from local nerve-lesions. From myelitis the distinction is made by the stage of excitation affecting sensibility and motility, and the stage of depression also affecting sensibility and motility and the bladder. From hæmorrhage, the distinction is made first on account of its rarity, next the suddenness of the attack, sensibility being destroyed as well as motility, usually, and the sphincters paralyzed. From progressive muscular atrophy, the distinction is made by the age of the subject, the slow development, and the affection of isolated muscular groups in turn. From cerebral lesions, the distinction is made by the pronounced cerebral symptoms, by the hemiplegia, by the electrical reaction, the electro-contractility rather heightened than lost, and by the appearance and condition of the paralyzed members. From paralyses due to local injury of nerve, the distinction is made by the history of the case, the evidence of injury, by the absence of fever, by the diffusion of the paralysis at first, followed by localization.

Treatment.—During the attack of fever with which the disease begins, only symptomatic treatment is proper, since a diagnosis is not possible. When paralysis has occurred the damage to the cord is complete, but, as the functional disturbance is more extensive than the symptomatic expression of the real lesions, the improvement which follows from the first paralysis is simply the disappearance of the merely functional troubles. Any active treatment, therefore, instituted with a view of combating an inflammation, is improperly applied. The problem is to prevent further destruction of the gray matter, and to restore damaged but still functionally capable tissue. The remedies best adapted to accomplish this, and which in the author's hands have acted best, are quinine and belladonna (from a fourth to four grains, according to age, of quinine, and from $\frac{1}{20}$ to $\frac{1}{4}$ grain of belladonna extract); hot douche to the spine and tepid wet packs; the application of galvanism, inverse current, stabile, large volume and low intensity, and rest, as nearly absolute as possible, until the period of restoration. When the period of improvement comes on, the muscles must be faradized, if they react to the faradic current, or galvanized if they react only to the galvanic current. Massage is suitably combined with electrical treatment. The wasted muscles are much improved by aquapuncture; still more by the intramuscular injection of strychnine ($\frac{1}{100}$—$\frac{1}{60}$ grain) two or three times a week. The injections of strychnine should not be practiced until after the period of restoration—the stationary period.

PROGRESSIVE MUSCULAR ATROPHY.

Definition.—By the term *progressive muscular atrophy* is meant a gradual and progressive wasting of the voluntary muscular system, which pursues a certain defined course.

Causes.—Numerous examples of hereditary transmission, some of them very remarkable, have been reported. The male sex is much more susceptible, and this is equally the case when the disease is hereditary. The most active period of life—from thirty to fifty—is the period of greatest liability; but youth and early manhood are by no means exempt, cases occurring before ten. Powerful muscular exertion, or overstrain of a group of muscles in certain occupations, seems to excite the disease; and in children the disease is invited to the lower limbs by prolonged effort on the legs. Exhausting diseases, the poisons of lead and syphilis, and certain dyscrasiæ, seem to exert an influence in developing the disease. Exposure to cold and mechanical injuries have apparently given rise to progressive atrophy.

Pathological Anatomy.—The morbid alterations are of two groups—spinal and muscular. The changes in the spinal cord are similar to those which take place in the spinal arthropathies in general, i. e., atrophy and degeneration of the anterior columns, wasting and disappearance of the multipolar ganglion-cells, of the anterior horns, hyperplasia of the neuroglia, corpora amylacea, granule-cells and fat-corpuscles. The anterior roots are similarly affected—are wasted, atrophied, and degenerated. In one third of the reported cases in which the cord was examined, no changes were found of any kind. The alterations in the muscles have been most elaborately studied by Friedreich,* who holds to the muscular origin of the disease. He asserts that the initial change consists in an inflammation with hyperplasia of the interstitial connective tissue uniting the primitive bundles. Morbid changes occur in the primitive bundles: proliferation of the nuclei and multiplication of the muscular corpuscles. Wasting of the muscular substance goes on, *pari passu*, with the increase of the connective tissue, and fatty degeneration contributes to it. The final result is, that the muscle is converted into a mere fibrous band with numerous fat-cells, the development of this latter material taking place outside of the muscular elements and in the newly formed connective tissue. The theory of Friedreich, which he maintains with remarkable skill and learning, is that the disease begins in the muscles, the intramuscular nerves are next affected, and an ascending neuritis conveys the morbid process to the spinal cord, which becomes in turn diseased. The other view is, that the changes in the muscles are secondary to the morbid process in the spinal cord, especially in the multipolar ganglion-cells of the cornua.†

Symptoms.—According to Friedreich's statistics, of one hundred and forty-six cases, there were one hundred and eleven instances of the dis-

* "Ueber progressive Muskelatrophie," etc., von Dr. N. Friedreich, Berlin, 1873, cap. ii, p. 46.

