PARALYSIS, ARSENICAL.—Paralysis due to arsenical poisoning is not very rare. The larger number of cases have the appearance, and are, doubtless, mere instances of multiple neuritis. Most text-books do not allude to arsenical paralysis otherwise than by simply mentioning arsenic as one of the causes of multiple neuritis.

Etiology.—Usually the paralysis results from acute arsenical poisoning, the poison being taken either designedly or by accident. The paralysis corresponds in some measure to the intensity of the general poisoning. Not infrequently the paralysis follows the repeated administration of the drug. It has been observed after the ordinary medicinal doses: for instance, after taking from three to ten drops of Fowler's solution three times a day for a number of weeks. In these instances the paralysis is usually of a mild grade. A few years ago there was quite an epidemic of arsenical paralysis in some British towns, Liverpool, Manchester, and other places, which occurred in drinkers of beer, the examination of which revealed the presence of arsenic. In these instances the alcohol may also have played a part in producing the paralysis, but the sufferers were mostly moderate drinkers, and the pain attending the paralysis was more severe than that usually found with alcoholic neuritis.

Paralysis occurs, but less frequently, from the external use of arsenic (salves, baths, etc.), and from contact with fabrics (wall paper, carpets, and artificial flowers) which contain arsenic. Barton reports two cases, in husband and wife, with paralysis following acute poisoning, the man having for some years been rubbing a mixture of four parts of arsenic and three parts of plaster-of-Paris into the skins of animals and birds, while his wife cleaned the room twice a week. Similar cases, from dealing in stuffed birds, working with arsenical draperies, or living in rooms where there were draperies or wall paper containing arsenic, have been reported.

Symptomatology.—In cases of acute poisoning the paralytic manifestations appear shortly after the disappearance of the severe gastro-intestinal disturbance. Paralysis of twenty-four hours' duration immediately after the ingestion of the arsenic has been spoken of, but must be rare or must escape detection. The onset of the paralysis occurs usually from several days to several weeks after the poison has been taken. It is commonly preceded by sensory symptoms, tingling, numbness, intense pains, etc. The paralysis itself begins more or less gradually. Not uncommonly its onset is not observed, but when the acute symptoms have subsided and the patient attempts to get out of bed, it is found that he cannot walk or cannot hold anything in his hands. Occasionally the paralysis is complete, or at least no longer progressive, at an early period, but usually some weeks elapse before it reaches its greatest intensity. Almost always, when observed from the beginning, it has been found to commence at the distal ends of the extremities, the fingers and toes, usually first in the lower extremities, and thence extend upward. The parts below the knees and elbows are most profoundly paralyzed. In bad cases the muscles of the thigh are also paralyzed, and not infrequently the trunk is more or less paretic. The extensor muscles—the radial group in the upper and the anterior tibial group in the lower extremities—are, as a rule, most profoundly affected. When the paralysis is complete the paralyzed muscles are quite flaccid, and we find wrist-drop, foot-drop, etc. At a later period there are very frequently contractures, more or less strong, affecting particularly the least paralyzed muscles. Tremor, often fibrillary in character, is not infrequently observed in the affected muscles. The paralysis just described, affecting the four extremities, the lower more than the upper, is that usually found. In rare instances it is otherwise distributed; sometimes in hemiplegic, and more rarely in monoplegic form. In almost all instances we find great loss of flesh and a general wasting, in addition to pronounced atrophy of the paralyzed muscles. The electrical reactions are those of peripheral paralysis. The reaction of degeneration becomes more or less pronounced a few days, or a few weeks, after the occurrence of the paralysis. At a later period, if the paralysis be profound, all electrical reactions may cease. In some instances slight changes in the electrical reactions may be observed before there is any manifest paralysis, particularly in so far that it requires a stronger faradic current than usual to produce muscular contractions. In lighter cases such electrical responses, especially in the anterior tibial group of muscles, may be the only indication of incipient paralysis.

