderness, the absence of the characteristic deformity, and the prompt recovery under constitutional treatment, usually make the distinction between the two conditions an easy one.

Treatment.—This is the same as in all early syphilitic manifestations, for which see the article on Syphilis. Locally, the part requires in the early stage only protection and rest. Should suppuration occur in the neighbouring joint, or should osteo-myelitis develop, these conditions should be treated surgically as they are when due to other causes.

Chronic Osteo-Periostitis.—This is the usual form of bone disease which is seen in late hereditary syphilis, and it is one of the most frequent and most characteristic lesions of that stage of the disease. Occurring in adults, this would be classed as a tertiary symptom. Chronic syphilitic osteo-periostitis is rarely seen before the third year, and most of the cases occur between the fifth and fourteenth years. The most frequent seat of disease is the tibia, and next to this the bones of the forearm and the

cranium. The following is the frequency with which the different bones were affected in the series of cases reported by Fournier:* tibia in 91 cases, ulna in 22, radius in 15, cranium in 16, humerus in 12, all others in 37. The process may result either in a diffuse or a localized hyperplasia of bone or in necrosis.

The typical changes are seen in the tibia. The shaft of the bone is

Fig. 148.—Syphilitic disease of the tibia, showing the sabre-like deformity, in a boy nine years old.

principally or solely affected. There is often produced a very characteristic deformity, consisting of a forward curve of the anterior border of the tibia, which has been compared to a sabre blade (Fig. 148). In some cases the bone is bent inward at its lower third, resembling somewhat a rachitic curvature (Fig. 149). Sometimes the entire shaft of the bone is affected, and it may be enlarged to nearly twice its normal dimensions.

* Syphilis Héréditaire Tardive, Paris, 1886.

At other times the swelling is chiefly near the epiphysis, where large bosses may form of sufficient size to interfere with the functions of the joint. Instead of affecting the bone uniformly, the disease often affects only certain parts, leading to the formation of large nodes which are more likely to be followed by necrosis than are the other lesions. In most of the cases the process is purely a hyperplastic one, leaving the bone permanently enlarged. Less frequently, there occur gummatous deposits

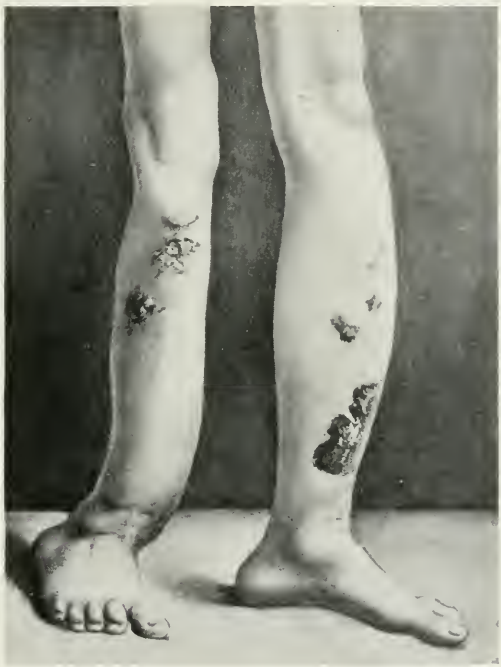

Fig. 149.—Syphilitic disease of both tibiæ. The left shows a general enlargement of the bone, the characteristic curve of its anterior border, with ulcers due to necrosis. The enlargement of the right tibia is less marked, and there is a pseudo-rachitic curve at its lower third. Cicatrices near the knee mark the site of former ulcers. (After Fournier.)

in or beneath the periosteum, which may soften, suppurate, and lead to superficial necrosis, with the formation of sinuses that remain open until the sequestrum is exfoliated (Fig. 150). Syphilitic deposits sometimes take place in the interior of the bones, generally near the articular ends; these may soften and break down with abscesses, sinuses, etc., very much after the manner of a tuberculous inflammation (Fig. 147).

The lesions of the other long bones are essentially the same as of the tibia. They are nearly always symmetrical and often multiple. In a case recently under observation in a boy of four years, the disease involved both tibiæ, both radii, the right ulna, the left metatarsus, and the metacarpal bone of the left thumb. The course of syphilitic osteo-periostitis

is very chronic, and some permanent deformity is the rule, unless cases come very early under treatment.

When affecting the bones of the cranium the disease usually takes the form of a gummatous periostitis, which leads to the formation of large nodes. These may remain as permanent deformities, or they may break down and suppurate, with necrosis of one or both tables of the skull. This may be followed by inflammation of the dura, the pia, and even of the brain itself.

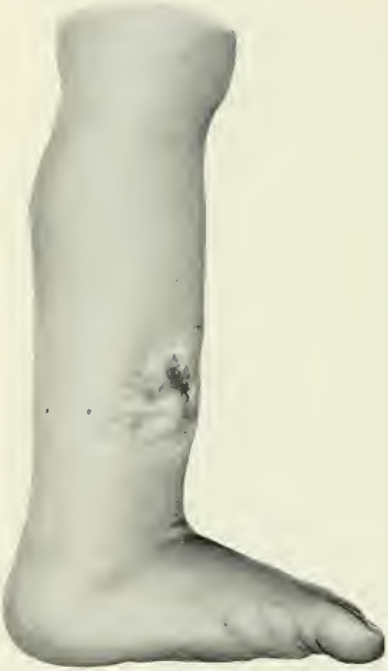

Fig. 150.—Syphilitic necrosis of the tibia, showing moderate enlargement of the bone and a sinus. (From the same patient as Fig. 147.)

Symptoms.—When the long bones are affected, the symptoms are pain, tenderness and deformity. These come on very gradually, and often the deformity is noticed before either pain or tenderness is sufficiently marked to attract attention. The pain is regularly worse at night, and often felt only at that time; it may be mild and occasional, or so severe as virtually to prevent sleep. There is tenderness on pressure over the bones affected, the acuteness of which will depend upon the activity of the process. When suppuration occurs, it comes very slowly, and never with symptoms of acute inflammation. Sinuses usually continue to discharge until a sequestrum is exfoliated. The course of the disease is very tedious, and the whole duration is usually several years.

When the cranium is affected, there are seen the irregular nodes, especially upon the frontal and parietal bones. They are from one to two inches in diameter, and project from one eighth to one fourth of an inch above the general outline of the skull. There may be pain, tenderness, softening, suppuration, and necrosis, as in the long bones.

Diagnosis.—It is so very rare that disease of the bones of the cranium is due in childhood to any other cause than syphilis, that this disease may always be assumed to exist if traumatism can be excluded. The bosses upon the cranium in rickets (page 226) are always large, smooth, and regular in position, and belong to infancy.

Syphilitic disease of the long bones is recognised by the nocturnal pain, the tenderness and peculiar deformity, and by the association of other late manifestations of syphilis,—i. e., the peculiar notched teeth,

the interstitial keratitis, the enlarged epitrochlear glands, etc. Tuberculous disease generally affects the articular ends of the bones; syphilis nearly always the shaft. The diffuse hyperplasia of the tibia and the sabre-like deformity of its anterior border, are rarely if ever due to any other cause than syphilis.

The deformities of the long bones have in some cases a certain resemblance to those due to rickets, but on close examination there are seen striking differences. The epiphyseal enlargement at the wrist in rickets affects both bones (Plate V, page 222); in syphilis it is usually of one bone only (Fig. 147). The differences between rachitic curvatures of the tibia and the deformities from syphilis may be readily seen by comparing Figs. 38, 39, and 40 (pages 227 and 228) with Fig. 149.

Fig. 151.—Multiple syphilitic dactylitis, in a child two years old. The disease affects the first phalanges of both thumbs, both little fingers, and the index finger of the left hand.

Treatment.—The constitutional treatment of these lesions is the same as that of the other late manifestations of syphilis,—mercury and the iodide of potassium; for details, see the chapter on Syphilis. Surgical treatment is required in cases which terminate in necrosis, whether of the cranium or the extremities. They are to be managed like the same conditions in adults.

Syphilitic Dactylitis.—This belongs to a somewhat earlier period of syphilis than the disease just described, and is usually seen in children under five years old. It is not a frequent manifestation of syphilis, and as compared with tuberculous dactylitis it is rare. It was first fully described by Taylor (New York). The symptoms closely resemble the tuberculous form. It may begin as a periostitis but more frequently as an osteo-myelitis. Like the tuberculous form it usually goes on to suppuration and necrosis. According to Taylor, dactylitis is more often single than multiple, but in my own cases several phalanges have generally been

involved, and the lesions have often been symmetrical (Fig. 151). In one case, the first phalanx of every finger of both hands was affected. This occurred in a child nine months old who was under observation for over two years, and who presented during this period almost every lesion of hereditary syphilis.

The symptoms and course of syphilitic dactylitis are essentially the same as in the tuberculous form. The differential diagnosis is considered with the latter disease (page 851). The prognosis is much the same in the two varieties, with the exception that in the early stage the syphilitic cases may often be arrested by constitutional treatment. This is the same as in other late lesions of syphilis. The same local treatment should be employed as in the tuberculous cases.

CHAPTER V.

DISEASES OF THE SKIN.

The skin at birth is covered with a whitish sebaceous secretion, the vernix caseosa. The skin itself is of a deep purplish colour, which changes to a bright red over the face and trunk in a few minutes, with the establishment of normal respiration, and in a few hours the whole body has the same tint. This excessive redness slowly fades during the first month, at the end of which time the skin has assumed the pale pink of infancy. On the third or fourth day there are usually seen the first signs of icterus; this generally fades by the end of the second week.

The epidermis which is present at birth soon loosens and is thrown off. This normal desquamation usually begins upon the fourth or fifth day, and is completed in ten days or two weeks. If the skin is frequently oiled and properly bathed, desquamation is scarcely noticeable unless a close examination is made. In some infants, especially those who are delicate and cachectic, it is very much more marked, and closely resembles that seen in scarlet fever. Ritter has described an *exfoliative dermatitis* of the newly born, appearing generally during the second and third weeks, a condition which is regarded by Kaposi as simply an exaggeration of normal physiological desquamation. This process may be mistaken for that due to hereditary syphilis; the latter, however, is rarely general, appears later, and is much more prolonged.

Perspiration is rarely present before the end of the fourth month, and is then seen only upon the forehead. In healthy infants it is scarcely noticeable during the first year. Copious perspiration is most frequently a symptom of rickets; less marked perspiration may occur with any general weakness or during acute illness.

CONGENITAL ICHTHYOSIS.

Congenital, or more properly fœtal, ichthyosis, sometimes known also as diffuse keratoma, is a rare disease, characterized by the formation, usually all over the body, of a thick, horny epidermis resembling parchment. This is divided by fissures or shallow furrows into irregular patches; sometimes these are two or three inches wide, at others as small as a pin's head. The disease begins in the early months of fœtal life, and is an abnormality in the development of the skin, there being an excessive proliferation of the layers of the epidermis.

Symptoms.—In the gravest form of the disease the child often lives but

FIG. 152.—Congenital ichthyosis in a child ten months old. The large scaly patches are well shown on the lower part of the right chest and abdomen, and the constricting bands upon the legs. (From a photograph by Dr. Cabot.)

a few hours, and rarely more than a week. The openings of the nostrils and the ears may be occluded by the excessive production of epithelial cells. The eyes are in a condition of ectropion, and there are often deformities of the mouth and other orifices due to the contractions of the skin. The nails and hair are usually imperfectly developed. The body seems incased in a hard, horny covering, and looks as if it had been varnished or covered with collodion. The skin cracks or splits and the edges curl up, an appearance which has been aptly compared to the skin of a boiled potato.

In the milder form, the duration of life is indefinite, depending upon

the degree of development of the disease; but even in such cases there are frequently seen the deformities at the orifices of the body, and there may also be a continued exfoliation of the epidermis in large irregular patches. After this has separated, the skin beneath appears red and moist, but gradually becomes dry, hard, and shining, slowly contracting until it splits in various directions. In a case recently under observation in the Babies' Hospital,* a picture of which is shown in the accompanying illustration (Fig. 152), it was stated by the mother that during the first ten months of life complete exfoliation of the skin had occurred in the course of every two or three months.

The outlook is bad in all cases; in most of the severe forms death occurs in infancy, but in some of the milder ones, life may be prolonged throughout childhood. The "alligator boy" of the Dime Museum is an example of this class.

Treatment.—The indications are to keep the skin moist and soft by the use of oils, continuous baths, etc., and to prevent infection by perfect cleanliness. Although a certain amount of improvement usually follows these measures, a cure is not to be expected.

MILIARIA.

The term miliaria is applied to an obstruction of the sweat glands, which may occur either with or without inflammation. The non-inflammatory form is known as *sudamina*, the inflammatory forms as *miliaria rubra*, *miliaria vesiculosa*, and *miliaria papulosa*.

Sudamina.—In this form there is no inflammation. The sweat ducts, according to Crocker, are blocked by an accumulation of epithelial cells while no perspiration is going on; and when the process is restored the fluid, being unable to escape, accumulates in the form of tiny vesicles. These appear like small pearly bodies very closely set, and disappear in the course of a few days by absorption. Fresh crops may appear from time to time. Sudamina may be seen in any of the continued fevers or exhausting diseases. It requires no treatment.

Miliaria Rubra.—This condition, also known as *red gum*, *strophulus*, etc., is a sweat rash, usually seen in young infants as the result of excessive clothing. It is most frequently observed upon the cheeks and neck, often upon the side of the face upon which the infant sleeps, or the side held against the mother's body while nursing, if this is done upon only one breast. The eruption consists of scattered red papules, sometimes with tiny vesicles. Miliaria rubra is an inflammation about the sweat

* This case has been fully reported by Cabot, New York Medical Record, July 6, 1895. For fuller description of the disease, see Ballantyne, Diseases of the Fœtus, vol. ii, 1895; also Archives of Pædiatrics, April and June, 1894.

glands, the result of which is a retention of their secretion. There is generally little or no itching. The treatment consists in the removal of the cause, and the application of some absorbent powder, such as boric acid and starch.

Miliaria Papulosa (Lichen Tropicus, Prickly Heat, etc.).—This is the most common and most important variety of miliaria. There is in this disease an obstruction of the sweat glands by inflammatory products. The lesion consists in the formation of bright-red papules, which are very closely set, the summits of some of them being surmounted by tiny vesicles, and here and there in severe cases even small pustules may be seen. If not interfered with by scratching, the vesicles dry up without rupture, and are followed by a slight desquamation. Where there is much scratching, an eczematous condition may result. Miliaria papulosa comes out with great rapidity, especially upon the neck, forehead, back, and chest. It is accompanied by an almost intolerable itching and stinging sensation. Over other parts of the body profuse perspiration occurs. The disease is produced by very hot weather and excessive clothing. Although the duration of a single attack is but two or three days, in susceptible patients it may keep recurring for weeks, being exceedingly intractable. Where there is much scratching the resulting eczema is very troublesome. It is not infrequently followed by furunculosis.

The diagnosis of miliaria rubra and miliaria papulosa is usually easy. They are distinguished from eczema by the suddenness with which they appear, by the associated sweating of other parts of the body, by the transitory character of the eruption, and by the fact that the rash never occurs in circumscribed patches. Prickly heat sometimes resembles the rash of scarlet fever, but the fact that the tiny papules are in some places crowned by vesicles and that constitutional symptoms are absent, usually make the distinction an easy one.

Treatment.—Prickly heat is to be prevented by light clothing, frequent bathing, and the plentiful use of a good toilet powder, such as boric acid and starch. During an attack, the bowels should be freely opened by calomel or a saline, and secretion of the kidneys stimulated by the use of nitrate of potassium or the sweet spirits of nitre. The skin should be protected against the irritation of flannel undergarments by the interposition of silk or linen. When the inflammation is at its height, relief is obtained by the application of a calamine and zinc lotion (page 869), or by a dilute solution of the acetate of lead; carbolic acid may be added to either, where the itching is intense. In some cases powders are preferable to lotions. One of the best is the stearate or the oxide of zinc, twelve parts; bismuth, three parts; powdered camphor, one part; or equal parts of starch and boric acid may be used, or simply rice flour. All of these must be very freely applied. The diet should be light and fluid, and if milk is the food it should be considerably diluted.