† Charcot and Joffroy, "Archives de Physiologie," vols. ii and iii, *op. cit.* A. Hayem, ibid. See also, as explanatory of spinal affections consecutive to nerve-injuries, A. Vulpian, ibid., p. 221.

ease beginning in the right upper extremity, twenty-seven in the lower, and eight in the lumbar muscles. Sometimes the tongue, sometimes the palate muscles, an example of which the author has seen, are first affected. The first dorsal interosseus is usually the first muscle attacked in the upper extremity, then the muscles of the thenar and hypothenar eminence, the deltoid, etc. Sometimes the pectoralis major and serratus magnus are the first to undergo atrophy. In children the lumbar muscles are usually the first to atrophy, the degeneration taking the form of pseudo-hypertrophic. The loss of volume which the muscles undergo is not always a measure of the real degeneration, since a very considerable hyperplasia of the fatty tissue sometimes takes place, with the effect to increase the apparent size. The next symptom is fibrillary contraction: the muscle undergoing atrophy so long as it remains, is agitated by fine tremors, which consist in waves or oscillations of movement of the muscular fibrillæ. If, now, the muscles of the diseased hand are tested by the dynamometer, they will be found extremely weak as compared with the sound hand. The hand also becomes greatly deformed, rigid, and claw-like, presenting the appearance of a bird's talons. The electro-contractility is preserved so long as muscular fibers remain to be stimulated, but the reaction to the galvanic persists for some time after the faradic excitability has disappeared. In most patients a good deal of pain is experienced in the muscles about to be affected and during the process of wasting, but the sensibility to pain and to temperature diminishes to below normal in the last stages. The temperature of the wasted parts is also reduced several degrees, and they are cold to the touch; and the integument appears normal or pale, or blue, and cyanosed. The perspiration is usually increased in the affected member or part, and sometimes generally. Changes in the joints, comparable to those which take place in locomotor ataxia and other spinal diseases, are also observed in progressive muscular atrophy.* Changes in the pupil and other oculo-motor phenomena occur when progressive muscular atrophy is associated with glosso-labio-pharyngeal paralysis. This disease may be accompanied with fever during the first weeks or months, often associated with the joint-lesions. How far this is accidental or a necessary part of progressive muscular atrophy does not appear to be well understood.

Course, Duration, and Termination.—The course of this disease is extremely protracted in many cases. The manner of spread of the myopathic process is not in accordance with a uniform plan. It sometimes extends by contiguity of tissue, sometimes leaps over groups of muscles to attack distant muscles. The extension is limited by the larger joints. Beginning in the hand, an extension to the arm does

* On this point consult Weir Mitchell's "Spinal Arthropathies," in "American Journal of the Medical Sciences," April, 1875, p. 339.

not take place; some of the extensors of the forearm undergoing atrophy, the muscles of the arm are not attacked; the deltoid and arm muscles affected, the elbow-joint is not passed; similarly in atrophy of the leg-muscles, the knee-joint seems to prevent extension to the thigh. Some muscles are never affected; those of the head are not often; and, when the tongue and lip muscles and the laryngeal muscles are affected, the disease is complicated with glosso-labio-laryngeal paralysis. The diaphragm and the respiratory muscles and the accessory muscles of respiration are finally invaded. Death then ensues by hypostatic congestion and œdema of the lungs. When the larynx is invaded, the voice is lost, and there is difficulty of breathing from cessation of the laryngeal movements. The muscles of the ear may also be invaded, and impaired hearing result. Friedreich gives a remarkable example, pictorially represented, of a man all of whose voluntary muscles are wasted, and who seems to retain alone the power of breathing. The march to this end is exceedingly slow, unless, as is not unfrequently the case, the morbid process involves the anterior cornua of the medulla oblongata, the effects of which have already been described. At first no trouble is produced by the wasting of the muscles of the extremities; the general health does not suffer; the powers of body and mind are otherwise adequate to their work. Sometimes the disease is arrested, and remains stationary for years. A few cases are terminated by bed-sores; many by intercurrent maladies, of which pulmonary tuberculosis is the chief.

Diagnosis.—A fully formed case can never present any difficulty in this respect, but at the initial period there may be doubt whether the wasting is due to local injury, injury of the nerve-trunk, or the result of rheumatism. The distinction rests on the pains, the fibrillary trembling, and the absence of any local cause to account for the atrophy.

Treatment.—Nothing has ever been accomplished by the use of internal medicines. The author has apparently effected great improvement in a case, confined as yet to the left upper extremity, by the injection of glycerine solution into the wasting muscles. The strength of the solution is one third glycerine, and it is injected three times a week. The two remedies of unquestionable utility are galvanism and massage. The author has had good results from galvanism, and he can not share the despondency of authors generally in regard to its utility. Erb reports favorably as to the good effects of the galvanic current. Strong currents must be used to excite vigorous contractions for a brief period—two minutes. A descending current should also be applied to the whole length of the spine, daily, for a minute or two. Massage, using with friction a fat, preferably lard, is also highly serviceable. This should consist of friction, kneading, and tapping the muscles. Hot douches to the spine and the rubbing wet pack for the affected members are also to be highly commended.