The sensory symptoms are at times even more pronounced than the motor paralysis. These symptoms are paræsthesiæ of various kinds, tingling, numbness, coldness, etc.; pains, tenderness, and anæsthesiæ. As a rule the tingling and numbness, and very frequently the pains, precede the paralysis. The pains are often intense and constitute the most distressing symptom of the disease. They are usually described as burning, cutting, boring, etc., and are often accompanied by sudden starts, cramps, or spasmodic movements of many muscles of the body. They are usually more or less paroxysmal, and are likely to be worse at night and to keep the patient awake. They occur most frequently in the feet and hands, sometimes appear to be in the joints or bones, and occasionally are in the course of the nerves. Tenderness of the affected muscles is also a common and prominent symptom. The tenderness may be so great as to make the handling of the patient extremely painful. In some instances the tenderness is observed to be in the course of the nerves, but generally it is so diffused that it is hard to state that it is especially over the nerve tracts. Often hyperæsthesia or hyperalgesia is observed, although this may be only an expression of the great tenderness. Loss of or impaired sensation, anæsthesia, is also a common symptom. It is found most frequently in the feet and hands, and especially in the finger tips and toes or soles of the feet, although it corresponds somewhat to the extent of the motor paralysis. Rarely it occurs only in the distribution of certain nerves. Anæsthesia is found in all bad cases of paralysis, and not infrequently in mild cases. Loss of tactile sensation is most common; the loss of the senses of temperature and of pain is also common; and muscular sense is not infrequently impaired. The knee-jerks are usually abolished. This is often true even when the paralysis is slight. Vaso-motor and trophic symptoms, such as œdema, particularly in the feet, profuse perspiration, loss of nails and hair, pigmentation of the skin, herpes, and muscular atrophy, are common.

On the other hand, the cranial nerves and bladder are very rarely affected. In some instances the pulse is more rapid than seems consistent with the patient's general condition; a fact which may be due to an affection of the vagus or of the cardiac ganglia.

As has already been stated, usually several days or several weeks elapse before the disease reaches its acme. Then after an interval, which may be of only a few days' duration, but which sometimes runs into months, the patient begins to improve. The anæsthesia begins to disappear before the motor paralysis. It disappears in the reverse order of its appearance: first from the trunk and the upper part of the extremities, remaining longest in the fingers and toes. The motor paralysis disappears in the same manner: first in the upper part of the extremities, then in the muscles below the knees and elbows. The flexors usually improve more rapidly than the extensors, until finally the only motor symptoms which remain, in the mild cases, are paralyses of the extensors of the feet and toes, and of the extensors and small muscles of the hand. At a late period there are liable to be contractures in the still paralyzed parts. The pains also become modified in intensity after the lapse of some time, but unfortunately they are likely to persist with a greater or less degree of severity throughout the whole course of the disease. The vaso-motor and trophic symptoms (œdema, pigmentation of the skin, etc.), usually disappear at a comparatively early period. The whole duration of the disease is extremely variable. Alexander puts it as from eight days to a number of years. It may be stated in general that mild cases usually get well within

six months, possibly in an even shorter time, whereas in severe cases one or two years elapse before there is complete recovery, or, in those cases in which a cure remains incomplete, before there is a definite cessation of improvement.

In some cases of arsenical paralysis muscular incoordination, ataxia, is a more prominent symptom than paralysis, and to this group of cases the term pseudotabes has been applied. The pains, anæsthesia, absence of knee-jerks, together with the ataxia, make that term seem very appropriate.

The disease so far described is that following acute arsenical poisoning. In some cases following chronic poisoning the symptoms are much the same, only the onset is likely to be less sudden and the symptoms less severe. In other chronic cases the symptoms are very slow in appearing, and may have been in part masked by those of gastric irritation. The pains are likely to be proportionately greater, and the paralysis slight in degree. But, even when the paralysis is slight, the changes in the electrical reactions are likely to be found. In such instances the motor manifestations may be rather like those of ataxia than of paralysis. Sometimes a sense of profound prostration is the only indication of motor impairment.

There is another class of cases in which the poisoning appears to have been exceedingly slow and insidious. The symptoms in these cases are usually very obscure, and the cause is commonly to be found in arsenical wall papers or the like. Among the symptoms are, gastro-intestinal irritation, neuralgia, headache, insomnia, general prostration, mental depression, impairment of memory and mental endurance, and epileptiform convulsions. As this article is devoted to the subject of arsenical paralysis no further attention will be given to this class of cases.

Pathology.—In the larger number of cases examined only neuritis was found. In a few instances disease has been found in the central nervous system, in the spinal ganglia, in the cord, and even in the brain. Experiments have been made on various animals, but the results are not all in accord. In some instances neuritis only, in others, inflammation in the spinal ganglia, in the anterior cornua of the cord, and in other parts of the central nervous system was found. The probability is that the peripheral neuritis and the affection of the central nervous system, when present, occur at the same time, but that neuritis is the more frequent and of a higher grade of intensity. The clinical picture indicates that the characteristic condition in most instances is multiple neuritis, and if further pathological changes are present they do not contribute to the symptoms presented.

In those rare instances of incontinence of urine, etc., and those with profound and lasting paralysis, the symptoms are probably due to central disease.

Diagnosis.—When it is known that the patient has been poisoned with arsenic, as is usual in acute cases, the cause of the resulting paralysis is apparent. When there is no such knowledge, the history of severe acute gastro-intestinal disturbance preceding the kind of paralysis just depicted should be almost enough to make the diagnosis certain. The picture of the paralysis is fairly characteristic. The four extremities are usually affected, the lower ones being more often and more extensively involved than the upper ones. The paralysis is always greatest in the digital extremities and is attended by atrophy and altered electrical reactions. In addition there are the intense pain and extreme tenderness, and the absence of knee-jerks, of bladder symptoms, and of bed sores.