SEBORRHŒA.

Seborrhœa is considered by dermatologists generally, as a functional disease of the sebaceous glands; although Unna regards all such cases as inflammatory, and classes them as seborrhœic eczema, which is of parasitic origin (page 865). The disease may affect almost any part of the body, and children of any age, but the most frequent form is that which is seen upon the scalp in young infants. This is the most important variety, and the only one which will be here considered.

Seborrhœa of the scalp is characterized by the formation upon the vertex, of dirty-yellow crusts, which are soft, greasy, and friable. They are composed of epithelial cells, fat-globules, and granular masses, to which is always added dirt. In neglected cases the hairy scalp is nearly covered by a dense crust, which may be as thick as heavy pasteboard. If the crusts are removed the underlying scalp may be found perfectly healthy, but more frequently, in cases of long standing, it is eczematous. The eczema is set up by the decomposition of the exudation, or by the efforts to remove the crusts by such means as the fine-toothed comb, commonly employed in domestic practice. There is little tendency to spontaneous improvement or recovery, and the condition often lasts for months. Every seborrhœa should be treated, for when neglected it furnishes a favourable soil for the development of eczema.

Treatment.—Only local measures are required. The crusts are first to be softened with oil, and then removed by washing thoroughly with warm water and soap, after which an ointment of resorcin (resorcin, gr. x; ungt. aquæ rosæ, ℥ j) or sulphur (precipitated sulphur, ʒ j; lanoline, ℥ j) should be applied. The oil and soap and water are repeated every few days, or as often as the crusts form. In the meantime the scalp is kept covered with the ointment.

ECZEMA.

Eczema may be defined as a catarrhal inflammation of the skin. It is the most frequent and altogether the most important disease of the skin in early life. The scope of the present work permits only a discussion of such features and varieties as are peculiar to infants and young children. The eczema of older children does not differ in any essential points from that of adults.

Etiology.—The conditions in infancy which predispose to eczema are, first, that the skin is extremely delicate, and hence more easily affected by external irritants and micro-organisms; secondly, its more intense glandular activity. While all children are susceptible, there are certain ones in whom the susceptibility is very marked, and in them the slightest amount of external irritation, or the most trivial disturbance of digestion may produce a severe eruption. It was formerly the fashion to class

eczema of the face and scalp among the manifestations of infantile "scrofula." It is true that certain infants are prone to eczema, as others are to catarrhal processes of the mucous membranes, but no more can be positively affirmed. We certainly can not connect eczema with any single diathetic condition; but it is much more often seen in children with gouty antecedents than in others; or to state it differently, the most frequent manifestation of gout during infancy is the tendency to eczema. Children of rheumatic families are also prone to the disease. Eczema of the face is common in fat, healthy-looking infants, and is seen both in those who are nursing and in those who are artificially fed. It also occurs in flabby, poorly nourished children, but rarely in those suffering from marasmus.

The exciting causes of eczema may be external or internal. Of the former the most important are heat, cold dry air, and winds—as in the familiar chapping of the face—the use of hard water or of strong soaps in bathing. The disease may be due to the irritation of clothing, to want of cleanliness, or to irritating discharges from mucous surfaces, as in the eczema of the upper lip, thighs, or buttocks. It accompanies most of the parasitic skin diseases, particularly pediculosis, scabies, and ring-worm. It is probable that in many forms of eczema micro-organisms play an important part; even though they may not have been the primary factor in causing the disease, they may suffice to continue the inflammatory process.

The internal causes of eczema are chiefly associated with deficient elimination from the kidneys and bowels, and digestive disturbances. It often accompanies chronic constipation where there is intestinal torpor and the white stools of deficient biliary secretion; and it is seen where the urine is scanty and concentrated because children partake too largely of solid food. The latter is true both in the first and second years.

Eczema may be produced by any form of digestive disturbance, but it is especially frequent in the intestinal indigestion which results from overfeeding, or the too early or excessive use of farinaceous food, or from breast milk in which the percentage of fat is very high. From personal experience in the post-mortem room, I can confirm the observation of Bohn regarding the frequency with which fatty liver occurs in very fat infants. Enlargement of the liver may sometimes be made out during life. It is highly probable that the interference with the hepatic functions which accompanies these fatty changes has much to do with the production of eczema in such subjects. In children fed upon cow's milk the excessive fat may be the cause, or it may be due to excessive proteids. Of farinaceous articles, the two which are most often to be blamed are potato and oatmeal. Although eczematous patients usually appear to be well nourished, it is rare that some symptoms of indigestion are not present.

Eczema is often due to some form of reflex irritation. Such are the cases which accompany dentition, and the rare ones due to genital irrita-

tion. By many writers the eczema caused by disorders of the stomach or intestines is regarded as reflex. The stronger the predisposition, the more trivial is the reflex irritation which will induce an eruption.

Simple Chronic Eczema—Eczema Rubrum.—This is the most frequent form of eczema occurring in infants and young children, and is usually seen upon the face. It affects by preference the cheeks, forehead, and scalp, not infrequently the ears and neck, and may occur upon any part of the body. Upon the trunk and extremities the eruption is usually in patches, but in rare cases may cover nearly the entire body. The disease generally begins upon the cheeks with the formation of small red papules; later these coalesce, and there is a moist, red surface exuding serum or sero-pus. The secretion dries and forms thick, gummy crusts, which may be so hard as to form a mask for the face. From the scratching caused by the almost intolerable itching, the surface bleeds freely, and the dried blood gives to the crusts a dirty-brown colour and adds to the distressing appearance. The skin is often much swollen. After the removal of the crusts there is seen, in acute cases, a red, inflamed, granular surface, discharging pus or serum and bleeding readily. When the process is less active, there are redness, thickening, induration, and scaliness of the skin, and marked itching. In the same case these stages may alternate, exacerbations occurring whenever the exciting cause is particularly active. From the cheeks the disease spreads to the forehead, ears, and scalp, and here similar lesions are seen. Upon the trunk and extremities thick crusts rarely form, but the skin is red, thick, and scaly. The parts most often affected are the forearms, chest, elbows, knees, abdomen, and back; occasionally the eruption is general.

Swelling of the lymph nodes in the neighbourhood of the eruption is a constant feature of eczema of the face and scalp; these may reach the size of a chestnut or walnut, and occasionally they suppurate. Intense itching is a characteristic feature of all cases of eczema of the face or scalp. It causes restlessness and loss of sleep, and usually it is only in this way that the disease affects the general health of the patient; but in most cases the health remains good. With eczema of the occipital region of the scalp, pediculosis is usually associated.

Eczema of the face is very chronic, easily improved, but cured only with great difficulty. There is a strong tendency to relapses, brought on by neglect of local treatment or by any digestive disturbance.

The predisposition to eczema often ceases with the second year; those who have suffered from it almost constantly during infancy may be free from it during the remainder of childhood. This is in part to be explained by the loss of fat in consequence of more active exercise and a diet which is more largely nitrogenous. Where the disease continues through the third and fourth years, the associated infantile condition—obesity—is not infrequently present.

Seborrhœic Eczema.—This form of eczema has been brought into prominence by the writings of Unna, according to whom not only are all the cases usually classed as seborrhœa to be regarded as eczematous, but also many others classed as ordinary eczema. Instead of seborrhœic eczema being a form of disease in which the fat-producing glands are involved in the inflammatory process, Unna believes it to be parasitic and due to a certain "mulberry coccus" which he has described. Although his investigations have not yet been corroborated, there are many arguments in favour of the pathology which he has advanced for this disease. Elliot, who accepts Unna's views, defines seborrhœic eczema as follows: "An inflammatory disease of the skin, catarrhal in nature, due to micro-organisms—a parasitic dermatitis—characterized by its primary seat being upon the scalp, whence it tends to spread downward, involving by preference the middle portion of the face, the sternal and interscapular spaces, axilla, and inguinal regions, but may affect any part of the body."* The lesions upon the scalp may be of the nature of a dry seborrhœa with yellow greasy crusts, or like pityriasis. Upon the body, the eruption is scaly, with red macules or papules, or it may be accompanied by greasy crusts like those seen upon the scalp. The skin is not usually thickened and the lesions are not elevated. Itching in most cases is only moderate, and it may be absent; but in some of the most severe cases it is marked and accompanied by tingling. An extensive weeping surface is never seen. All the crusts are soft and contain fatty matter. The lesions are not deep, and the disease frequently shifts from one part of the body to another, often coming out very rapidly. In most cases the patches are rather sharply defined and have rounded borders.

Pustular Eczema of the Scalp.—This condition, often called "simple impetigo," is less frequently seen in infants than in children from two to five years old. There are usually present from half a dozen to fifty greenish-yellow crusts, matting the hair, usually discrete, but sometimes coalescing to form a mask over half the scalp. There is very little itching, in some cases none at all. The lymph glands are invariably enlarged. There is frequently continued auto-infection, and in this way the disease may be prolonged indefinitely. It is possible, too, that infection may spread to other children.

Intertrigo.—This term is rather indiscriminately applied to any eruption which develops upon two moist surfaces, which are in contact. It is often regarded as a form of eczema, although, as Elliot has well pointed out, there are seen several processes which are quite distinct from one another. The most frequent is a simple erythema; in other cases there is an eczema resulting from traumatism or the decomposition

* Morrow's System of Genito-Urinary Diseases, Syphilology, and Dermatology, vol. iii, D. Appleton & Co., 1895.

of secretions, or a seborrhœic inflammation. Intertrigo is seen in the folds of the groin, between the scrotum and the thighs, between the buttocks, about the anus, in the axillæ, in the neck, or behind the ears. Its essential causes are moisture, friction, want of cleanliness, and sometimes infection. The disease is generally seen in its worst form about the thighs, genitals, and buttocks; it sometimes covers the sacrum and extends down to the middle of the thighs. There is an intense uniform redness, and in some cases the epidermis is denuded over large areas, and the surface is moist. There is no thick crusting and little or no itching. Intertrigo is usually easy to control except in very poorly nourished or marantic children, among whom it is especially frequent.

Diagnosis of Eczema.—This is usually quite an easy matter. In the majority of cases, the disease affects the face or the scalp, and its appearances are typical. Eczema of the body or extremities may be confounded with scabies or syphilis, and occasionally with other forms of skin disease. Scabies resembles eczema in its intense itching and multiform lesions; but in the former, one may often find evidences of its presence in other members of the family; the parts most frequently affected are the flexures of the wrists, the elbows, the skin between the fingers, the margins of the axillæ, the lower part of the abdomen and back, and, in boys, the penis; and by careful examination with a lens some of the characteristic burrows are certain to be discovered.

Syphilis is likely to be confounded with papular eczema of the buttocks. The latter affects the parts near the anus, and the irritation may lead to the development of spots closely resembling mucous patches. The local appearances may at times be indistinguishable from syphilis, and the diagnosis is to be made only by the other symptoms present. In syphilis the characteristic eruption is seen usually upon the face, hands, legs, and sometimes the palms and soles; there is no itching and very little evidence of inflammation; the eruption is dark-coloured, and occurs as small circumscribed spots; there are usually present other symptoms, such as the coryza, the syphilitic cachexia, and enlargement of the spleen.

The diagnosis from pediculosis and ringworm of the scalp, rarely presents any difficulties.

Prognosis.—All cases of chronic eczema are tedious. There is only a slight tendency to spontaneous improvement, and very little to spontaneous recovery during infancy. In a given case, the prognosis depends upon the duration of the disease, its severity, and very much upon the co-operation of the mother or nurse. The results obtained depend not only upon the particular line of treatment adopted, but upon how well it is carried out. Usually it must be continued for several months. Eczema of the face is especially intractable when occurring in children suffering from chronic indigestion and constipation, for, unless these conditions can be controlled by diet and general management, local applications give but

temporary relief. Intertrigo is in most cases easily cured, unless the patient is suffering from marasmus.

Treatment.—It is never dangerous to cure an eczema, and always desirable to do so, in spite of the strong prejudice to the contrary, which still exists in the minds of the laity and in some members of the medical profession. To treat eczema successfully there is required a careful study of the exciting cause, for, although improvement often results from the use of local measures alone, yet in the great majority of cases this is only temporary. A permanent cure is brought about only by the removal of the cause. The physician must first endeavour to decide whether the eczema is due to some external or internal cause, or to both. External causes are for the most part easily discovered by carefully questioning the mother and observing how the child is cared for. Internal causes, as before stated, usually relate to the digestive tract or to functional disturbances of the kidneys.

Diet.—A thorough investigation into the food is necessary, not only as to its character, but as to quantity and preparation, the manner and frequency of feeding, etc. If the patient is a nursing infant, an examination of the nurse's milk is indispensable to intelligent treatment. If the child is very fat and well nourished, it is generally the case that the fat of the milk is too high and must be reduced according to the rules given elsewhere (page 164), the most important thing being to exclude from the nurse's diet malt liquors and alcohol in all forms, and reduce the amount of meat. In a smaller number of cases the trouble is with the proteids of the milk; there will then be other signs of indigestion, such as colic, the appearance of curds in the stools, etc. The amount of food should be reduced by lengthening the period between the nursings, and shortening the time which the child is allowed to remain at the breast at one nursing. Plain water, or better, some alkaline water, should be given freely between the nursings. In children fed upon cow's milk, the trouble is probably more often with the proteids than with the fat. The physician should try the effect, first of giving a milk which is low in proteids and moderately high in fat (e. g., formula iii or iv, page 175) afterwards, one in which both fat and proteids are low (e. g., formula xv or xvi, page 176). These and other changes are to be made in the manner described in the chapter on Infant Feeding (pages 175–182). During the latter part of the first and the entire second year, the usual error is that of overfeeding with in most cases an excessive use of solid food, especially farinaceous articles. The diet should then be much reduced, and the amount of farinaceous food restricted, potatoes and oatmeal being absolutely prohibited. The diet which suits most children best is one composed of milk, beef juice, broth, fruit, eggs, and a little red meat, with the addition in some cases of rice, wheat, or barley. In severe and obstinate cases, however, all cereals and even meat are best omitted during the active stage of the

disease. The form of indigestion which exists is to be managed according to the special indications in each case.

The diet of older children needs to be watched no less closely than that of infants. The general rules laid down elsewhere for feeding after the second year (pages 188–190) should be observed. The great majority of cases do best upon a diet which is largely fluid, and composed principally of milk or some of its substitutes,—kumyss or matzoon.

Elimination by the kidneys should be stimulated by the very free use of water, to which it is well to add—especially in cases with a gouty tendency —the citrate,* or acetate of potassium, from ten to twenty grains daily.

Attention to the condition of the bowels is of the greatest importance. To overcome the tendency to constipation is in many cases to cure the eczema. Suggestions under this head will be found in the chapter on Chronic Constipation (page 374). Special importance is to be attached to the occasional use of a purge of calomel, one half to one grain being given every third or fourth night. The best effects from this are seen in over-fed children. It has a favourable effect upon the kidneys as well as upon the bowels. The bowels must not only be opened, they must be kept freely open by the daily use, if necessary, of some of the milder laxatives, such as phosphate of sodium, rhubarb, or cascara. Sometimes nothing acts so well as castor oil, which may be given in from half a teaspoonful to teaspoonful doses every night for two or three weeks at a time. It should be administered in emulsion.

When the disease occurs in flabby, anæmic, or poorly-nourished children, iron and bitter tonics are required, and occasionally alcohol and cod-liver oil. In other words, the child's general condition should be treated just as if no eczema existed. Theoretically, arsenic is indicated when the disease is in a chronic stage with dry, scaly eruption, but its effect is often disappointing in infancy. It is in no sense a specific remedy.