PSEUDO-HYPERTROPHIC PROGRESSIVE MUSCULAR ATROPHY.

Pathogeny and Symptoms.—This disease differs from progressive muscular atrophy, in the remarkable fact that the atrophied muscles increase in size, and are apparently hypertrophied, because of an hyperplasia of the connective and fatty tissue. The anatomical change consists, in brief, in a proliferation of the connective tissue between the fibrilla (Friedreich) and the adventitia of the small vessels. The newly formed connective tissue is remarkable for the number of its cells and nuclei, which are transformed into fat-cells. As the connective tissue develops the muscular elements disappear, or at least only in part remain, much altered, and thinner. Now and then are encountered some muscular fibers which have undergone hypertrophy. The muscular elements are also invaded by an irritative process, become granular and degenerate, so that the atrophy is not wholly a simple atrophy from overgrowth of the connective tissue. When the process is complete the muscles present a grayish or yellowish-white appearance, and can hardly be distinguished from the adjacent fatty and connective tissue.

This disease occurs almost wholly in childhood, and before ten years of age. In eighty cases, it began from the first to the fifth year in forty-five; from the sixth to the tenth, twenty-two times; from the eleventh to the sixteenth, eight times; and in five cases it occurred from the twenty-second to the forty-third year (Erb). Hereditary influence plays a very important part in the development of the disease. Other causes have been assigned, and probably with little reason, for all the facts go to prove the existence of a peculiar neurodiathesis.

The morbid process begins in the lower limbs—chiefly in the legs, although it may begin in the thighs. Before the hypertrophic enlargement manifests itself, muscular weakness has occurred; fatigue is quickly experienced; the legs trip easily and give way; the gait is awkward. After a time a child thus affected is not able to rise, when down, unless aided, and can not walk unless steadied; the gait assumes a straddling manner, somewhat like that of a duck, and when the thigh-muscles are affected he can not rise unless he supports his thighs by his hands, and in sitting down can not control the act, but plumps down suddenly. When recumbent, the legs are wide apart, the soles of the feet turned toward each other, the heels drawn up, and the knee and hip joints flexed. All the movements of the foot are imperfectly executed, except flexing the toes; the movements of the thigh are equally imperfect, except mere flexion of the knee. The position in standing is very characteristic: the lumbar portion of the spine is greatly incurved (lordosis), the dorsal portion bent outward (gibbosity). The diminution in power offers a remarkable contrast to the enormous bulk of the affected members. If the disease attacks the upper extremity, it takes the form of progressive muscular atrophy, and the two may exist together. Before the muscular tissue has disappeared, the same fibrillary twitchings occur as in the other form of the disease. The electro-contractility declines progressively with the diminution of the muscular elements, and in this disease the more decidedly because of the great collection of fatty and fibroid tissue overlying the muscular elements. There is more or less pain experienced by these patients, in the back, and through the parts to become affected. The temperature declines several degrees in the hypertrophied and atrophied parts. The termination of these cases has been by some intercurrent disease, usually of the respiratory organs.

CHRONIC POLIOMYELITIS ANTERIOR—ATROPHIC SPINAL PARALYSIS.

Definition.—As there is an acute poliomyelitis affecting the anterior columns, so there is a chronic disease of similar character, but possessing some distinctive features. It consists in a chronic atrophic degeneration—a muscular paralysis with wasting and atrophy of the affected muscles—secondary to degeneration of the gray matter of the anterior horns.

Causes.—While the acute anterior poliomyelitis attacks children more especially, although occasionally appearing in adults, the chronic affection occurs at the middle period of life—from thirty to fifty years of age. As very similar symptoms and lesions are caused by lead, copper, and some other metals, the modern use of these metals in domestic life may have a causative relation to this and corresponding nervous affections. Injury to the spine, excessive fatigue, exposure to cold and damp combined, sexual and alcoholic excess, have all been supposed to bring on this disease. In any case thus produced it is quite certain that a neuropathic type of constitution must have existed.

Pathogeny and Symptoms.—The seat of the pathological changes and their general character correspond to those of acute anterior poliomyelitis. The multipolar ganglion cells are degenerated and wasted, the vessels are thickened, the perivascular lymph-spaces crowded with leucocytes, red corpuscles, and granular matter, and the anterior nerve-roots more or less advanced in atrophy. The paralyzed muscles exhibit the characteristic degenerative changes, consisting in atrophy and disappearance of the muscular elements, and the substitution of connective and fatty tissue.