When the paralysis is less marked, and the whole array of symptoms less conclusive, the greater prominence of the sensory symptoms and the presence of altered electrical reactions in the slightly paralyzed muscles (in this instance it requires a very careful examination to reveal such reactions) may arouse a suspicion of the true condition.

The cases with ataxic gait may suggest locomotor ataxia as the correct diagnosis, and Seeligmueller mentions a case of his own which was falsely looked upon as one of locomotor ataxia. The severe pains and lost knee jerks heighten the resemblances of the two diseases. But with careful examination such errors will rarely occur, for it will usually be found that the difficult gait is rather paretic than ataxic; and, furthermore, symptoms on the part of the bladder and the eyes (such common and early manifestations in locomotor ataxia) are absent.

In cases of chronic poisoning the detection of arsenic in the urine may assist materially in the diagnosis. In acute cases the arsenic has usually been eliminated before or soon after the paralysis appeared.

Prognosis.—Mild cases may get well in a few months, though rarely in less than six; but when there has been profound paralysis, recovery cannot be expected in less than one or two years.

In not a small proportion, possibly in one-fifth, of the cases of extensive paralysis, complete recovery does not take place.

Treatment.—The treatment is that of multiple neuritis. *Philip Zenner.*

PARALYSIS, DIPHTHERITIC. — This is paralysis which occurs during or after diphtheria, and is due to changes in the nerves or muscle fibres.

The statistical frequency of diphtheritic paralysis has varied greatly according to different writers. The American Pediatric Society's collective investigation[7] showed that it occurred in 9.7 per cent. of all cases of diphtheria. This is rather low, some figures running as high as twenty-five per cent. or even higher. It is probable that at least ten per cent. of all cases of diphtheria in private practice, properly treated, may be expected to show this complication.

Etiology.—That the pathological lesions upon which the symptoms of diphtheritic paralysis depend are due to the action of the poisons of the disease on certain of the tissues there can be no question, since both the lesions and the symptoms have been produced experimentally in animals by the injection of the toxins.[1,2] Paralytic symptoms may follow diphtheria of all degrees of severity, and instances in which it has complicated cases so mild that the causative sore throat has been unnoticed or forgotten by the patient are sometimes seen in large clinics. As to whether it is more common after the severe cases than after the mild ones there is a difference of opinion. Gowers[3] and Henoch[13] believe that its frequency does not depend upon the severity of the original disease. Goodall[8] and De Gassicourt believe that it does. The former reports a series of cases which seem to show that its frequency is greatest in the cases showing the most extensive membrane.

The influence of the antitoxin treatment upon the frequency of the paralysis is also a matter of doubt. Certain statistics have shown that the use of this remedy has been followed by an increased number of cases of paralysis; but such studies are misleading, since under the use of this method there are many severe cases of diphtheria which recover and have paralysis,—cases that would otherwise die before the time at which the palsy generally begins. It is said that the condition is comparatively rare among cases in which the larynx is the part mainly affected by the diphtheria. Males appear to be slightly more susceptible than females. As to age, Gowers[3] agrees with Landouzy that adults are much more frequently affected. Goodall[8] and Ross[14] find that it is relatively more common in children.

Pathology.—Lesions are found in the central nervous system, in the peripheral nerves, and in the muscles, but there is no doubt that the dominant lesion—the one upon which the clinical picture, at least so far as the peripheral palsy is concerned, mainly depends—is degeneration of the lower motor neurones, *i.e.*, peripheral neuritis. According to J. J. Thomas and others who have made careful studies of the nervous system, using modern histological methods, the process is one of fatty degeneration, which begins in the myelin sheaths. The axones afterward become beaded, break up and disappear. Both

motor and sensory nerves are affected. These changes would appear to be much more frequent than the palsy, since they are very generally found at autopsy, even though no paralysis has been noted before death. In all of Thomas' cases the vagus nerve showed more or less marked degeneration. Others have observed the same thing, as well as similar changes in the cardiac plexus, the fifth cranial nerve, the nerves supplying the larynx, and elsewhere.

The effect of the toxic substances is not confined to the peripheral nerves, as there is a diffuse parenchymatous degeneration of the nerve fibres of the spinal cord and brain. Several observers, notably Bikeles, have found that these changes were most marked in the posterior columns and in the posterior nerve roots, a fact of interest in connection with those cases which show ataxia as a symptom. As to the effects on the nerve cells opinions differ, some authorities even considering that the characteristic symptoms are due to primary degeneration of the motor nerve cells in the anterior horns, rather than to changes which are primary in the peripheral nerves. The weight of evidence is strongly against this view, however, and the changes in the nerve cells are probably of relatively slight importance and degree. The brain and cord often show hyperæmia, and in rare cases myelitis or hemorrhages, the latter sometimes being severe. But the symptoms in most cases depend on changes in the peripheral nerves rather than in the central nervous system.