The *general management* of cases is important. The skin must be carefully protected by an ointment whenever the child is in the open air; if the weather is very cold, or there are high winds, children with active eczema should not go out, but take the fresh air indoors. Never should an eczematous surface be washed with plain water, and much less with castile soap and water, so frequently employed by the ignorant. Where washing is necessary, it may be done with bran water, milk and water, or starch and water, to which borax (a teaspoonful to the quart) may be added. The clothing should not be so excessive as to keep the child constantly in a perspiration. Napkins should not be washed in strong soda solutions, nor, in case of eczema of the buttocks, should they be used a second time after being simply dried.

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* While the citrate can not be depended upon as a diuretic, unless dispensed from a newly-opened bottle, it is generally to be preferred, as being more easily administered.

In eczema of the face it is absolutely necessary to prevent the child from scratching the parts. The use of a mask is not always sufficient, nor the wearing of mittens; nor is the local application of anti-pruritic lotions or ointments invariably successful. In severe cases mechanical restraint is absolutely indispensable. The most satisfactory method is to surround the arms at the elbows by pastéboard splints, and hold them in place by bandages. This allows free use of the hands, but makes it absolutely impossible for the child to reach the face.

Local treatment.—Local treatment is always necessary, for not only are the causes sometimes entirely external, but the condition may persist after the original internal cause has been removed. There are several indications to be met by local treatment at different stages in the disease: (1) To remove crusts and other inflammatory products; (2) to allay congestion and acute inflammation; (3) to relieve itching; (4) to protect the delicate new skin which is forming; (5) to prevent infection; (6) to stimulate the skin in the chronic stages of the disease.

Preparatory to the use of any application, the scales, crusts, and other products of inflammation must be softened and removed in order that the diseased surface may be reached. In most cases it is sufficient to soften the crusts by the use of olive oil for twelve or twenty-four hours, and then remove them by soap and warm water. If the crusts are very hard and thick, they can be softened by a poultice. During the stage of acute inflammation only sedative applications should be used. One of the best of these is a lotion of zinc and calamine:

℞ Pulv. calaminæ preparatæ..... ʒ ij
Zinci oxidi..... ʒ ss.
Glycerinæ..... ʒ j
Liquor calcis..... ʒ ij
Aquæ rosæ..... ʒ viij.

A piece of muslin should be dipped in this solution, and applied to the affected part, being kept in place by a bandage. If there is much itching, one per cent of carbolic acid may be added.

Another plan of treatment, where there is much secretion, is to keep the surface covered with equal parts of boric acid and starch or the stearate of zinc. An application which is often successful in allaying the intense burning and itching is black wash. This is applied with absorbent cotton for ten or fifteen minutes several times a day, and allowed to dry on, after which a protective ointment is used. If the black wash in full strength is painful, it may be diluted with water. Ichthyol may be used in the same way, but only in dilute solution—i. e., from one half to one per cent.

As a simple protective ointment to follow any of the above, one containing starch, zinc oxide, or bismuth, either alone or in combination, may be used. An excellent formula is Lassar's paste:

℞ Acidi salicylici..... gr. x
Zinci oxidi..... ʒ ij
Amyli..... ʒ ij
Vaseline..... ℥ j

Later, when the inflammation is less acute and the itching severe, nothing is so generally useful as a combination of tar and zinc, as in the following:

℞ Ungt. picis liquidæ..... ʒ iij
Zinci oxidi..... ʒ iss.
Ungt. aquæ rosæ..... ʒ vi

For more chronic cases, the amount of tar may be increased. All ointments used should be spread upon muslin, and kept in close contact with the inflamed part by means of a bandage or mask. Little or nothing is accomplished by simply rubbing the ointment upon the affected part. Where it is difficult to keep a mask applied, or in situations where it is impossible to use the ointment, Pick's paste may be tried:

℞ Pulv. tragacanthæ..... ʒ j
Glycerinæ..... ʒ iss.
Aquæ rosæ..... ℥ iv

To this may be added zinc oxide gr. xl and carbolic acid gr. v, or tar ♏ x. A similar basis for ointments, made from gum tragacanth has been suggested by Elliot and is known as bassorin paste. It may be combined with tar, zinc, salicylic acid, or resorcin.

The methods of treatment above mentioned are especially applicable to eczema of the face and scalp. For pustular eczema of the scalp the best application is the white-precipitate ointment, which should be combined with three or four parts of vaseline. This is excellent also for small eczematous patches upon the body, but it is not to be used over a large surface.

In intertrigo, the treatment should have reference to the pathological condition which is present. Cases of simple erythema usually yield promptly to cleanliness and the free use of absorbent antiseptic powders, such as boric acid and starch in equal parts. If there is an acute dermatitis, the calamine and zinc lotion may be used, and later some protecting ointment. When infection has been added, lotions of resorcin or ichthyol, one half or one per cent strength, should first be applied, and the skin then covered with the powder mentioned; both are to be repeated as often as the parts are wet by urine or soiled by fæces. It is important in all cases that the diseased surfaces should be kept separated, which is best done by starch and absorbent cotton. All napkins should be immediately removed when soiled. Other useful applications are Lassar's paste and Pick's paste combined with zinc oxide.

In cases of chronic eczema, where the skin remains thickened, red,

scaly, and itching, stimulating applications are to be used, such as the tincture of green soap or stronger preparations of tar than those mentioned. They should be applied every three or four days.

In the seborrhœic form of eczema, whether affecting the face, scalp, or body, nothing is so generally useful as resorcin:

℞ Resorcin..... gr. x
Ungt. aquæ rosæ..... ʒ j

This may also be advantageously combined with bassorin paste.

FURUNCULOSIS.

A furuncle, or boil, is a circumscribed inflammation of the subcutaneous cellular tissue, beginning in a hair follicle, sweat gland, or sebaceous gland, and usually ending in suppuration. When severe, it may result in necrosis of the follicle, which forms the "core," or the necrotic process may extend to the surrounding tissues for a variable distance. The ordinary boil need not be described, as it presents nothing peculiar in early life. The condition, however, which is characteristic of young children is the formation of small ones in great numbers. It is to this more especially that the term *furunculosis* is applied. The principal seat of these small abscesses is, in nearly all cases, the scalp, face, and shoulders, although they may be found upon any part of the body. They are sometimes numbered by hundreds, and appear in crops for a period of several months. In size, they usually vary from a pea to an almond, and they rarely contain a core. Infants are much more often the subjects of this disease than are those who have passed the second year. In the great majority of cases the condition is not serious, yet, occurring, as it often does, in infants who are already suffering from extreme malnutrition or marasmus, whose tissues possess but little resistance, the process may develop into a gangrenous dermatitis, which may prove fatal.

Furunculosis is seen in children who are in other respects apparently healthy, even robust; but the majority are in a more or less debilitated condition, and often are the subjects of digestive disturbances. The disease is quite frequent in syphilitic infants; but these simple abscesses are to be sharply distinguished from those which result from the breaking down of gummata of the skin. Want of cleanliness of the skin is a factor of some importance in producing the disease. Furunculosis may be associated with eczema. The exciting cause in all cases, as shown by the recent investigations of Escherich and others, is the entrance of pyogenic germs, usually the staphylococcus aureus, into the follicles of the skin.

Treatment.—The internal treatment is to be directed toward any disturbance of digestion or general nutrition which is present. General tonics are indicated in most cases, particularly iron, arsenic, and the compound syrup of the hypophosphites. But little reliance can be placed

upon internal remedies, such as sulphide of calcium, for the purpose of arresting this disease. Local treatment should have for its first object thorough cleanliness of the skin. This is best secured by frequently bathing the parts affected with a saturated solution of boric acid. Single furuncles may often be aborted by the frequent application of spirits of camphor, or a few applications of tincture of iodine, or by touching them with pure carbolic acid. The last mentioned, although efficient, can hardly be intrusted to the hands of a mother or nurse. A remedy which has been used with considerable success is a plaster of salicylic acid. In my experience the best plan of treating the multiple small furuncles, is to delay incision until they have pointed, then to incise freely and empty the follicle completely by compression. It is then washed out thoroughly with a solution of bichloride (1 to 2,000), and small pledget of absorbent cotton applied till the bleeding has ceased. After this the part should be covered with simple collodion or that in which iodoform has been dissolved. Where the abscesses are of large size and upon the scalp, it is wise to make compression by applying a snug bandage for a day. It is very exceptional for abscesses so treated to refill. When the suppuration is more diffuse and there is necrosis of the cellular tissue, ichthyol, either in the form of an ointment or lotion (one to five per cent strength), is one of the best applications. Early and free incisions must be practised in all such cases.

GANGRENOUS DERMATITIS.

This is not a frequent disease, and is seen almost exclusively in infancy. It may be primary or it may follow other diseases, and hence has been described under many different names—viz., *varicella gangrenosa*, *ecthyma gangrenosa*, *pemphigus gangrenosa*, etc.

The lesion consists in small, discrete areas of inflammation of the skin, ending in necrosis. In the primary cases there is usually first seen a vesicle, about as large as a pea, with a dusky areola; it increases in size and becomes a pustule. Crusts form which are quite adherent, and on removing them a loss of tissue is seen. The ulcers usually have sharp but not undermined edges, often presenting a "punched-out" appearance. By the coalescence of several small ones, ulcers an inch or more in diameter are sometimes formed.

The primary form of gangrenous dermatitis occurs in wretched, poorly-nourished infants, and, according to Elliot, is most often seen upon the buttocks. In this location it may be mistaken for syphilis. The secondary form is more common, and usually follows varicella, less frequently vaccinia, measles, or pemphigus. My own experience with this disease has been confined to cases following varicella. In such, the lesion is usually seen upon the upper half of the body, especially upon the neck and chest. It follows the ordinary lesions of varicella and continues usually, in spite

of treatment, from one to four weeks, in most cases ending fatally. The disease always occurs in infants of poor vitality, often in those suffering from marasmus, and is seldom seen outside of institutions. It may be accompanied by fever, and other severe constitutional symptoms.

For the production of the disease, two factors are necessary: first, the constitutional condition referred to; and, secondly, the entrance of pyogenic germs, usually the streptococcus pyogenes.

Treatment.—Every means possible should be employed to build up the general health of the infant by tonics, fresh air, careful feeding, etc. Locally, strict cleanliness and antiseptic applications are necessary. The best application is a solution of bichloride (1 to 5,000), or an ointment of ichthyol or iodoform.

IMPETIGO CONTAGIOSA.

Impetigo contagiosa is a disease characterized by the formation of discrete vesiculo-pustules, occurring most frequently upon the hands and face. Cases are usually seen in groups affecting several children in one family or institution. It may be communicated from one person to another, and spread by auto-inoculation from one part of the body to another.

One rarely has an opportunity to see the disease until vesicles have formed. These are usually from one fourth to one half an inch in diameter, and are flaccid, never distended. Later, their contents become slightly yellowish; then they rupture and dry, forming thick yellow crusts, which have the appearance of being "stuck on," the surrounding skin being quite healthy. After the crusts fall off, a small red patch remains, which slowly fades. The true skin is not involved, except in poorly-nourished, cachectic subjects, as a result of continued local irritation, like scratching. Under such conditions ulceration may occur. Instead of the small vesiculo-pustules described, bullæ from one to two inches in diameter may form, filled first with serum, afterward with sero-pus. Very little inflammation is seen about these patches, and in most cases the intervening skin is normal.

The favourite seat of the eruption is the face, especially about the chin, next the hands, the neck, the feet and legs, the forearms, and the scalp; it is rarely seen upon the abdomen, and never upon the back. There may be only half a dozen vesiculo-pustules, or from thirty to forty may be present. The smaller ones sometimes coalesce and form others of considerable size. Itching is never a prominent symptom, and in most cases it is absent altogether.

The usual duration of impetigo contagiosa is two or three weeks; it, however, runs no regular course, and by continued auto-inoculation may last much longer than this.

The disease is undoubtedly due to some form of local bacterial infection,

but the exact nature is not yet determined. It may occur in any child, but is usually seen in one who is cachectic and poorly nourished.

The diagnosis is not often difficult, and is made by the following features—viz., the occurrence of several cases together, the isolated vesiculopustules situated upon the face and hands, the slight itching, and the prompt cure by local measures only. The bullous form, however, is sometimes confounded with pemphigus, and there are cases in which the differential diagnosis may be quite difficult.

Treatment.—This is simple and usually very effective. The crusts are to be softened and removed by thoroughly washing the part with soap and water or a bichloride solution, after which the white precipitate ointment, combined with three parts of vaseline, should be applied.

URTICARIA.

Urticaria is a frequent disease in early life, and presents some features, particularly in infants and young children, which are quite different from those seen in adults. This is due to the fact that papules and vesicles, and occasionally pustules, are associated with the wheals. As the wheals quickly subside, it frequently happens that the other lesions mentioned are the only ones present. This fact has given rise to considerable confusion in names, and the urticaria of infancy has been called *lichen urticatus*, *urticaria papulosa*, *strophulus*, etc. It is now pretty generally agreed that the clinical picture, which is a familiar one, belongs to a single disease, and that this is urticaria.

The initial lesion is the wheal, but on account of the extreme susceptibility of the skin in young children, the process is more intense than in older patients, so that it may result in the formation of an inflammatory papule or a vesicle. In a few hours the wheals may subside, and only the papules or vesicles remain, and without a good history the disease may be a very obscure one. The papules and vesicles occur with greatest frequency upon the hands and feet, particularly the palms and soles. The more severe form of the disease in poorly nourished children is sometimes accompanied by a pustular eruption, and there may even be deep ulceration (ecthyma). The usual appearance of the eruption is a number of small inflamed red papules whose tops are covered with scabs, the result of scratching. The eruption may be limited to the extremities or it may be general. It is as a rule more severe in regions accessible to scratching.

There is usually severe itching, which leads to loss of sleep, and often in this way the disease affects the general health of the child. The urticaria of older children does not differ essentially from the same disease in adults.

The character of the eruption in urticaria and even its distribution strongly suggest scabies; and unless one has had an opportunity to witness the development of the lesions, a differential diagnosis may be very difficult,

as almost every lesion, except the wheal, may be identical in both diseases. Other cases may resemble varicella.

Urticaria in early life is most frequently the result of some disturbance in the digestive tract. Almost any sort of derangement may produce it, the exciting cause varying with the patient. Exceptionally, it may result from other forms of irritation, such as dentition or intestinal worms, and it has been ascribed to malarial poisoning.

Treatment.—The milder forms of urticaria usually respond quickly to treatment; but when it is severe and has existed for several weeks, it is one of the most troublesome and intractable skin diseases of childhood. The treatment is to be directed primarily toward the condition of the digestive organs. Children should be put upon a milk diet, and even milk may need to be partially peptonized. The bowels should be kept freely open by calomel, a nightly dose of castor oil, or a morning dose of magnesia. If the urine is excessively acid and scanty, alkaline diuretics should be given. The drugs most useful for the indigestion with which urticaria is associated are salicylate of soda and nitro-muriatic acid, each of which is to be given after meals.

All local causes of irritation, such as rough flannel underclothing, should be removed. The sleep may be so much disturbed as to require the use of trional or bromide and chloral. The two remedies which are of most value for the disease itself are antipyrine and atropine; they may be used separately or in combination, and should be administered in moderately large doses.

The local irritation and itching may be relieved by a lotion of menthol (gr. ij, water ℥ j), by a very dilute solution of the subacetate of lead or carbolic acid, or by a mixture of vinegar, or the fluid extract of hamamelis, and water. Where pustules are present, the white-precipitate ointment may be used, combined with four parts of vaseline; in the papular and vesicular forms, an ointment of ichthyol or naphthol, one per cent strength. In many cases the improvement in the general health by the use of tonics, change of air, etc., will accomplish more than any measures directed especially to the relief of the urticaria.

SCABIES.

Scabies is a contagious disease due to the burrowing into the skin of the female acarus, with secondary lesions which result from scratching. This disease is not a common one in New York, even among dispensary patients, while among the better classes it is extremely rare.

The burrowing of the acarus is usually where the skin is thinnest—viz., between the fingers, on the flexor surfaces of the wrists, the axillæ, and, in males, the genitals. It is not seen upon the face, except in infancy, when it may be infected by contact with the breasts of the mother.

The lesion excited by the acarus is usually a papule or a vesicle, sometimes a pustule. In some cases no evidences of inflammation are present, but in infants and young children they may be marked,—pustular eruptions being frequent and often extensive, especially upon the hands and feet. The characteristic burrow is from one fourth to one half inch in length, and appears as a fine brown or black line, at the end of which the acarus may be discovered as a small white speck. The burrows are often difficult to find in infants. They are generally to be seen along the inner border of the hand and between the fingers. The intensity of the inflammatory lesions varies greatly in different cases; in some they are very few, while in others, particularly in delicate, cachectic, and neglected children, they are sometimes very severe, so that the skin of the affected part is nearly covered with pustules. This is especially true of the hands, where a pustular eruption should always suggest scabies. The lesions which result from scratching may be found on any accessible portion of the body. There are usually at first linear, bloody marks, but after a time these may not be visible, and there may be only a traumatic eczema. In little children urticaria is often associated.

The diagnosis of scabies is usually quite easy, as several children in a family are likely to be affected, particularly if they occupy the same bed. The diagnostic features of the eruption are the presence of papules, vesicles, or pustules, especially upon the hands, wrists, and genitals. A careful examination with a lens will usually disclose some of the characteristic burrows, or even the acarus. In infancy, scabies may be easily confounded with the vesicular form of urticaria, unless the development of the lesions has been observed.

Scabies may always be cured, provided sufficient precautions are taken to prevent re-infection. This necessitates boiling or baking, not only the patient's clothes, but all the bedding as well.

Treatment.—This should always be begun by a hot bath, in order to soften the epithelial scales about the burrows. The body should be thoroughly scrubbed with soap and water, preferably with a nail-brush, the bath being continued for at least half an hour. It is well to do this at night. After the bath, the body is anointed with the parasiticide, which should be thoroughly rubbed into the skin, clean clothing applied, and the child put into a perfectly clean bed. In the morning the ointment may be washed off, but none of the clothing previously worn should be put on. This treatment is to be repeated on two or three successive nights, and if thoroughly done it will effect a cure. The ordinary sulphur ointment is too irritating for use in little children, and one of the following may be substituted: naphthol, 15 parts; creta preparata, 10 parts; vaseline, 100 parts (Kaposi); or, precipitated sulphur, 1 part; balsam of Peru, 1 part; vaseline, 8 parts; or the simple balsam of Peru may be applied without dilution. After the use of the parasiticide there is generally

required for a few days, some soothing application like those mentioned in the chapter upon Eczema.

TINEA TONSURANS—RING-WORM OF THE SCALP.

Ringworm of the scalp is a very frequent disease in institutions for children, often occurring as an epidemic. According to Crocker, the primary lesion consists in a red papule surrounding a hair, which soon increases to a small circular patch; this spreads at its outer margin, gradually increasing in size until it is from one to two inches in diameter, but rarely larger than this. Sometimes several of the patches coalesce. These affected areas always have rounded borders, and are sharply outlined. Here the hairs are very brittle, and often broken off close to the scalp, so that it may appear to be bald. Where they have not fallen off, the hairs have lost their lustre. The stumps of the broken hairs point in all directions.

The fungus which produces the disease is the *trichophyton tonsurans*. It penetrates the shaft of the hair, both the spores and the mycelium being seen under the microscope. The spores are present in great numbers in the hair, but the mycelium is most abundant in the scales. The amount of inflammation found in the diseased areas varies much in the different cases. There may be only a scaliness of the scalp, or a formation of pustules in the hair follicles, the hairs loosening and falling out in consequence. In young infants where the hair is scanty and thin, the disease resembles tinea circinata—i. e., it is superficial, and the hair follicles are often not involved. Children of all ages are liable to tinea tonsurans. It flourishes particularly in those who are dirty and poorly cared for.

The diagnostic feature of the disease is the presence of scaly patches, with loss of hair. The patches are usually circular, and by examination with a lens the stumps of broken hairs are seen all over the diseased area. By a microscopical examination the fungus is discovered. In typical cases the diagnosis is easy if the process is at all advanced, but there are many atypical forms and many mild cases where the recognition of the disease is difficult. The symptoms are often masked by the inflammatory conditions present. The disease may be confounded with seborrhœa; but in the latter the lesion is diffuse, never sharply defined; there is general thinning of hair over the scalp, and never the stumpy, broken hairs. Psoriasis has points of resemblance, but it is usually found on other parts of the body, especially the knees and elbows, and upon the scalp the patches are more numerous and smaller. In eczema the loss of hair in circumscribed patches is never seen, nor are the broken stumps.

Tinea tonsurans is always curable, provided the patient can be kept under close surveillance, and treatment thoroughly carried out. There is no tendency to spontaneous recovery. In a recent case, treatment must

usually be continued for one or two months, and in chronic cases, from six months to one year, with the closest watchfulness.

Treatment.—The great difficulty in treatment is to get the parasiticide deeply enough into the scalp to reach the fungus, since this is often at the very bottom of the hair follicles. As a first step, the hair should be cut short all over the patch and for at least an inch beyond it; this is necessary in order to get at the diseased part and to detect new foci of infection early—if possible before the fungus has extended deeply into the follicles. The parasiticide should be applied not only upon but around the patch, and the entire scalp should be washed thoroughly two or three times a week. To prevent the disease spreading, all the scales are to be kept softened by the use of carbolic soap. The hair should not be brushed, as this tends to scatter the spores and spread the disease. All patients while under treatment, should wear a cap of muslin or oiled-silk, or one lined with paper, in order to prevent infecting others. In institutions, affected children should invariably be isolated.

To destroy the fungus almost every germicide on the list has been advocated at one time or another, which proves that the disease is a very obstinate one, and that no one application is invariably successful. Those which have the sanction of the widest use are the tincture of iodine, the bichloride, white precipitate, and oleate of mercury, kerosene, creosote, and croton oil. As a vehicle for ointments, lanoline is greatly to be preferred to vaseline or lard; according to Crocker, the addition of three parts of lanoline to one part of olive oil is much better than lanoline alone. Most of the germicides mentioned are used in the strength of one to five per cent, according to the age of the child and the irritability of the scalp. In an epidemic of ring-worm in the New York Infant Asylum the following combination of bichloride and kerosene proved extremely satisfactory: ten grains of the bichloride were dissolved in alcohol, and to this were added two and a half ounces each of olive oil and kerosene. This was applied every day, being thoroughly rubbed into the diseased patches, and the whole scalp saturated with it. Considerable irritation usually resulted, and every few days the parasiticide was omitted and some simple emollient applied until the irritation had in a measure subsided. In some of the cases, the tincture of iodine was alternated with the bichloride and kerosene. Twenty-six cases were treated after this plan and all cured, the average duration of treatment being eight and a half weeks.*

Epilation is necessary in many cases as an accessory to the application of germicides, particularly in older children.

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* A full report of these cases was made by C. G. Kerley, M. D., in the New York Medical Journal, October 10, 1891.

CHAPTER VI.

ACUTE OTITIS.

Otitis is a frequent affection during infancy and early childhood, attacks usually occurring in the cold season. Of all the inflammatory conditions which may be met with in early life, there is perhaps none which more frequently gives rise to obscure febrile symptoms than this.

Etiology.—Acute otitis, as a rule, is a secondary disease, and is generally preceded by some infectious process in the rhino-pharynx. The usual avenue of infection is through the Eustachian tube. The catarrh of the pharynx may be a simple one, the ordinary head-cold, or it may occur as a complication of the acute infectious diseases. Downie gives the following statistics of 501 cases of tympanic involvement treated in the Children's Hospital in Glasgow:

Originated during measles	131	cases,	or	26·1	per cent.
" " scarlet fever	63	"	"	12·6	" "
" " whooping-cough	15	"	"	3·0	" "
" " mumps	3	"	"	0·6	" "
" " simple catarrh	147	"	"	29·4	" "
" " dentition	101	"	"	20·0	" "
Syphilitic	8	"	"	1·6	" "
Doubtful	33	"	"	6·7	" "
	501			100·0	

The most common condition preceding severe otitis is scarlet fever, and next in the order of their frequency, epidemic influenza, simple acute pharyngitis or tonsillitis, measles, diphtheria, and typhoid fever. Otitis when following simple inflammations of the throat is usually much less severe than when it complicates scarlet fever or diphtheria. Cold and exposure frequently play the rôle of exciting causes. In a few cases the disease is the result of traumatism, such as a blow or traction upon the external ear, or the entrance of fluids through the Eustachian tube from the nasal douche. It sometimes results as an extension of inflammation from meningitis, especially the cerebro-spinal form. When seen as a complication of scarlet fever, measles, or diphtheria, the symptoms are usually manifested from the sixth to the tenth day of the disease.

Lesions.—The ordinary course of events in the pathological process is, first, acute hyperæmia and swelling of the mucous membrane of the rhino-pharynx, which extends into the Eustachian tube, causing obstruction more or less complete. The inflammatory process may be limited to the tube, or it may extend to the mucous membrane lining the middle ear.

There are two varieties of acute inflammation of the middle ear: (1)

The catarrhal form, which usually accompanies simple catarrh of the rhino-pharynx or complicates measles. This is an inflammation of the mucous membrane merely, and its products are serum and mucus or muco-pus. It is not usually accompanied by great pain or followed by serious consequences. It is generally confined to the lower part of the tympanic cavity, and is the form most frequently seen in infants. (2) The phlegmonous form, which affects older children principally. This is a much more serious inflammation, and is often excited by the infectious catarrh of scarlet fever, diphtheria, or epidemic influenza. In this variety micro-organisms find their way into the middle ear in great numbers, and set up an inflammation of a more or less virulent type, which may involve not only the mucous membrane lining the tympanum, but also the cellular tissue in the upper part of the tympanic cavity.

The catarrhal form of inflammation frequently subsides in a few days with proper treatment, the only result being a slight deafness, which is temporary. The phlegmonous form causes a stoppage of the Eustachian tube, rupture or sloughing of the tympanic membrane and discharge of the products of inflammation, or rarely pus finds an outlet by burrowing along the cartilages. The inflammatory process may extend to the bones, causing necrosis of the ossicles or the bony walls of the tympanum. The remote results are periostitis and necrosis of the petrous bone, pachymeningitis, infectious thrombosis of the lateral sinus, general purulent meningitis, and cerebral abscess. These will be considered under Complications.

Symptoms.—These are usually few in number, but present great variability as regards their combinations and intensity. The two most constant symptoms are pain and fever. In a typical case in an infant, there is generally at the beginning some discharge from the nose, slight congestion of the pharynx and tonsils, and a temperature of 100° to 102° F. There is nothing characteristic about this catarrh. After two or three days the objective symptoms subside, but the infant continues to be restless, worries much of the time, wakes frequently at night with a start, nurses poorly, and if the thermometer is used, it is found that the temperature remains elevated, usually from 99° to 101° F. The infant seems decidedly ill, and yet no very definite symptoms are present. Sometimes there is marked tenderness about the ear, and the child refuses to lie upon the affected side, or shows signs of pain when the ear is touched. After a week or ten days a discharge is found in the auditory canal, and usually there follows a rapid subsidence of the constitutional symptoms. In some cases there is seen only a high temperature, ranging from 101° to 104° F., which persists for several days without outward evidences of pain or other signs of inflammation, the discharge being the first symptom which leads the physician to suspect disease of the ear. In other cases there are marked dulness, apathy, anorexia, and sometimes nausea and vomiting,

but for several days no evidence of pain; the temperature may be but little elevated. Thus, in most of the attacks seen in infancy, pain is not very marked, and it is this which so often leads to the great obscurity of the symptoms.

In older children the symptoms are more characteristic. Pain is usually sharp and severe, and is complained of early in the attack. The temperature is nearly always elevated two or three degrees, and occasionally it is 103° or 104° F., with severe headache, extreme restlessness, and even delirium or convulsions, so that meningitis may be suspected.

The inflammation does not necessarily go on to suppuration and rupture. There are even more frequently seen, accompanying ordinary head-colds or mild attacks of influenza, cases in which the pain is quite severe for twenty-four or thirty-six hours, and accompanied even by a moderate elevation of temperature, and yet which rapidly subside without further symptoms. In these cases the pain is too constant and too prolonged to be an attack of neuralgia. They are simply cases of a mild form of inflammation.

In infants suffering from malnutrition or marasmus, otitis not infrequently comes on without any objective symptoms, the first thing noticed being the discharge. This association of otitis with marasmus is to be attributed to the frequency of swelling of the adenoid tissue in the pharyngeal vault, upon which the catarrhal process depends.

Of the individual symptoms, fever is the most constant, and is present in all except the cases of marasmus just mentioned. The usual range of temperature is from 100° to 102° F.; exceptionally it may be from 103° to 105° F. The course of the temperature is irregular and remittent. After spontaneous rupture or incision of the drum membrane the temperature usually falls, but often not immediately; occasionally it continues almost as high as before for twenty-four hours. Pain is more marked in older children than in infants: first, because in the latter the drum membrane is not so firm, yields more readily, and ruptures earlier; and, secondly, because the inflammation is usually of the catarrhal and not the phlegmonous type. Tenderness is sometimes elicited by pressure just in front of the external auditory meatus; there may be increased sensitiveness of all parts of the ear and even of the whole side of the head. Children not infrequently complain of noises in the ear. One little girl with obscure symptoms and high temperature, first called attention to her ear by the remark, that she "heard pussy in the room." A sense of discomfort resembling that which is felt when the ears are stopped, frequently leads children to pick at them. Cerebral symptoms are infrequent, and occur chiefly in cases not receiving proper early treatment; they are practically limited to the phlegmonous form of inflammation, and they may indicate meningeal congestion, less frequently localized meningitis or thrombosis.

The local appearances in the early stage—provided a view of the tympanic membrane can be obtained—are acute redness and congestion; later there is distinct bulging of the membrane. If perforation has taken place, its site may or may not be visible, but, according to Pomeroy, its existence may always be assumed, if there is pulsation of the membrane, if bubbles of air are seen deep in the canal, if the perforation whistle occurs upon blowing the nose or inflating the ear, and, finally, if much mucus or pus is present, as inflammation of the external canal almost never causes much discharge. A discharge is not present until perforation has taken place. The pus in rare cases may burrow along the cartilages and open externally behind or at the side of the ear. The nature of the discharge depends upon the variety of the disease; in the catarrhal form it is at first sero-mucus, whitish in colour, rather thick, quite profuse, and usually continues when once established; later it is usually purulent. In the phlegmonous form it is always purulent, generally less abundant, and liable to a sudden arrest with an exacerbation of the constitutional symptoms. As the case improves the discharge diminishes in quantity and gradually assumes a serous character.

Diagnosis.—In typical cases characterized by pain and temperature, this is usually easy, particularly in older children. Otitis in infancy is frequently obscure, sometimes because the patient is too young to direct attention to the seat of pain, but more often because the pain is slight or entirely absent. The temperature is almost invariably elevated, and the usual problem presented to the physician is to discover a cause for this fever. In the absence of definite otoscopic signs, one must rely upon the presence of faucial congestion, a history of a previous acute catarrh, restlessness at night, and the absence of other signs in the throat, lungs, or digestive tract, which might explain the fever. Local tenderness, deafness, or noises in the ears are of much significance when present, but they are very often wanting. Otitis is so common a cause of high temperature in infants during the cold season, that one should always be on the lookout for it. In older children a neuralgia arising from a carious tooth may give rise to a pain resembling that of otitis.

Prognosis.—The ordinary catarrhal form of acute otitis is not often followed by serious consequences, unless there are repeated attacks. The phlegmonous form, especially when it complicates scarlet fever, is always serious, and in the majority of cases it is followed by some degree of impairment of the sense of hearing.

Complications and Sequelæ.*—Remote consequences are most likely to be seen in cases following scarlet fever, probably because of their severity, particularly when early treatment has been neglected. In many cases the symptoms are obscure because the discharge from the ears has been

* See Pitt's Gulstonian Lectures, 1890.

slight or wanting. It is to be remembered in this connection that the Eustachian tube, middle ear, and antrum, in young children are relatively large, and hence easily infected, while the mastoid cells are imperfectly developed. These anatomical conditions explain the greater frequency of extension of the disease to the petrous bone and the brain, and, as compared with adults, the infrequency of mastoid complications.