Of the muscles, the one in which the changes are of the greatest clinical significance and in which they have been most carefully studied is the heart. According to Councilman, Mallory, and Pearce[6] degeneration in the myocardium is one of the most common conditions found in diphtheria. The simplest form is fatty degeneration, which is found in the majority of all cases, and which seems to precede the more advanced forms of degeneration which lead to complete destruction of the muscle fibres. In these there is destruction of the sarcous elements, which become swollen, broken up, and converted into hyaline masses. The degeneration may become so extensive as to account fully for the impairment of the heart's action. Acute interstitial lesions are also found, and occasionally cardiac thromboses and less important vascular lesions.

Changes are also observed in the skeletal muscles. It is probable that, as Baginsky[5] believes, the paralysis of the palatal muscles represents a myositis due to the direct effects of the membrane in their close proximity, rather than a neuritis. But we must also remember that the vagus nerve, which supplies these muscles, is almost always involved. Councilman, Mallory, and Pearce[6] observed marked fatty degeneration of the tongue, diaphragm, and various muscles of the extremities, and they conclude: "It seems probable that in all cases where fatty degeneration of the heart and nervous system has occurred, a similar change will be found in the skeletal muscles."

The changes produced in the nervous system by diphtheria are thus summed up by Thomas:[4] "(1) A marked parenchymatous degeneration of the peripheral nerves, sometimes accompanied by an interstitial process, and hyperæmia and hemorrhages. (2) Acute, diffuse, parenchymatous degenerations of the nerve fibres of the cord and brain. (3) No changes, or but slight ones, in the nerve cells. (4) Acute parenchymatous and interstitial changes in the muscles, especially the heart muscle. (5) Occasional hyperæmia, or infiltration, or hemorrhage in the brain or cord; in rare cases severe enough to produce permanent troubles, such as the cases of multiple sclerosis and of hemiplegia which have been observed. Finally, the probability that the cases of sudden death from heart failure in diphtheria during the disease or convalescence are due to the effects of the toxic substances produced in the disease upon the nerve structures of the heart." The changes produced in the nervous system, then, involve chiefly the lower (spino-muscular) segment of the motor path.

The SYMPTOMS in the ordinary peripheral form of paralysis may begin as early as the fourth day, while the patient is still ill and while the membrane is still present, or they may be delayed for many weeks. The usual time is from one to three weeks after the disappearance of the membrane.

The distribution of the paralysis may be judged from the following figures of the American Pediatric Society's[7] 189 collected cases: Throat, 124; extremities, 22; eyes, 11; respiratory muscles, 5; heart, 32; neck, 1; general, 8. Ross[14] collects 171 cases, distributed as follows: Palate, 128; eyes, 77, of which 54 involved the muscles of accommodation; legs, 113; arms, 60; trunk or neck, 58; respiratory muscles, 33. In Goodall's[8] 125 cases the palatal muscles were involved 102 times, the ciliary muscles 56 times, the legs 52 times, the external ocular muscles 26 times, the arms 21 times, and the pharyngeal muscles 11 times. Obviously the throat is the region most often affected and the eyes and extremities next. When the extremities are involved the disease is characteristically more common in the legs than in the arms, which, in severe cases, are involved later.

The symptoms begin gradually, and usually a number of different parts are involved successively, improvement taking place in one while the disease advances in another. It most commonly begins in the throat, and the palate may be the only part affected. As a result of the involvement of the palate the nasal cavity is not shut off from the nasopharynx in swallowing or in speaking. The child is observed to regurgitate its food, a symptom which may become so severe that deglutition is impossible. The voice becomes nasal in character. Extension to other muscles of the throat and mouth may lead to inability to blow, whistle, suck, or gargle. The muscles are bilaterally or rarely unilaterally involved. Paralysis of the constrictors of the œsophagus is evidenced by the entrance of food into the glottis. Cough, hoarseness, aphonia, and paroxysms of dyspnœa are seen when the larynx is involved. There may be difficulty in articulation or in protruding the tongue. Facial paralysis is rare, as is also paralysis of the tongue or œsophagus.

When the eyes are involved dimness of vision for near objects is noted, and is due to loss of the power of accommodation from paralysis of the ciliary muscles. The patient usually first complains of dimness of vision. The failure of accommodation reaches its height in a few days and generally lasts two or three weeks. The pupil may be dilated. Ophthalmoplegia externa—squint or double vision—and ptosis are rarer. Sometimes most or even all of the muscles of the eyes become paralyzed. Involvement of the hearing, taste, and smell are recorded, but are rare.

When the extremities are involved there is gradual loss of power, beginning almost always in the legs, afterward extending to the arms in severe cases or even to the trunk and neck, so that in the worst cases the patient becomes a helpless mass. But such cases are rare, and the loss of power is seldom absolute. The distribution is usually symmetrical. In contrast to the alcoholic form of neuritis subjective sensory symptoms are ordinarily slight. There may be numbness, tingling, hyperæsthesia, or anæsthesia; but in most cases these symptoms cause little distress. According to Gowers[3] anæsthesia is always most severe toward the extremities of the limbs, and he mentions cases in which sensation was lost only in the finger-tips. As in all forms of degeneration of the lower motor neurone, the muscles become flabby and atrophy occurs. The knee-jerks are commonly lost, and lost reflexes are sometimes the only evidence that the nerves of the extremities are involved. It is said that in some cases they are retained. During convalescence there may be increased knee-jerks and ankle clonus.

The electrical reactions are variable; according to Northrup so much so that they are of little value in diagnosis or prognosis. As a rule they are altered, the muscles showing decreased reaction to faradism with the reaction of degeneration, and the nerve trunks showing

decreased irritability to both currents.[3] The bladder and rectum are rarely involved.

Reference has already been made to the occurrence of ataxic symptoms in diphtheritic paralysis, and to the view of Bikeles, which explains them as due to degeneration of the posterior columns of the cord. These symptoms are clumsiness in the use of the limbs and unsteadiness in walking. The gait is seldom so markedly ataxic as in true tabes. The Romberg symptom is prominent. Lack of knee-jerks and sluggish pupillary reactions complete the picture, which, however, is often masked by the muscular weakness.

One of the most serious complications of diphtheritic paralysis is extension to the muscles of respiration. It occurred in 33 of Ross' 171 cases,[14] and 21 times in 275 cases reported by Meyers.[17] Either the diaphragm or the intercostals may be involved. The average time of onset in Meyer's cases was the thirty-seventh day, but it occurred as early as the eleventh and as late as the fiftieth. Dyspnœa is a marked symptom, and is seen in the form of asthma-like paroxysms or in spasmodic attacks due to the accumulation of mucus. There are anxiety and mental distress with a sense of impending suffocation. When the diaphragm is involved abdominal breathing is reversed, the abdomen sinking in on inspiration, and *vice versa*.

The symptoms of cardiac paralysis occurring in diphtheria are of the greatest importance, owing to their frequency, seriousness, and bearing upon questions of treatment. Their exact cause is a matter of doubt. Changes are found at autopsy both in the myocardium and in the nerves controlling the heart. As has already been said, the myocardium may show degeneration sufficient to account in full for all the symptoms. Some, however, believe that the nerve changes are primary. Thomas and Hibbard,[10] in an exhaustive investigation of the subject of heart failure in diphtheria, favor Hesse's view,[18] that heart failure results directly from the effects of the poisons of the disease rather than from the degenerative processes which they cause in the tissues; but they consider that these toxins act through the nervous mechanism. It seems probable that the origin of the heart symptoms may be due either to changes in the myocardium or to changes in the nerves controlling the heart, or to the effects of toxins on the nerve centres. In any given case the exact cause of heart failure can be stated only after death, but it seems fair to suppose that those due to the direct effect of the diphtheritic poisons will occur early in the disease, just as they do in other acute conditions, while those due to organic degenerations may occur later.

Cardiac symptoms are observed in diphtheria at a relatively early date as compared with the other palsies; according to Woodhead,[15] mostly between the fifth and tenth days. The average in Meyer's cases[17] was the seventh. But sometimes they arise late in convalescence. Occasionally they are overlooked, and sudden death after slight over-exertion takes place in a case apparently well. As a rule, some abnormal condition of the pulse first calls attention to the heart. It is unusually rapid or unusually slow, or oftener it is irregular or intermittent. The heart, when mapped out by percussion, generally shows some enlargement. Systolic murmurs, most of which are temporary, may be heard. Pallor, cold extremities, dyspnœa, and cardiac distress are noted. Vomiting is an important symptom, whose occurrence, unless otherwise explained, in diphtheria should always call attention to the heart. Its association with cardiac failure is supposed to depend upon the common relationship of the vagus nerve to both the heart and the stomach. Death may occur within twenty-four hours after the onset of cardiac symptoms, or, as has already been said, it may take place suddenly without warning. In most of the heart cases there is evidence of paralysis elsewhere.