Meningitis.—This may be a cause of death in young children. There may be a localized pachymeningitis with the formation of pus, or a general purulent meningitis. It may be secondary to other lesions, such as thrombosis of the lateral sinus, or the rupture of a cerebral abscess, but is usually due to the passage of pus through the roof of the tympanum, or along the internal auditory meatus. Meningitis is more frequent as a complication of old cases, but may develop soon after the early acute symptoms. Its onset is usually sudden, and its duration rarely more than a week.

Cerebral abscess.—This is due to a direct extension of the infectious process from the bone, vein, or dura mater. In about two thirds of the cases the abscess is in the temporo-sphenoidal lobe. The next most frequent seat is the lateral lobe of the cerebellum. Körner states that disease of the mastoid and middle ear leads to cerebral abscess, and disease of the labyrinth to cerebellar abscess. Abscesses may be complicated by thrombosis or by meningitis. They are often latent until just before death, which more frequently occurs from the development of purulent meningitis than from any other cause. They are rare except in cases of long standing.

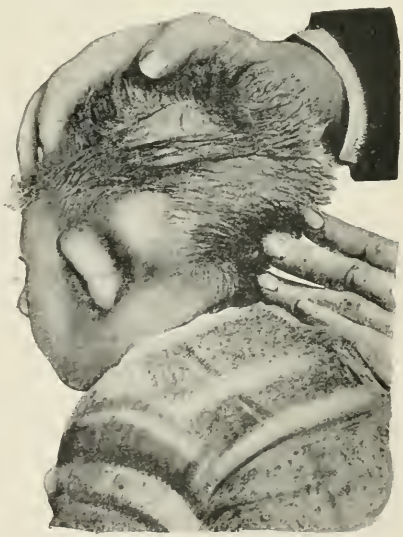

Fig. 153.—Mastoid abscess following acute otitis.

Thrombosis of the lateral sinus occurs as a condition antecedent to meningitis or abscess, or without them. It usually develops suddenly, with recurring chills and a high temperature, which is subject to sudden and wide fluctuations.

Mastoid disease, as previously stated, is not so frequent a complication of otitis in children as in adults, one reason being that the mastoid process contains but a single cavity, the antrum, whose walls are so thin that spontaneous rupture externally readily occurs, while the mastoid cells are very imperfectly developed until after puberty. Mastoid disease may accompany either acute or chronic otitis. There are local

pain and tenderness and a very characteristic swelling, which causes the ear to stand out from the head (Fig. 153). Usually the process ends in suppuration, with the symptoms of external abscess, but resolution sometimes occurs. This may often be promoted by the early application of cold either in the form of an ice bag or a coil.

The labyrinth is less frequently involved, although cases are recorded by Pye, Phillips, and others, in which the necrosis and discharge of the entire labyrinth has occurred after scarlet fever. In most of these cases the deafness was complete, and in several vertigo was present.

Facial paralysis rarely occurs in the acute cases, but accompanies a considerable proportion of the chronic ones. It is due to an extension of the inflammatory process from the bone to the seventh nerve, where it passes through the canal. The symptoms are those of ordinary peripheral facial palsy.

Treatment.—If the case is seen in the early stage, the inflammation may not infrequently be cut short by local blood-letting and the use of heat. Blood-letting is not to be advised in the case of young infants, but may be used in children over two years old. It should be urged in spite of its obvious disadvantages, as nothing is so efficient. Either leeches or wet cups may be employed. They should be applied just in front of and close to the tragus. Dry heat is to be preferred to moist heat, both as a means of arresting inflammation and of relieving pain. It may be applied by means of a bag of hot water, salt, or bran, or by a hot brick or soapstone. These should be placed beneath a thin pillow, upon which the child's head rests. If the child will not lie upon his hot pillow, a small bag of salt or hot water may be bound over the ear, which has been first covered by cotton. Perhaps the best of all is Dench's device of filling the tip of the finger of a kid glove with salt, and inserting this into the canal after heating; cotton should be applied over it. Hot poultices may be used for a short time, being changed frequently, but prolonged or continuous poulticing encourages suppuration and should never be allowed. On no account should oil, or oil and laudanum, be dropped into the ear, as is so often done in domestic practice. If the child is not comfortable in the course of a couple of hours after the blood-letting or dry heat, an opiate should be given. This not only relieves suffering, but has a favourable influence upon the inflammation.

A return of the severe pain on the following day, or its continuance in spite of ordinary measures, with a steadily high temperature, are indications for operative interference. If to the above, cerebral symptoms are added, operation is imperative. An early incision of the drum membrane is usually followed by a discharge of blood only; but tension is relieved and with it the pain disappears, and the inflammation often quickly subsides without the formation of pus. Much suffering is thereby avoided, and, as the wound heals quickly, much less damage is done than by allow-

ing the disease to go on to a spontaneous rupture. Later operation may be required either for the relief of pain or the evacuation of pus, in order, if possible, to prevent the disease from spreading to the bony parts.

After incision or spontaneous rupture of the drum membrane, the pain usually ceases, although the temperature may not fall to normal for twenty-four or thirty-six hours, even with good drainage. The discharge is now the principal object of treatment. Nothing else is necessary than to keep the ear perfectly clean. The canal should not be plugged with cotton, nor should it be stopped by the insufflation of powders. It should be syringed with a solution of bichloride (1 to 5,000), or a saturated solution of boric acid, or simply with boiled water. All these fluids should be used warm, and, if the discharge is purulent and abundant, as often as every two or three hours—in all cases several times a day. A bulb ear-syringe of soft rubber is the most satisfactory instrument for general use. It is a mistake to keep the ears covered by a thick mass of cotton or flannel, as is so often done. In the house no protection is necessary. A sudden rise in the temperature usually means that drainage is imperfect; if it is accompanied by pain, a second incision may be necessary. If the temperature remains high, one should be on the lookout for mastoid disease.

In most cases the discharge ceases in from one to three weeks; should it continue longer, some measures for checking it may be used. Dench advises as better than other applications, the use of a few drops of a saturated solution of boric acid in alcohol after syringing. It should be applied with a medicine dropper. Where the discharge has become fetid, syringing once a day with a solution of peroxide of hydrogen (1 to 4, or even stronger) is often useful. A persistent discharge often depends upon the fact that the child's general condition is poor, and improvement in this will do more to stop the discharge than any variation in local treatment.

One attack of otitis is frequently the precursor of many others. Children sometimes have one or more attacks every winter for several years. Such children are usually those who are very prone to catarrhal colds, and in most of them will be found adenoid vegetations in the pharynx. In order to get rid of this tendency to attacks of otitis, such growths should be removed and all other associated pathological conditions treated. The nose should be kept as clean as possible by frequent use of the hand atomizer with some mild cleansing solution, such as Dobell's or Seiler's. The rhino-pharynx may be touched once in two or three days with a solution of nitrate of silver (10 to 30 grains to the ounce).

Cold sponging about the neck and chest should be employed, as well as every means to reduce the susceptibility to acute catarrh. The remote dangers from these recurring attacks are often overlooked. They may be the beginning of a chronic condition, the full effects of which are not

seen until adult life is reached, both the physician and the parents often thinking that all danger has passed when the acute symptoms have subsided.

The treatment of chronic otitis and of the associated conditions is largely surgical, and belongs to the specialist; but it is extremely important that the general practitioner should be familiar with their symptoms, and realize the danger from these neglected cases, not only to the function of hearing, but also to life itself. The essential thing in treatment is to operate sufficiently to secure free drainage, and to permit thorough cleansing of the parts. Too much can not be said against the expectant treatment of these cases, or against the practice of prolonged poulticing.

SECTION IX.

THE SPECIFIC INFECTIOUS DISEASES.

Accurate classification of the infectious diseases is at the present time impossible, but there are two quite distinct groups into which, with one or two exceptions, those here considered may be placed.

The first group includes scarlet fever, measles, rubella, varicella, and pertussis. The nature of the specific poison in each of these is as yet unknown. They are, strictly speaking, contagious; for it is practically certain that any of them may be contracted by proximity to a person suffering from the disease, without actual contact. In no one of these diseases is the poison given off in a single definite discharge, and in no one is there a characteristic visceral lesion. Mumps resembles the members of this group in all points except the one last mentioned. These peculiarities, together with the fact that thus far the poison of each of these diseases has resisted all attempts at isolation, render it not improbable that these poisons are some other variety of micro-organisms than bacteria.

In the second group may be placed diphtheria, typhoid fever, and tuberculosis, in each of which the specific poison is a known form of bacteria. Each of these diseases is associated with definite and characteristic visceral lesions. The poison is discharged from the body in a certain well-understood manner from the tissues which are affected by the disease, and in no other way. These diseases can not be contracted by proximity to a diseased person, but only by receiving into the body the specific germs, either by contact with a person suffering from the disease or contact with something upon which the special germs of the disease have been discharged. In other words, though communicable, they are not, strictly speaking, contagious.

Syphilis, influenza, and malaria have not been included in either of the above groups. Syphilis must still be placed in the doubtful class, although its general characteristics ally it with the second group. The fact that a certain germ—Lustgarten's bacillus—is quite uniformly found in syphilitic lesions also points in the same direction; the evidence, however, is not conclusive that this bacillus is the cause of the disease. In its communicability, influenza resembles the first group, although there is now little doubt that it is due to a form of bacteria—Pfeiffer's bacillus.

Malaria belongs in a class by itself, differing in nearly all its essential features from the other diseases of this general group, as its specific poison is known to be a form of protozoa.

CHAPTER I.

SCARLET FEVER.

Synonym: Scarlatina.

Scarlet fever is an acute, contagious, self-limited disease, one attack usually protecting the individual through life. The period of incubation is usually from two to six days; that of invasion, from twelve to twenty-four hours; that of eruption, from four to six days; that of desquamation, from three to six weeks. The disease may be communicated at any time from the first symptom of invasion throughout desquamation, and sometimes even during the existence of purulent discharges from the nose or other mucous membranes. It is usually ushered in by vomiting, high fever, and sore throat, and is characterized by an erythematous rash appearing first upon the neck and spreading rapidly over the entire body. Its chief complications are otitis and membranous inflammations of the pharynx, which frequently extend to the nose, more rarely to the larynx. The most important sequelæ are deafness and nephritis.

Etiology.—Analogy leads to the belief that scarlet fever is due to a micro-organism, but as yet its nature has not been discovered. The complications are usually associated with the growth of the streptococcus pyogenes. Some have gone so far as to claim that this germ is the cause of the disease. From present knowledge, however, it appears rather to play the rôle of a secondary or accompanying infection, for the development of which the mucous membranes of a person suffering from scarlet fever seem to afford most favourable conditions. To the streptococcus may be ascribed the membranous inflammations of the tonsils and pharynx, the otitis, the inflammation of the lymph nodes and the cellular tissue of the neck, and probably also the nephritis, pneumonia, and joint lesions. In many of the above conditions, the streptococcus is associated with other pyogenic germs, and in some cases with the diphtheria bacillus.

Predisposition.—The susceptibility of children to the scarlatinal poison is much less than to that of measles; still, it is much greater than that of adults. Billington (New York) records observations made in twenty-six families living in tenements where little or no attempt at isolation was made. In these families there occurred 43 cases of scarlet fever; but 47 other children, although unprotected by previous attacks and constantly exposed, did not contract the disease.

Johannessen reports that of 185 children under fifteen years who were

exposed, 28 per cent contracted the disease; while of 314 adults, only 5 per cent contracted the disease. It may be stated that, approximately, not more than one half of the children exposed take the disease. The susceptibility is not great in early infancy, but it increases until about the fifth year, after which it steadily diminishes. Both sexes are equally liable to scarlet fever. Epidemics are more frequent in the fall and winter than in summer, and cases occurring in the cold months are apt to be more severe. Whitelegge, in 6,000 cases, found the highest mortality in the month of October; and in Caiger's report of 1,008 cases this was also the month showing the greatest mortality.

Incubation.—Of 113 cases* in which the period of incubation could be accurately determined, it was as follows:

24 hours or less	6 cases.	8 days	2 cases.
2 days	15 "	9 "	5 "
3 "	28 "	11 "	1 case.
4 "	25 "	14 "	1 "
5 "	6 "	21 "	1 "
6 "	15 "		
7 "	8 "		113 cases.

Thus in 87 per cent of these it was between two and six days, and in 66 per cent between two and four days. The incubation is rarely over a week; it is particularly short in surgical cases, a well-authenticated instance being on record in which it was but six hours. Speaking generally, if, after exposure, a week passes without symptoms, the chances of infection are very small. A short incubation is more frequently seen in severe than in mild cases.

Mode of infection.—The chief source of infection is the patient himself. It is somewhat doubtful whether the poison of scarlet fever can be conveyed by the breath, but it may be by discharges from the mucous membranes involved, from the scales during desquamation, and probably from all the excretions,—urine, fæces, and perspiration of the patient. Infection often takes place from the carpets or furniture of the sick-room, and from the clothing of the patient. In a city the bed-clothing, while airing in the window, has been known to convey the disease to an adjoining house. Instances are recorded of the spread of scarlet fever by the washing of infected with other clothing. Toys or books may be carriers of the disease. A bouquet of flowers sent from a sick-room to an institution, in one instance proved a vehicle of infection. Cats, dogs, and other domestic animals are known to have conveyed the disease. Scarlet fever is sometimes spread by food, particularly by milk, as in the well-known epidemics of Hendon and Wimbledon (England). It is possible, under

* Part of these are from personal observation, but the great majority are isolated cases scattered through medical literature, occurring under circumstances which made it possible to determine the exact length of incubation.

these circumstances, that a disease resembling scarlatina existed in the cows; but that this was identical with scarlatina, as seen in man, was not demonstrated.

The transmission of the disease through a third party is not frequent, but numerous instances of it are on record. The persons most likely to carry it are the nurse and the physician. Physicians have in many cases carried scarlatina to their own children, but only when there had been pretty direct contact with the patient, and where the interval before seeing the second child was short. The clothing of the nurse may be almost as infectious as that of the patient. The transmission of the disease by one who, although living in the house, does not come in contact with the patient is extremely improbable. I can find no instance recorded where scarlatina has been transmitted through two healthy persons.

Duration of the infective period.—There is no evidence to show that the disease is communicable during the period of incubation. It, however, becomes so from the beginning of invasion, even before the rash appears. Infection is doubtless most active during the febrile period—from the second to the fifth day—and, next to this, during the stage of active desquamation.

In simple cases, the average duration of the contagious period may be placed at six weeks, or until desquamation is complete. However, physicians generally have been accustomed to place too much stress upon the danger from the scales, and too little upon that from the discharges from the mucous membranes. Early infection comes chiefly from the throat, nose, or possibly the breath. Late infection may arise from a purulent otitis, rhinitis, chronic pharyngitis, suppurating glands, eczema, empyema, and possibly also from the urine in nephritis. The infectious nature of these purulent discharges has not been sufficiently recognised. It is possible for them to convey the disease during a period of several months. One case is recorded in which scarlatina was communicated through a purulent nasal discharge after eleven weeks; another in which the opening of a post-scarlatinal empyema in a surgical ward was followed by an outbreak of scarlet fever.

In winter especially, a chronic pharyngeal catarrh may long contain the germs of infection. Ashby found, on careful investigation, that from two to four per cent of patients discharged from a scarlet-fever hospital subsequently conveyed the disease. There is particular danger from a child who has recently had the disease sleeping with other children. Line records a case in which this was the means of conveying the disease after fourteen weeks, and when the patient had been considered perfectly well for three weeks. It is impossible to say that at any specified time absolute safety exists. All patients before being discharged from a hospital or released from quarantine in private practice, should be carefully examined as to the condition of the mucous membranes, and quarantine

continued as long as catarrhal inflammations are present. The poison of scarlatina clings more tenaciously to clothing, upholstery, and apartments than that of any other contagious disease, possibly excepting tuberculosis. Authentic cases are on record in which more than a year had elapsed between the first and second cases, where the source of infection seemed certain.