Brodie,[19] as a result of animal experimentation, and Biernacki[20] from clinical observation, consider that the fall of blood-pressure occurring in diphtheria is a result of paralysis of the muscular walls of the vessels. It is a question, however, whether the action is local or central, and to what extent impairment of the heart's action is responsible for the phenomena noted.

There are other forms of paralysis which are sometimes associated with diphtheria, but they occur more rarely and they need but brief mention. One is sudden cerebral hemiplegia, due generally to embolism, more rarely to hemorrhage or thrombosis. Meningitis is sometimes a sequel of diphtheria, and cases of multiple sclerosis have been reported by Schönfeldt. The paralytic symptoms which they cause could hardly be mistaken for the common form.

DIAGNOSIS.—This depends on the recognition, during an attack of diphtheria or in convalescence from the same, of a peripheral neuritis or, in some cases, of a myositis. The most significant peculiarities of the diphtheritic form of neuritis are the frequency of involvement of the throat and eyes, the process often beginning in the former, the symmetrical distribution, the tendency to involve the legs before the arms, the slightness of sensory symptoms, and the frequency of cardiac manifestations.

In making the differential diagnosis other forms of peripheral neuritis must be excluded, especially in cases in which the causative diphtheritic infection has escaped recognition, in which more than one cause is present, or in which the palate and eye symptoms have been slight or lacking. The chief forms of neuritis to consider are those due to alcohol and to lead. Alcoholic neuritis is rare in children, does not involve the throat, and has more prominent sensory symptoms. Other evidences of alcohol are usually present. Lead palsy begins generally in the arms, involving the extensors and giving the characteristic wrist-drop. It is usually associated with other evidences of lead poisoning, such as the blue line on the gums, the cachexia, colic, traces of lead in the urine, and granular degeneration of the red corpuscles of the blood. Other forms of neuritis may be ruled out on similar lines. Mixed forms occur, but in such cases, apart from questions of prophylaxis, the cause is rather of academic than of practical importance. Cases of diphtheritic paralysis showing marked ataxia may be mistaken for tabes dorsalis. The post-diphtheritic disease, however, develops more rapidly; true lightning-like pains are not experienced; and the loss of motor power with atrophied and flabby muscles is absent in true tabes. Acute poliomyelitis is also of importance. Here the onset is more acute, paralysis is not symmetrical, and the sensory symptoms are lacking. Hysterical palsy seldom involves the palate, as diphtheritic paralysis almost always does. In hysteria the knee-jerk is retained. The greatest difficulty arises in cases in which a true diphtheritic paralysis is combined with hysteria.

PROGNOSIS.—This will depend upon the site of the paralysis, the severity of the case, and the previous condition of the patient. Of Goodall's 125 patients 17 died: 4 in the acute stage of diphtheria; 6 fatal cases were cardiac, 4 respiratory, 2 vomiting and cardiac, 1 convulsions.[8] Of Ross' 171 patients 45 died: 8 from intercurrent diseases, 8 from sudden syncope, 10 from heart failure, 14 from respiratory paralysis, and 2 from the aspiration of food into the trachea.[14] According to Gowers,[3] the sooner the paralysis begins after the diphtheria the greater the danger to life.

Paralysis of the extremities of itself causes little danger to life, and recovery may be predicted, even though it may be months before power is completely restored. The duration is often from six to eight weeks. The loss of knee-jerks is generally the last sign to disappear. In the throat cases there are two sources of danger. The first is that arising from the frequency with which palatal involvement is associated with heart symptoms. The second danger arises from the difficulty of deglutition, patients dying from inanition on this account, or from the aspiration of food into the trachea with resulting asphyxia or bronchopneumonia. In respiratory cases the outlook is always more dubious. Meyers[17] reports 21 patients affected with diaphragmatic palsy, of whom 11

died, this being 13.7 per cent. of all deaths from diphtheritic paralysis in his series.

Cardiac paralysis is even more fatal. Statistics are of little value, as different observers vary so greatly in the criteria upon which they base their diagnoses. All 32 cases collected by the American Pediatric Society[7] were fatal. Hibbard[11] reports 47 per cent. of deaths in cases with irregular pulse. Vomiting during convalescence from diphtheria generally means heart failure, is an unfavorable sign, and occurred in over half of Hibbard's fatal cases. According to Burrows[12] it is especially to be feared when it occurs in a patient whose heart is irregular, or who presents other evidences of nerve degeneration. A very slow pulse is also unfavorable, especially in children. The patients may die within twenty-four hours of the onset of cardiac symptoms, or later during an exacerbation. Sudden death from heart failure without previous symptoms may occur in the acute stage, or it may suddenly and unexpectedly terminate a case after convalescence has appeared to be completed.

Treatment.—The varying results of statistical studies as to the effect of antitoxin have already been mentioned in connection with the subject of etiology. It seems probable that the early and vigorous use of antitoxin in any given case will decrease the likelihood that this complication will occur, but only in so far as it lessens the severity and duration of the causative disease. That this view is correct is proven on the experimental side by the work of Ransom,[1] who shows that doses of the toxin capable of producing paralysis in animals are neutralized in this respect by antitoxin injected simultaneously and modified, though not prevented, by large enough doses given from fifteen to twenty-two hours after those of the toxin. On the clinical side the report of the London Clinical Society[21] shows that the frequency of paralysis as a complication of diphtheria is less when antitoxin is used on the first two days of the disease than when its use is delayed. After the injury to the nerves or muscles has been done, it is not probable that antitoxin will have any effect in restoring them to the normal condition. The same conclusions probably hold in regard to the heart manifestations. In other words, antitoxin, given early and in large doses, has some value as a prophylactic in preventing the paralytic complications of diphtheria.

On the peripheral neuritis no form of treatment has much effect. The case should be managed like a neuritis from any other cause. Rest, careful nursing, liberal diet, tonics, massage, and electricity are of some value. Strychnine is much used, but its influence in restoring the degenerated nerves and muscles to their normal condition is at least questionable. In palatal, and especially in pharyngeal and laryngeal cases, great care should be taken in feeding the patient to prevent the entrance of food into the larynx. The œsophageal or nasal tube may be used if needed, great care being taken to avoid introducing it into the larynx and to get the end well below the glottis. In some cases it will be better to feed by the rectum. Forced feeding had better not be delayed in hope that the child will begin to eat, especially in cases in which the patient is much debilitated by a severe attack of diphtheria. Proper and sufficient nourishment is of importance both in the treatment of the general depressed condition and in that of the paralysis.

One precaution should be insisted upon. Every case of diphtheria should be watched closely for the occurrence of cardiac or respiratory symptoms, especially when evidences of palatal paralysis are present. Thomas and Hibbard[10] advise that every case of diphtheria, however mild, should be kept in bed till the throat is clear, and, if there has been any prostration, for at least two weeks more. After four weeks with no heart symptoms there is little danger. If heart symptoms arise, the greatest care must be taken to keep the patient quiet, morphine being used for this purpose if needed. Careful feeding is of importance. Medicines are of value only in meeting special symptoms. Alcohol, digitalis, and strychnine may be of service. In the respiratory cases strychnine is the most valuable remedy. Electricity may also be used. *Ralph C. Larrabee.*

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(For further bibliography consult the articles of Northrup, Ross, and Thomas.)

PARALYSIS, FACIAL. See *Facial Paralysis.*

PARAMUCIN.—Mitjukoff has obtained from the mucoid contents of ovarian cysts a mucin-like substance, which differs from *pseudomucin* and *mucin*, chiefly in the fact that without previous boiling with dilute mineral acids it will reduce an alkaline solution of copper. ("Ueber das Paramucin," *Arch. f. Gyn.*, Bd. 49, 1895.)
Aldred Scott Warthin.

PARAMYOCLONUS MULTIPLEX.—(Synonyms: Myoclonus multiplex, Myoclonia, Polyclonia.) Originally described by Friedreich[1] in 1881, this disorder has been recognized by clinicians in Germany, France, Italy, England, and America. Its existence as a disease has been questioned, some asserting it to be a variety of hysteria, others of chorea. Still others cut the Gordian knot by claiming that both a true form and a hysterical form of the disease exist. However, the preponderance of authority as well as of evidence at the present day appears to favor its validity as a clinical entity.

It is a rare disease. Gowers[2] states that he was able to collect but fifty-two cases in the literature up to July, 1895, of which only thirteen were considered by him true examples.

One of the best accounts of the disease accessible to American readers is that of Starr,[3] which is also accompanied by a bibliography.