Lesions.—The only essential lesions of scarlet fever are those of the skin and the mucous membrane of the throat. The other changes occurring in this disease are considered in the light of Complications, under which head they are described.

The earliest changes in the skin consist in an intense hyperæmia with dilatation of all the small blood-vessels; following this, there is an exudation of round cells into the rete Malpighii, and considerable swelling, due partly to the exudation of cells and partly to œdema. There are also thickening of the lining membrane of the sweat ducts, and infiltration about these ducts with round cells. In some cases there is destruction of the epithelium lining the sweat ducts, and the lumen of the duct is filled with granular detritus, occasionally with blood. The local process results in death of the epidermis, which is cast off during desquamation. It is essentially an acute dermatitis, which varies in intensity with the severity of the attack. The only constant lesion in the throat is an erythematous pharyngitis, with the usual changes of a catarrhal inflammation.

Symptoms.—*Invasion.*—As a rule, the invasion of scarlet fever is abrupt, the symptoms at the onset usually being directly in proportion to the severity of the attack. In the majority of cases there are vomiting, a rapid rise in temperature, and soreness of the throat. Often the vomiting is repeated; it is frequently forcible, and without nausea. In severe cases the rise in temperature is very rapid, to 104° or 105° F.; in the mildest cases it may not be above 101°. A child may complain of soreness of throat, or the throat symptoms may be entirely objective. In most severe cases, there is a uniform erythematous blush covering the pharynx, tonsils, and fauces, but on the hard palate consisting of minute red points. The appearance of this is usually coincident with the rise in temperature. Occasionally membranous patches may be seen upon the tonsils the first day, but not generally before the third or fourth day In mild cases the throat shows only a very moderate congestion, and in some presents nothing abnormal. Severe cases are sometimes ushered in by convulsions, especially in very young children. Diarrhœa is not uncommon in summer. There is general prostration, which is directly proportionate to the height of the fever.

Eruption.—This usually appears from twelve to thirty-six hours after the first symptoms of invasion; exceptionally, not until the third or even the fifth day. A later appearance than this is somewhat doubtful, for the rash not infrequently recedes and reappears, having been overlooked in

the first instance. In 108 cases observed in the New York Infant Asylum, the duration of the rash was as follows:

Two days or less	5 cases.
Three to seven days	81 "
Eight to eleven days	16 "
Over eleven days	4 "
Recurring	2 "

These statistics are confirmed by the observations of most writers, that the rash lasts from three to seven days. The full development of the rash is generally seen in from twelve to twenty-four hours from its first appearance, and not infrequently the whole body is covered in the course of four or five hours. Very rarely its extension is so slow that it is two or three days before the body is covered. Its first appearance is almost invariably upon the neck and chest. Where the rash is faint, it is sometimes earliest and most intense over the sacrum, buttocks, and back of the thighs. In the cases of moderate severity the typical rash is seen. It is of a bright scarlet colour, and on close inspection is seen to be made up of very minute points; it covers the entire body, including the face. There is often a peculiar pallor about the mouth, in striking contrast with the rest of the face, which is quite characteristic of the disease.

Variations in the eruption are very frequent, and often extremely puzzling. In the mild cases the rash is not seen upon the face; it is often faint upon the body, and may be present only upon certain parts; it may last only one day, and sometimes may be so slight as to escape notice altogether. It may be absent in some very mild cases, in certain others where the throat symptoms are severe, and in malignant cases. In the very severe cases many irregularities are seen, both as to the time of the appearance of the eruption and its character. Sometimes it occurs as large, irregular patches; at others it is macular, closely resembling the rash of measles; occasionally it is of a dark purplish colour; and very rarely it is hæmorrhagic. An eruption of fine miliary vesicles has been observed in connection with a fully-developed rash. Much importance is attached by the laity to the early disappearance of the rash, an especial danger being believed to exist because the disease has "struck in." A well-developed bright scarlet rash indicates strong heart action, and a sudden recession of the rash is a sign of heart failure. Often a rash which is faint and doubtful in character, may be brought out fully by a hot bath.

With the eruption at its height, there is intense itching or burning of the skin, and in severe cases considerable swelling, chiefly noticeable upon the hands and face. All the constitutional symptoms increase in intensity as the rash develops, and usually diminish gradually as it fades.

Desquamation.—Shortly after the rash has faded there is an exfoliation of the dead epidermis, known as desquamation. This is even more characteristic of the disease than the rash. It is usually first seen upon

the neck and chest, where it appears as fine scales or small patches. The desquamation of the trunk is completed in from one to three weeks. If baths and inunctions are being used, it is scarcely perceptible. It continues longest where the epidermis is thickest—viz., upon the hands and feet—and here it lasts from three to six weeks, and not infrequently eight weeks. The appearance of the fingers and toes during desquamation is characteristic. The finger tips usually peel first, and the new epidermis is pink and fresh-looking, while that which has not yet separated is of dull gray colour and loosened at the margin. Occasionally the epidermis of a considerable part of a finger may be loosened at once, so that a partial cast may be thrown off like the finger of a glove. Sometimes the patient comes under observation for the first time during desquamation, the history of the early symptoms being doubtful or absent. Such desquamation as has been described, occurring both upon the hands and feet, may be regarded as conclusive evidence of scarlet fever, no matter what the history may be.

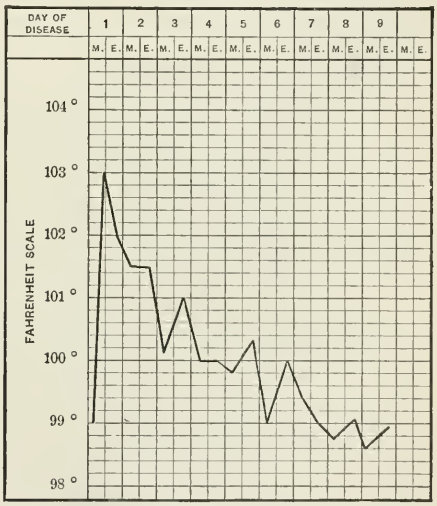

Fig. 154.—Typical temperature curve of mild scarlet fever; uncomplicated; in a child three years old.

1. *The mild cases.*—The symptoms may be so slight as to be entirely overlooked, nothing being noticed until desquamation occurs. Usually, however, there is a rather abrupt invasion, with vomiting and a temperature of 100° to 103° F. The tonsils and pharynx are congested, while the palate shows a punctate redness somewhat like the cutaneous eruption. Nearly always within twenty-four hours the rash makes its appearance, generally first upon the neck and chest. Very often it is not seen upon the face, but the rest of the body is usually covered. The rash fades on the third or fourth day, and has disappeared by the fifth day. There is very little prostration, the child often being with difficulty kept in bed.

The highest temperature is coincident with the full eruption, and is seen during the first thirty-six hours of the disease. It gradually falls to normal by the fourth or fifth day. Its typical course is shown in Fig. 154. In the mildest cases the temperature may never be above 100° F., and the rash may last but one day, and even then may come out very imperfectly and over only a portion of the body—the chest or the loins.

Desquamation is often faint over the body, but is unmistakable over

the hands and feet. It begins about the end of the first week, always being most marked where the eruption has been most intense.

The mild cases are usually uncomplicated, but the possibility of otitis and of late nephritis should always be kept in mind, as these may occur even with the mildest attacks. The difficulties in diagnosis in mild attacks of scarlet fever are often great. It should be remembered that these cases are just as contagious as severe ones, and that from a mild attack a severe one is often contracted. It is frequently by these mild cases that this disease is spread in schools. In dispensaries I have often seen patients desquamating from scarlet fever, who had been attending school regularly up to the time when they were brought for treatment for nephritis or some other disease.

2. *Cases of moderate severity.*—The onset is sudden with vomiting, which is usually repeated, or with convulsions. The temperature rises

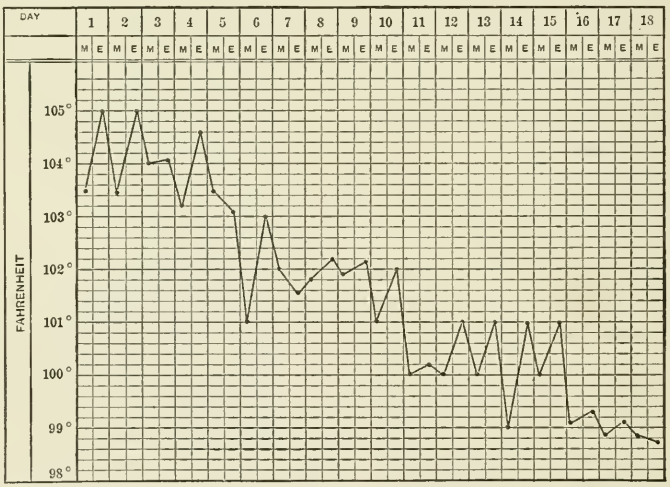

Fig. 155.—Moderately severe scarlet fever, running a prolonged course, but without complications; the patient, a boy two and a half years old.

rapidly, and by the end of the first twenty-four hours has reached 104° or 105° F. The rash usually appears within the first twenty-four hours, and its intensity is directly proportionate to the severity of the attack. Appearing first upon the neck or chest, it extends rapidly, covering the entire trunk, extremities, and often the face in a few hours. It is usually typical in appearance, being made up of minute points, but giving the appearance of a uniform blush, which has been compared to a boiled lobster. Little change takes place in the rash for four or five days. After this it fades quite rapidly, and disappears by the seventh or eighth day.

The throat resembles that of the mild form, except that the redness is more intense and there is slight swelling of the tonsils, fauces, and uvula,

and often pain upon swallowing. Occasionally small yellowish patches are seen upon the tonsils by the second or third day, but these can be wiped off and are not distinctly membranous. There is usually a moderate discharge of a sero-purulent character from the nose. The lymphatic glands at the angle of the jaw are swollen and quite tender. The tongue shows first a white, frosty coating, and after a few days may clear at the border. The intense redness at the tip and margin of the tongue, with the enlarged papillæ, gives rise to what is known as the "strawberry tongue," which, though not peculiar to scarlet fever, is a very frequent symptom.

During the height of the fever there are restlessness, thirst, and not infrequently slight delirium. The temperature reaches the maximum by the second or third day, and usually falls gradually after the fourth or fifth day, but even in uncomplicated cases the fever often lasts from ten to fourteen days (Fig. 155). The pulse in the early part of the disease is rapid and full, but later it may be weak. There is much prostration, frequently followed by quite a marked degree of anæmia.

This form of the disease rarely proves fatal apart from complications, but it may do so in very young infants. The complications seen most frequently in this form of scarlet fever are broncho-pneumonia or pleuro-pneumonia and otitis, the latter being usually double and occurring between the sixth and the fourteenth days. Nephritis is the only common sequel.

3. *The severe cases.*—The severe type of scarlet fever usually declares itself from the beginning. The incubation is short, and the full rash may be seen within a few hours after the initial symptoms. It covers the entire body, including the face. The severity of the infection is shown by the fact that the temperature is higher and continues for a longer period, and by the frequency and severity of the complications, particularly those of the throat. For the first two days the throat presents nothing different from what is seen in the milder cases. By the third or fourth day, however, membranous patches often appear on the tonsils, and spread to the soft palate, uvula, and pharynx, sometimes to the nose and through the Eustachian tube to the ear, rarely to the larynx. The mucous membrane of the mouth is intensely congested, and often partly covered by membrane; there is sordes on the lips and teeth, and there may be superficial ulcers, which bleed readily. The glands of the neck swell rapidly, often to a great size, and the cellular tissue about them is infiltrated. The head is thrown back to relieve the dyspnœa which the pressure from this swelling occasions. There is an abundant discharge from the nose and mouth; the breath is offensive, often fetid. The general symptoms are those of a severe septicæmia. The temperature is steadily high, usually between 103° and 105° F., the fluctuations being usually narrow for the first week or ten days. In cases which recover, the subsequent course is

greatly modified by the presence of complications (Fig. 156). The fever generally lasts from three to four weeks. In fatal cases the temperature may be steadily high till death (Fig. 157), or may fluctuate widely. The pulse is rapid, weak, and irregular. There is complete anorexia; both food and stimulants have to be coaxed or forced down. There is low delirium or apathy, and sometimes all the symptoms of the typhoid condition are present.

Signs of a broncho-pneumonia are often found in the chest, and by the end of the first week or early in the second the ears begin to discharge. The urine is rarely free from albumin, but the amount present is not usually great; there may be hyaline and epithelial casts, and often blood. In some cases the throat symptoms predominate; in others, those of general sepsis, but more frequently the two are combined and are directly proportionate to each other. In still other cases, instead of the membranous inflammation, it may be of a gangrenous character, and extensive sloughing may take place in the throat, and even in the cellular tissue of the neck.

The duration of the symptoms in fatal cases is from six to fourteen days. There are generally increasing prostration and finally a septic stupor, with death from exhaustion, from sudden heart failure, or from some of the complications,—broncho-pneumonia, pleurisy, nephritis, hæmorrhages following sloughing, laryngitis, pericarditis, or endocarditis. In cases which recover, the acute symptoms nearly always continue for a full

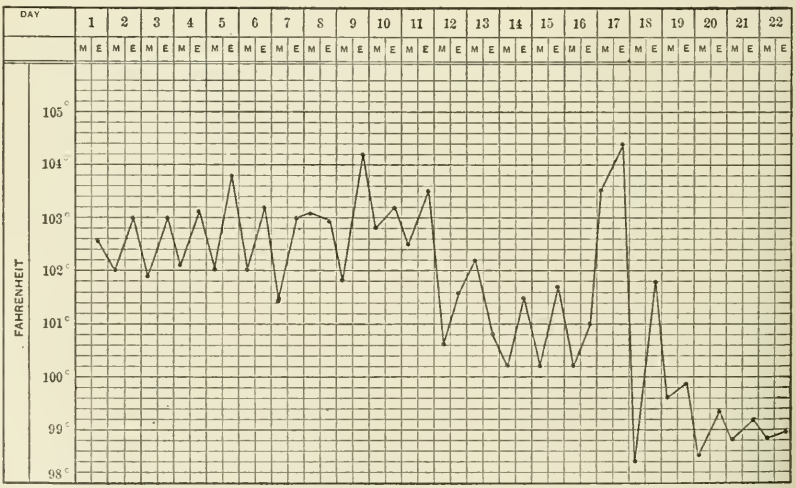

Fig. 156.—Severe scarlet fever complicated by double otitis and nephritis; primary fever prolonged; otitis began on the thirteenth day; nephritis on the nineteenth day; recovery; the patient a girl twenty months old.

month; and after escaping the dangers of sepsis and the early complications, the child has still to run the gantlet of all the late complications—nephritis, pneumonia, endocarditis, pyæmia, etc. A case may prove fatal

as late as the end of the seventh week; nearly all such results are due to nephritis or to its complications.

4. *Malignant or cerebral cases.*—These are rare cases which are more frequently described than seen, and in which death takes place usually within the first forty-eight hours. The system is overpowered by the scarlatinal poison. Such cases are seen only in severe epidemics. Under other circumstances, many cases of unexpected death with high temperature are diagnosticated malignant scarlet fever which have no connection with this disease.

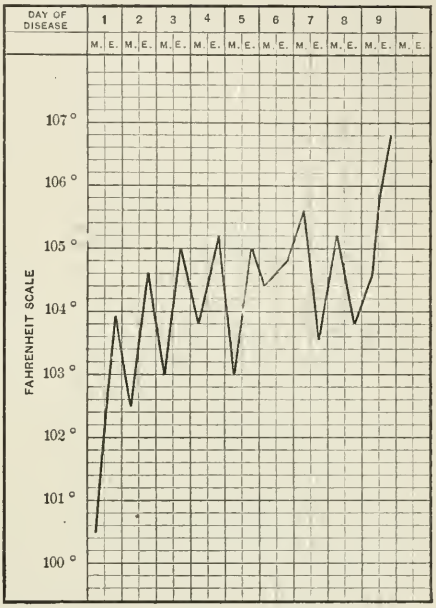

Fig. 157.—Severe scarlet fever, septic type; double otitis, severe membranous angina; death on the ninth day; the patient a girl seven years old.

The onset is sudden and violent, usually with convulsions, the child passing in a few hours into a condition of deep stupor, with great prostration and hyperpyrexia, the temperature ranging from 105° to 107° F. The rash appears irregularly, late, or not at all. There are frequently repeated convulsions, cyanosis, and invariably a fatal termination. The autopsy often gives no satisfactory explanation of these cases. Death occurs from toxæmia, without any characteristic local evidences of disease.

5. *Surgical scarlet fever.*—Patients with recent wounds, or those who have been subjected to surgical operations, are peculiarly susceptible to the scarlatinal poison, and are almost certain to contract the disease upon exposure, unless protected by a previous attack. Whether the infection takes place directly through the wound, or whether the susceptibility depends upon the diminished resistance of the patient, is still an open question. This disease doubtless explains some of the unexpected deaths occurring after minor surgical operations. Scarlet fever may occur after any operation, even one so trivial as tenotomy or circumcision. Patients with burns are generally believed to be especially susceptible. The effect of scarlet fever upon the wound, and some of its peculiar clinical features, are illustrated by the following cases from Walton Browne (Belfast):

A healthy child was operated upon for hare-lip; sixteen hours afterward it became seriously ill, the skin was covered with a dark scarlatinal rash, and death quickly followed. Another patient who, it was afterward

learned, had been recently exposed directly to scarlatina, was circumcised for congenital phimosis. In thirty hours he was covered with a scarlatinal rash and had a temperature of 104° F. In forty hours the wound became gangrenous and the patient passed into a condition of coma, in which he died in seventy hours. A child admitted to the hospital with a lacerated wound of the leg was accidentally placed in a bed next to one in which was a patient who had just developed scarlatina. The exposure lasted less than an hour, but in six hours the child was taken with vomiting, high fever and headache, became rapidly comatose, and died in fifteen hours, no rash having appeared. After death, however, a purpuric rash could be seen upon the skin.

Surgical scarlatina is nearly always irregular in its symptoms; the incubation is very short, the rash usually atypical, and the general symptoms, particularly those relating to the nervous system, especially severe. There may or may not be throat symptoms. It should be said that many writers deny that surgical scarlet fever is anything more than septicæmia with an erythematous rash. This is undoubtedly true of some of those reported as surgical scarlet fever; but it certainly is not the explanation of all. That some of these are cases of genuine scarlet fever is shown by the fact that they have been known to communicate that disease, and that they are often followed by nephritis and usually by desquamation, although the latter is not invariable. But in the absence of throat symptoms, desquamation, and contagion, the diagnosis of scarlatina should be made with extreme caution. Care should be taken to exclude erythematous eruptions due to the various antiseptics used in surgical dressings.

Relapses, Recurrences, and Second Attacks.—As a rule, one attack of scarlatina gives immunity through life. The exceptions are very few, but some of them are well authenticated. Kinnicutt (New York) observed two attacks within eight months in a boy of five years; Pritchard (Glasgow) reports the case of a patient who had three attacks in the same hospital within two years; such cases are certainly extremely rare.

Relapses or recurrences within a brief period after the first attack are more frequent. There are to be excluded the cases of pseudo-relapses in which the rash, having temporarily subsided for two or three days, reappears; also those where the rash varies in intensity from time to time; and, lastly, the cases in which, occurring late in the disease, it is due to septicæmia or pyæmia. True relapses are usually due to auto-infection, sometimes to a new accession of poison from without. They are analogous to the relapses of typhoid fever. They occur most frequently during desquamation, between the seventh and twenty-fourth days. There may be not only a new eruption but a rise of temperature, sore throat, and vomiting, just as in the initial attack. These recurrences are sometimes shorter and milder than the first attack, but this is by no means uniform, since Körner mentions eight cases where the second attack proved fatal.

In considering the subject of second attacks, the liability to errors in diagnosis must be borne in mind and only cases included which have presented typical symptoms.

Complications and Sequelæ.—*Throat.*—Three distinct forms of angina are seen in scarlatina: simple or erythematous, membranous, and gangrenous.

1. Erythematous angina.—This can hardly be ranked as a complication, as it is nearly as constant as the scarlatinal rash. Usually there is only the general blush over the entire pharynx with the fine red points upon the hard palate; but there may be seen upon the tonsils grayish-yellow spots resembling those of follicular tonsillitis, which can be wiped off, leaving a clean surface. This simple angina is at its height with the maximum temperature, and fades as the temperature falls. It does not often extend to adjacent mucous membranes.

2. Membranous angina.—These cases were formerly classed as scarlatinal diphtheria, and whether this process was identical with primary diphtheria or not, was for a long time a subject of much discussion. This question has, however, been settled by bacteriology. It is now generally agreed that the membranous angina which occurs early in scarlet fever, and that which develops at the height of the disease, are almost invariably due to the streptococcus, the diphtheria bacillus being rarely found; but that the cases which develop late in the disease, and after the primary fever has subsided, are almost invariably true diphtheria, the bacillus being regularly present. The latter condition is to be regarded as scarlet fever complicated by diphtheria.

The lesions of this form of angina are considered in the chapter on Pseudo-Diphtheria. Usually on the second or third day of the disease the membrane appears upon the tonsils, and in the milder cases it covers only the tonsils. In the most severe form it may be seen within twenty-four hours of the onset, frequently before the eruption appears. Beginning upon the tonsils, the membrane rapidly spreads to the entire pharynx, the mucous membrane of the nose, the mouth, the Eustachian tube, and even the middle ear. In colour it may be gray, greenish, or almost black. There is so much swelling of the throat that swallowing becomes difficult. The infiltration of the cellular tissue of the neck and the enlarged lymphatic glands produce great external swelling, which may extend like a collar from ear to ear. The breath has a foul odour, the nasal discharge is thin and fetid, and nasal respiration is obstructed, so that the mouth is open constantly. Occasionally the larynx is invaded, with the usual symptoms of membranous croup.

These local changes are accompanied by constitutional symptoms of great severity, which are due to a general streptococcus septicæmia; broncho-pneumonia and nephritis are very frequent, otitis is almost constant, and suppuration of the lymphatic glands is not uncommon.

As the eruption in these cases is late and often very irregular in appearance, the diagnosis from true diphtheria is often a matter of great difficulty, and a positive diagnosis is possible only by making cultures from the throat.

3. Gangrenous angina.—This is seen only in the worst cases of scarlet fever. The process may be gangrenous from the outset, or preceded by a membranous inflammation. It is sometimes insidious in its development. There is a fetid odour to the breath, irritating discharges from the nose and mouth, with very great glandular swelling. The tonsils are gray or grayish-black in colour, and large masses of necrotic tissue may be removed with the forceps from the tonsils, uvula, fauces, or pharynx, and sometimes sloughing occurs in the cellular tissue of the neck. Blood-vessels of considerable size are often opened, and serious, or even fatal hæmorrhage may result. Little or no tendency to a reparative process is seen. The constitutional symptoms are those of great asthenia, prostration, and profound cachexia, followed almost invariably by a fatal termination.

Lymph nodes.—These are swollen in all cases accompanied by severe angina. The inflammation may be simply an acute hyperplasia, or it may go on to suppuration. Abscess does not often occur at the height of the disease, but may come at any time during convalescence. It may be confined to the glands or be complicated by suppuration in the cellular tissue of the neck. Disease of these glands is not an infrequent cause of torticollis.

Cellulitis of the neck.—This usually occurs toward the end of the first week, and is associated with grave throat symptoms. Rapid and extensive infiltration occurs, the skin becomes tense and brawny, the head is held back, and there may be considerable dyspnœa. The infiltration may be only in the neighbourhood of the lymphatic glands or it may be diffuse. Unless relieved by early incision, the diffuse form may result in suppuration and extensive sloughing, which may be deep enough to lay bare the large vessels of the neck. This is a complication of the gravest possible import. Death may occur from septicæmia before or after sloughing or from hæmorrhage due to opening by ulceration of the external carotid or some of its branches; or there may be associated thrombosis of the jugular vein, leading to thrombosis of the lateral sinus, meningitis, or pyæmia.

Ears.—The otitis is due to direct extension of the infection from the rhino-pharynx. It is the most frequent complication of scarlatina, and in doubtful cases may have some diagnostic importance. As a rule, the younger the child the greater the liability to otitis. It is more frequent in winter than at other seasons. Like all complications, it varies greatly with the epidemic, and is closely connected with the severity of the throat symptoms. In an epidemic occurring in the New York Infant Asylum in the spring and summer of 1889 there were 73 cases of scarlatina and not one of otitis. In a fall and winter epidemic in the same institution

two years later, of 43 cases 20 per cent had otitis. Of 4,397 cases reported by Finlayson, otitis occurred in 10 per cent, and of 1,008 cases reported by Caiger, in 13 per cent. In Burkhardt's statistics the proportion was as high as 33 per cent. Of cases accompanied by severe throat symptoms otitis is present in fully 75 per cent.

As a rule, both ears are affected, but not simultaneously, or at least rupture occurs at different times. This is most frequent early in the second week, but may occur during convalescence. In the cases where otitis develops at the height of the disease there are in some cases no new symptoms; in others there are pain and deafness. If it develops at a later period there is usually a rise in the temperature, which falls after rupture of the drum membrane takes place. The otitis is sometimes overlooked until symptoms of pyæmia or meningitis develop. The form of inflammation may be catarrhal or suppurative (page 880), the latter being often accompanied by necrotic changes.

Bezold makes the following report upon 185 cases showing the results of scarlatinal otitis: "In 30 there was entire destruction of the membrana tympani, with loss of one or more bones; in 59 the perforation comprised two thirds or more of the membrane; in 13 there were smaller perforations; in 44 there were granulations or polypi; in 15 there was total loss of hearing on one side, and in 6 of the cases upon both sides; in 77 of the cases the hearing distance for low voice was less than twenty inches."

As a cause of permanent deafness and deaf-mutism, no disease of childhood compares in importance with scarlet fever. May (New York) has collected statistics of 5,613 deaf-mutes, of whom 572 owed their condition to otitis following scarlet fever.

Kidneys.—Albuminuria accompanies nearly all the severe cases of scarlet fever. In many this is simply the ordinary febrile albuminuria due to acute degeneration of the kidneys (page 612). In those with severe throat complications, and in nearly all the septic cases, there is an acute inflammation of the kidney, usually of the variety described as acute exudative nephritis (page 613). This occurs at the height of the febrile process and is rarely accompanied by dropsy; but albumin, casts, and even blood may be found in the urine. The most severe and the most characteristic renal complication, and that generally designated as *post-scarlatinal nephritis*, is a diffuse nephritis which in most cases develops during the third week of the disease. It is accompanied by general dropsy; the urine is scanty and not infrequently suppressed, and it contains a large amount of albumin and great numbers of casts of all varieties. It may cause death by the occurrence of acute uræmia, or it may be followed by permanent damage to the kidneys. It is more fully described with the Diseases of the Kidney (page 615).

Joints.—Acute articular rheumatism may occur coincidently with the development of the scarlatinal rash, and occasionally during convalescence

in patients who have a predisposition to that disease. Acute swelling of the joints is sometimes of pyæmic origin. A case is reported by Henoch in which this was due to an infectious thrombus in the jugular vein, associated with cellulitis of the neck. In pyæmic arthritis the large joints are usually involved and the lesions are apt to be multiple. Joint disease may occur as a sequel of scarlet fever, where it is secondary to disease of the bone or to periarticular abscesses opening into the joint.

The foregoing include but a small proportion of the joint complications seen in scarlet fever. The most frequent and most characteristic form of inflammation is *scarlatinal synovitis*, or, as it is sometimes called, *scarlatinal rheumatism*. It occurs in different epidemics with varying frequency. Carslaw (Glasgow) in 533 cases of scarlet fever met with synovitis in 60 patients. It is seldom seen in children under three years of age, and is most frequent after five years. It may occur in mild as well as in severe cases. According to Ashby, it is more frequent when the febrile stage is prolonged, owing to other complications. Synovitis develops quite uniformly toward the end of the first or the beginning of the second week. The symptoms are generally mild, and are followed by prompt recovery. Suppuration is rare. Any of the joints may be attacked, but those of the wrist and hand are most frequently and often the only ones affected. Demme (Berne) has reported a case in which every large joint in the body was involved. The symptoms are redness, moderate pain, swelling, which is usually due to synovial distention, and sometimes a slight rise of temperature. The duration is generally but three or four days, and in most cases there is spontaneous recovery. This disease is distinguished from rheumatism by several points: it is not more frequent in rheumatic patients; cardiac complications are rare as compared with those seen in patients with genuine rheumatism; in some epidemics it is very common, and in others seldom seen; there is little or no tendency to relapses; anti-rheumatic remedies are without striking benefit; it does not skip about from joint to joint, and usually fewer joints are involved.

Lungs.—The pulmonary complications of scarlet fever are neither so frequent nor so important as those of measles. Broncho-pneumonia is usually found at autopsy in septic cases where death has occurred later than the third or fourth day, but it is not generally recognisable by physical signs.

In septic cases pleuro-pneumonia sometimes occurs early in the disease and at other times late, generally associated with nephritis, but occasionally without it. It is always a serious condition and not infrequently a direct cause of death. Empyema may follow pleuro-pneumonia or occur with pyæmia or nephritis, but with the latter, simple serous pleurisy is more common. Œdema of the lungs occurs chiefly with nephritis, in which it is the most common cause of death.

Heart.—Abnormal cardiac sounds, not dependent upon organic lesions, are frequent during the height of the disease. Endocarditis and pericarditis are not common. They are occasionally seen in septic cases and in those complicated by pyæmia, but principally as a complication of post-scarlatinal nephritis or in rheumatic patients. Endocarditis may be simple or malignant, and may be the cause of embolism and hemiplegia during convalescence.

A certain degree of degenerative change in the cardiac muscle is found in nearly every fatal case that has lasted over four days. More marked evidence of toxic myocarditis is not infrequent in the prolonged cases and in those of a septic type. This may be followed by acute dilatation of the left ventricle or of the entire heart, and it may be a cause of sudden death.

Digestive system.—Functional disturbances are very frequent, and, in fact, are seen in most of the cases, but organic changes are rare. Vomiting is the mode of onset in the majority of cases, but rarely continues through the attack. Late in the disease it is a frequent symptom of uræmia. Diarrhœa may be associated with it under both conditions. The tongue is nearly always coated, and clears off in quite a characteristic way, which, with the prominent papillæ, gives rise to the "strawberry" appearance. Catarrhal stomatitis is a very frequent complication, and in many cases of severe membranous angina the same process is seen in the buccal cavity.

Nervous system.—Nervous complications and sequelæ are seen less frequently with scarlatina than with most of the infectious diseases of such severity. Convulsions are frequent at the outset, and generally indicate a severe attack, though not invariably so. Occurring late in the disease, they are usually due to uræmia, and may be a cause of death. Meningitis may occur as a complication of otitis, in pyæmic cases, and sometimes with post-scarlatinal nephritis. Paralysis from peripheral neuritis is rarely seen. Hemiplegia sometimes occurs from meningeal hæmorrhage, or from embolism secondary to endocarditis and associated with nephritis. Chorea was noted as a sequel in only three of 533 cases reported by Carlslaw. In a report of 187 cases of epilepsy, Wildermuth states that it followed scarlet fever in 12 cases. Insanity has been occasionally observed, the usual form being acute mania, with complete recovery in a few weeks or months.

Gangrene.—Cases of symmetrical gangrene after scarlet fever have been reported by Wilson and others. The parts generally affected are the buttocks, thighs, and arms, but it may occur almost anywhere. The pathology of these cases is obscure. The process usually begins in several places simultaneously, or in rapid succession, and advances steadily till death occurs.