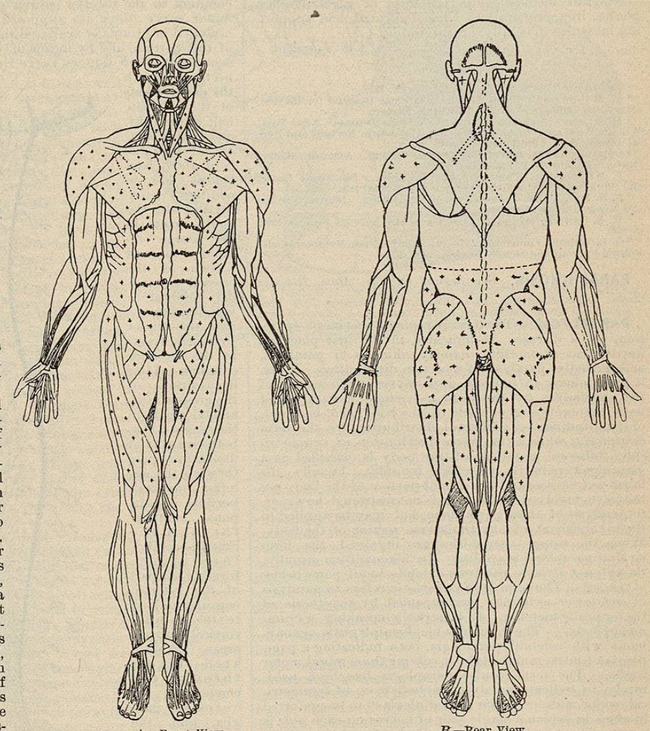
The disease is characterized by paroxysms of clonic muscular contractions, shock-like, bilateral, and symmetrical as regards the two sides of the body. The individual contractions are frequent, varying in rate from thirty to one hundred or more per minute. The duration of the paroxysms may vary between five or ten minutes and some hours. Likewise the frequency of paroxysms in a day may vary from one or two to twenty or more. The muscles affected in typical cases are the intrinsic truncal muscles and those connecting the trunk and extremities; those moving the face, hands, and feet being seldom, if ever, affected (see Fig. 3738). In one case observed by the writer,[5] the diaphragm and laryngeal muscles were involved at times, causing short, sharp, involuntary exclamatory sounds.

Negative characteristics are: consciousness is not affected; mental defects are absent as a rule, but, if present, are transient; voluntary movements of the face, hands, and feet are not abolished, even during a paroxysm, though the muscular power is much reduced; electrical changes of degenerative significance are absent, as is fibrillary twitching. Sensation is not diminished or lost, but a decided hypersensibility to sound and touch has been observed by the writer in one case.[5] A profound sense of fatigue was also noted in that case. The convulsive movements cease during sleep, and are brought on, or, if present, increased in violence, by emotional excitement, by irritation of the skin, by cold, etc., and by manipulative procedures generally.

Oppenheim[4] recognized a hysterical type as distinguished from the true form, but admits the difficulty of separation.

Unverricht, Weiss, Kreiver, and Sepilli (quoted by Oppenheim) describe cases of a familial type and associated with epilepsy (*loc. cit.*).

Causation. — The patients are usually neurasthenic. A majority of cases reported were of the male sex. The ages of patients have varied between thirteen and forty-eight. Mental worry, fright, injury, and physical strain are accredited causes. Of two cases reported by the writer one was attributable to mental worry combined with la grippe. The other was distinctly due to fright. A third case, observed by the writer through the kindness of Sir William Gowers, followed a fall from a considerable height without palpable injury. Fry's case[6] was due to overexertion, Starr's[3] to strain in lifting. Removal of the thyroid in dogs is said sometimes to cause symptoms of this disease.



A.—Front View. *B.*—Rear View.

Fig. 3738.—*A* and *B*, Paramyoclonus Multiplex. Distribution of myoclonic spasm indicated by plus marks.

The pathology is unknown. Autopsy in one case (Schultze) revealed no nervous changes. Friedreich, who was the first to describe the disease, believed it to be based on overexcitability of the spinal motor elements. Some have surmised that the cause of the faulty action lies in the muscles themselves or in an abnormal state of the nerve endings. To the writer its psychic antecedents, marked hemiplegic preponderance at times (in one case); its aggravation by mental and emotional states; the marked, though transient, mental changes in two cases; and the heightened muscle reflexes, would all suggest that the disease is to be viewed as the visible expression of a state of "*inhibitory insufficiency*," probably cortical in seat.

Diagnosis.—This is to be based on "the sudden shock-like character of the muscular contractions, their bilateral symmetry, and the comparative freedom of the extremities." (Gowers.[1]) This view is also concurred in by Walton[8] in a recent paper on the myospasms in general. Hysteria is ruled out by the absence of the stigmata and of the characteristic emotional state. There appears to be no tendency to simulation or desire for sympathy in paramyoclonus. Chorea is excluded by the non-involvement of the face and hands. Dubini's "electrical chorea," a disease endemic in a certain locality in Italy, is to be separated by its unilateral beginning, nerve and muscle degeneration, and a fatal termination in a few months.

The prognosis is variable, according to different authors. Friedreich reported that some of his patients recovered. Oppenheim considers the prognosis grave. In all the American cases reported to date the patients have recovered. Relapse may occur, but does not preclude ultimate recovery.

The duration may vary from three or four months to a year or more. In one of the writer's cases the convulsions ceased on the one hundred and second day, but recurred in twenty-four hours, to disappear again in eight days. There has been no recurrence to date (four months). In the case reported by Starr (*loc. cit.*) the patient recovered in about a year, as did also the patient in the first case reported by the writer.[5]

Treatment.—The drug treatment followed has been