Other infectious diseases.—Scarlet fever is not very infrequently complicated by other forms of infectious disease. It is seen with diphtheria,

measles, varicella, erysipelas, and occasionally with variola and typhoid fever. The symptoms are an irregular commingling of those belonging to the two diseases. They may begin simultaneously, or more frequently one develops as the other is subsiding.

Diagnosis.—The characteristic symptoms of scarlet fever are the abrupt onset, usually with vomiting, the marked elevation of temperature, the erythematous condition of the throat, and the appearance of the rash within twenty-four hours. Before the eruption it can not be diagnosticated from tonsillitis or many other diseases. The difficulties of diagnosis usually depend upon irregularities in the eruption, both as to the time of its appearance and its character. These variations are seen in the mildest, and in the most severe cases. In the former the temperature may not be above 100·5° F., the rash may last less than a day, and may be seen only upon the chest and neck, or there and upon the loins, but very often it does not cover the trunk and extremities. Nothing is positively diagnostic about these symptoms, even when associated with some degree of redness of the throat, which is by no means constant. But the appearance after them of desquamation is usually conclusive. In some cases, however, this is of so uncertain a character that, even after the entire course of the disease, the diagnosis may remain in doubt. A history of an undoubted exposure within a week prior to the onset, or the fact that other cases of scarlet fever subsequently develop in the family or hospital, greatly strengthens the diagnosis.

Cases of malignant scarlet fever which prove fatal before a characteristic eruption appears, can not be diagnosticated with certainty; but when such cases are preceded or followed by others of a typical character, the diagnosis can be made with a strong degree of probability.

The form of the disease in which the throat symptoms are of great severity and appear early, are often difficult to distinguish from true diphtheria. Here the only reliable ground of distinction is that afforded by the bacteriological examination. There are, however, points in the local appearances which are of some assistance in the absence of the culture test. These are discussed in connection with the Diagnosis of Diphtheria.

The eruption of scarlet fever may be confounded with that of measles, rubella, urticaria, and various forms of erythema. The typical eruption of measles has little that suggests scarlet fever, appearing as it does first upon the face and spreading slowly over the body; but in irregular cases the eruption may resemble neither disease. The diagnosis must then rest upon the other symptoms: the sudden onset with vomiting in scarlet fever, or the gradual onset with marked catarrhal symptoms in measles. The eruption of rubella is more difficult to distinguish. In this disease the important thing is that, although the rash may be well marked, often covering the body, the constitutional symptoms are few or entirely absent. In scarlet fever with an eruption of the same intensity there is in-

variably a considerable elevation of temperature, usually 102° to 103° F., and a bright red throat.

There are so many skin eruptions which may resemble that of scarlet fever, that it is always hazardous to make the diagnosis of this disease from the eruption alone. This is especially true of sporadic cases occurring in infants; there is seen at this age a great variety of eruptions, usually associated with digestive disturbances, which closely simulate a scarlatinal rash; but most of them are of short duration. A scarlatiniform erythema is occasionally seen in diphtheria, influenza, typhoid fever, and varicella, which may cause them to be mistaken for scarlet fever, or may lead to the diagnosis that both diseases are present. The same is the case with the septic erythema occurring in surgical patients. Belladonna, quinine, and occasionally antipyrine, may produce eruptions more or less closely resembling that of scarlet fever. This is also true of some cases of urticaria, and of several other forms of skin disease. There is little doubt that many of the cases reported as relapsing scarlatina are really examples of recurring erythema, particularly as some of the latter are followed by a desquamation which is very similar to that after scarlatina. In all doubtful conditions great importance is to be attached to the constitutional symptoms.

Prognosis.—The mortality of scarlet fever varies much in different epidemics. In some, nearly all the cases are of a mild type, and the mortality may be as low as 3 or 4 per cent; in others, a severe or malignant type prevails, and it may be as high as 40 per cent. The disease is, as a rule, more fatal in the youngest infants, becoming less so as age advances. This is well shown in two recent epidemics in the New York Infant Asylum. There were—

Under one year	29 cases;	mortality,	55 per cent.
From one to two years	37 "	"	22 "
" two " three "	28 "	"	7 "
Over three years	23 "	"	0 "

In the first epidemic the general mortality was 12·5 per cent; in the second it was 33 per cent, in the same class of children.

The following are the mortality records from various European sources:

Ashby, Manchester Hospital	681 cases;	mortality,	12·2 per cent.
Koren, a single epidemic	426 "	"	14·0 "
Bendz, Copenhagen	22,036 "	"	12·2 "
Ollivier, three Paris hospitals for five years	893 "	"	14·5 "
Fleischmann, five epidemics	1,356 "	"	10·0 "

The general mortality of the disease may therefore be assumed to be from 12 to 14 per cent; it is, however, much higher than this among young children, as shown by the following figures:

New York Infant Asylum...	116 cases	under	5	years;	mortality,	20	per cent.
Ashby, Manchester Hospital.	259 "	"	5	"	"	23	"
Bendz.....	not stated "		5	"	"	13	"
Heubner.....	136 cases "		7	"	"	30	"
Fleischmann.....	not stated "		4	"	"	43	"

Under five years of age the average mortality from scarlet fever is, therefore, between 20 and 30 per cent.

The fatal cases may be grouped in three classes: first, those due to late nephritis, in which the early symptoms of the disease are of moderate severity or even mild; secondly, the septic cases, usually associated with severe throat symptoms and dying most frequently in the second week from exhaustion, or from some local complication, such as laryngitis, pneumonia, pleurisy, meningitis, or nephritis; thirdly, the malignant cases, which are overpowered by the poison of the disease in the first two or three days of the attack.

Prophylaxis.—Even the mildest cases should be isolated for six weeks, or until desquamation is completed. If complications exist, such as otitis, rhinitis, pharyngitis, empyema, or suppurating glands, the quarantine should be continued until these conditions are cured. Patients should not be allowed to mingle with other children for at least a month after all symptoms have subsided, and should be forbidden to sleep with other children for three months. Children in the house who have not been exposed to the disease should be immediately sent away; and those who have been exposed, separately quarantined for at least a week. After recovery, the patient, before mingling with other children, should have at least two disinfectant baths, the entire body being scrubbed with soap and water and then washed in a solution of carbolic acid (1 to 50) or bichloride (1 to 5,000), and every particle of clothing changed. The hair, if long, should be cut short, and the scalp thoroughly washed and disinfected.

The nurse should be quarantined with the patient, and should not mingle with other members of the family until a complete change of clothing has been made, and hands and face thoroughly disinfected. The nurse and all others in close contact with a severe case should use an antiseptic gargle four or five times a day and a nasal spray at least twice a day.

The room should be in that part of the house most easily quarantined, usually on the top floor; during the attack it should be stripped of upholstery, hangings, and carpet, should be freely ventilated, and kept as clean as possible, the floor being frequently sprinkled with a bichloride solution (1 to 1,000). The presence in the room of vessels filled with antiseptic fluids is of no practical value, and often harmful, in that it creates a false sense of security. The same may be said of sheets wet in carbolic or other solutions and hung about the room. Carbolic-acid poisoning has been known to result from this practice. After an attack it should be remembered that the room is probably a greater source of danger than

the patient. Smooth walls should be wiped with damp cloths wrung out of a bichloride solution (1 to 2,000), or should be rubbed down very carefully with bread. The wood-work should be washed in the same solution and the floor thoroughly scrubbed with it. After a severe case, the walls should be painted or whitewashed, or if papered, the wall-paper should invariably be renewed and the wood-work repainted. Simply airing a room after an attack is of little or no benefit. An instance is on record of a patient contracting the disease in a room in which the windows had been open constantly for three months. The carpets, bedding, hangings, and upholstery are best disinfected by steam. Where this is impossible, after a severe case they should be burned; after milder cases, articles which can be boiled should be treated in this manner, and others exposed to sunlight for a long time out of doors, or, after having been moistened, should be fumigated with sulphur in the sick-room. The mattress should be burned. As ordinarily employed, sulphur fumigation is of very doubtful efficacy, and should never be alone depended upon.

The bedclothes, linen, and clothing removed from the patient during an attack, should be put at once into a solution composed of zinc sulphate, four ounces, common salt, two ounces, and water, one gallon, and afterward boiled at least two hours in the same solution. Instead of handkerchiefs, pieces of old muslin, surgeon's gauze, or absorbent cotton, should be used for cleansing the nose and mouth of the patient and burned immediately.

The physician in attendance upon a case should leave his coat and overcoat in an anteroom, and put on a long gown or rubber coat, buttoning tightly at the neck and sufficiently large to cover all his clothing. This should always be worn in the sick-room, and boiled or disinfected when the case is finished. The physician's visit should not be unduly prolonged, and a stethoscope should be used for examining the chest. For a single visit the overcoat may be worn in the room, but the clothing should be changed before visits to other children are made. After every visit the physician's hands and face should be thoroughly washed with soap and then with a disinfectant solution.

A physician in attendance upon scarlatinal patients should not attend obstetric cases or other patients with recent wounds. The great liability of such cases to contract scarlatina should never be forgotten. If, in emergencies, it becomes necessary to attend such patients, the physician should change all his clothing and disinfect his hands, face, hair, and beard, with the greatest thoroughness.

Schools are the hot-beds for the spread of scarlet fever. The greatest sources of danger are the mild or walking cases in which the disease has not been recognised, and the clothing of patients who have had a severe form of the disease. As a rule, a child should be kept from school six weeks from the beginning of the attack, and the certificate of a physician

should be required before re-admission, stating not only that the desquamation is complete, but also that the child is suffering from no sequelæ. Other children in the household should not be allowed to attend schools of any kind during the period of active symptoms; they should be kept at home on the average for a month. This precaution is necessary, first, because they might carry the disease from the child at home; secondly, because otherwise they might themselves attend school while suffering from the disease in a very mild form or during the period of invasion. Where the sick child is completely isolated, the danger from the first source is very slight. During severe epidemics it frequently becomes necessary to close all schools.

During desquamation the spread of the disease may be in a measure prevented by the free use of inunctions and warm baths. The bath water should always be disinfected. All the excreta from the patient should be disinfected throughout the disease, best by a carbolic solution (1 to 20). If cases of scarlet fever are to be transported, this should be done only in a vehicle which can be easily disinfected. Under all circumstances as few persons as possible should come in contact with the patient.

In general, it is to be remembered that the danger is first from the patient, secondly from the room, and thirdly from the nurse. Special attention should always be given to the complete and immediate isolation of the first case which appears in an institution or community, which should apply to mild as well as the severe forms of the disease.

Treatment.—There is as yet no specific for scarlet fever, so that the treatment is one of symptoms and complications. Mild attacks require no medicine whatever. Children should be kept in bed for at least a week after the fever has subsided, and upon fluid diet for a period of three weeks. This is an important matter in the prevention of nephritis (page 618). During the height of the eruption, the intense itching of the skin may be allayed by sponging with a weak carbolic-acid solution, or by inunctions with vaseline, or by the free use of rice powder. Plenty of fresh air should always be secured in the sick-room. As soon as the fever and rash have disappeared, daily warm baths with soap and water should be used, after which the entire body should be anointed with carbolized vaseline or a one-per-cent ichthyol ointment, or boric acid and vaseline, five per cent strength, with the two-fold purpose of facilitating desquamation and disinfecting the scales. In case the skin becomes irritated by this treatment, bran baths may be substituted for soap and water. The diet requires careful attention in all cases. With the exception mentioned above, it should be regulated as in other forms of severe illness (page 191).

The temperature does not usually require interference when it only occasionally rises to 104° or 104·5° F. But if there is hyperpyrexia, or a temperature which ranges from 103° to 105° F. or over, antipyretic measures

are called for. Cold is much safer and more certain than drugs. Sometimes cold sponging is sufficient, but in the great proportion of cases the cold pack or the cold bath (pages 47, 48) is required. The pack is almost as efficient as the bath, and usually meets with less opposition on the part of the parents. The use of cold in the reduction of temperature is especially indicated in septic cases with typhoid symptoms, and in those with pronounced cerebral symptoms. Where these are severe the bath should always be used, and repeated with sufficient frequency to keep the temperature below 103° F.

The nervous symptoms are frequently better controlled by ice to the head and by cold sponging than by medication. Antipyretic drugs may be relied upon to control restlessness and promote sleep, and in mild cases to effect a moderate reduction in temperature when this is accompanied by great discomfort. Phenacetine is usually to be preferred. For the nervous symptoms occurring in nephritis, as stated elsewhere, opium is to be used.

As soon as the pulse becomes weak or rapid and irregular, with a feeble first sound of the heart, stimulants should be given, no matter at what stage of the disease. In mild or moderately severe cases they are not generally required. In septic, or malignant cases, or in those accompanied by severe angina, adenitis, or cellulitis, alcoholic stimulants must be used fearlessly—carried even to the full toleration of the patient (page 49). Digitalis is next in value to alcohol, and is especially indicated where the pulse is weak and soft, with a low tension. The fluid extract may be given to a child five years old in minim doses, four times a day in the beginning, and later, if necessary, with greater frequency. Strychnine is also useful, and may be combined with digitalis or given separately, the usual initial dose being gr. $\frac{1}{200}$ to a child of five years.

The erythematous sore throat requires no treatment except the use of a mild antiseptic gargle. If there is profuse nasal discharge, nasal syringing (page 56) with a warm saline or boric-acid solution may be used with the hope of preventing infection of the middle ear. The local treatment of the membranous angina is the same as that of other cases of pseudo-diphtheria. Gangrenous inflammation of the tonsils or palate is sometimes benefited by injections of a 10-per-cent solution of carbolic acid in glycerin, but most such cases prove fatal, no matter what the treatment.

Milder forms of adenitis require no local treatment. When severe, an ice-bag should be applied in the case of older children. If this is not well borne, for young children a hot poultice may be used for a short time for the relief of pain. Prolonged poulticing, however, almost invariably does more harm than good, and favours suppuration. If abscess forms, early incision should be practised.

It is doubtful if otitis can be prevented by any form of local treatment. My experience has been that it rarely occurs in cases with mild

throat symptoms, but that where these are severe it almost invariably follows, whatever the treatment employed. The indications, however, are to keep the rhino-pharynx as clean as possible by syringing the mouth and nose. The indications for paracentesis of the drum membrane are the same as in other severe forms of otitis (page 884). The treatment of scarlatinal nephritis has been considered in the chapter devoted to Diseases of the Kidney (page 618). Diffuse cellulitis of the neck calls for free incisions early as the only means of preventing extensive sloughing.

During convalescence, tonics, particularly iron and digitalis, are called for. The urine should be frequently examined for a long time; antiseptic gargles and a nasal spray or syringe should be used as long as a purulent discharge from the nose or pharynx continues.

CHAPTER II.

MEASLES.

Synonyms: Rubeola, Morbilli.

Measles is an epidemic contagious disease, more widely prevalent than any other eruptive fever; very few persons reach adult life without contracting it. One attack usually confers immunity. It is highly contagious even from the beginning of the invasion, and spreads with great rapidity from the patient to all susceptible persons exposed. The poison, however, does not cling so long to clothing or apartments as does that of scarlet fever. Measles has a period of incubation of from eleven to fourteen days; a gradual invasion of three or four days with symptoms of an acute coryza; a maculo-papular eruption which appears first upon the face and spreads slowly over the body, and which lasts from four to six days. This is followed by a fine bran-like desquamation, which is completed in about a week. The mortality is low, except among infants and delicate children, where it may reach 30 or even 40 per cent. In institutions for infants and young children no disease is more to be dreaded than measles, not only on account of its severity, but the frequency with which, in such subjects, it is complicated by broncho-pneumonia.

Etiology.—The essential cause of measles is as yet unknown. It is generally believed to be due to a micro-organism, but, as in the case of scarlatina, all attempts to isolate it have thus far been unsuccessful. The poison is one which possesses remarkable powers of diffusion, but whose viability is much less than that of most of the pathogenic germs which are known. Only a short exposure is required to communicate the disease, and even close proximity to a patient does not seem necessary. One instance has come under my own observation where measles was appar-