The symptoms develop rather insidiously, with a sense of fatigue and exhaustion of the lower limbs, pain in the back, loins, and hips, headache and some slight feverishness, with the anorexia and general *malaise* belonging thereto. Then some tingling, creeping, and crawling sensations, "pins and needles," and distinct muscular weakness

are experienced. The weakness gradually deepens into a paralysis, a group of muscles or an extremity loses its power entirely, and next other muscular groups, or a member, become paralyzed. As a rule the morbid process begins in a lower extremity and extends to the upper members, but the process may be reversed, and the paralysis beginning above may extend downward. The flexors of the foot first, the flexors of the leg and thigh next, and then the extensor of the limb are affected. In the upper extremity there is no regular order for the paralysis to occur: the muscles of the hand—the flexors of the fingers and hand; again, the extensors of the forearm, are first paralyzed, and the process extends thence to the arm and shoulder-muscles, until all become paralyzed and wasting. Ultimately, the muscles of the back and of the abdomen, and sometimes, also, of the chest, are paralyzed, so that the breathing becomes difficult because carried on by the diaphragm, and the expulsion of the fæces and urine is inefficiently performed. When the paralysis reaches this extent, the patient is quite helpless, and can not maintain the sitting posture. The functions of organic life are, however, carried on in the normal manner. Digestion, assimilation, the circulation, the sexual functions, the bladder and rectum, remain unaffected. Sensibility is not impaired, and bedsores do not form. The reflexes decline and disappear with the progress of the changes in the spinal cord and muscles. The electric reactions are changed from the normal in harmony with the anatomical alterations. As the muscles atrophy, faradic excitability declines quantitatively, and presently ceases. The galvanic excitability increases in accordance with the formulæ of the reactions of degeneration. But galvanic excitability disappears finally in those muscles so far atrophied that none of the proper anatomical elements remain.

Course, Duration, and Termination.—The progress of this affection varies in rapidity between wide limits. In some cases paralysis supervenes rather suddenly soon after the initial symptoms appear; in others, after the first weakness and heaviness of the lower limbs, months, even years, intervene before complete paralysis occurs below and extends to the upper limbs. In the cases which take a favorable direction, the upward extension of the morbid process is arrested. If the changes in the cord continue to advance, after a time the medulla oblongata is reached, and then there is experienced that group of symptoms characteristic of disease of this part—as ataxia of speech, difficulty of swallowing, embarrassed respiration, cardiac failure, etc., and death from asphyxia. A considerable proportion of the cases improve: the upward extension of the disease is arrested; the paralysis slowly lessens and voluntary power gained, the electrical reactions changing accordingly—first, the abnormal galvanic excitability gradually declining, while the faradic excitability is recovered. It is by almost imperceptible gradations that improvement takes place, and although complete recovery does occur, more frequently there remain behind disabilities and deformities, the result of permanent changes in certain nerves and muscles, especially of the leg and foot.

Diagnosis.—The disease with which chronic anterior poliomyelitis is most apt to be confounded is *progressive muscular atrophy*, but the distinction between them is made by reference to the mode of onset, progress, and termination. The latter begins silently without systemic disturbance. The paralysis follows the atrophy in the latter, while in the former precedes it. The electrical condition of the wasted muscles is different; in progressive muscular atrophy the muscles react until far advanced in degeneration, but in chronic anterior poliomyelitis the reactions of degeneration appear soon after the paralysis occurs. In respect to rate of progress, duration, and mortality, progressive muscular atrophy differs widely from the other, in that it is slower in progress, much longer continued, and is fatal, whereas chronic poliomyelitis anterior frequently gets well.

Treatment.—The reader is referred to the section on acute anterior poliomyelitis for the method of treatment, which is as applicable to this disease.

ACUTE ASCENDING PARALYSIS—LANDRY'S DISEASE.

Definition.—This disease, as its name implies, is an acute motor paralysis, usually ascending, and involving all parts of the voluntary muscular system, with wasting of the affected muscles, but without change in the electrical reactions. It is entitled "Landry's Disease," because he first accurately described and differentiated it, although it had been observed before.

Pathogeny and Symptoms.—After exposure to cold, or in consequence of alcoholic excess, or because of the presence of metals in the system, or excited by some unknown cause, the patient, who is usually between twenty and forty years of age, is seized with a little fever, pains in the back and limbs, numbness, tingling, and a sense of extreme fatigue. These symptoms continue a few days—rarely several weeks; next come on extreme weakness of the feet, then of the legs, of the thighs, until in a few days there is complete paralysis of the lower extremities; the muscles are completely relaxed, and the legs lie inert wherever placed. It is very distinctly an acute ascending paralysis—for no sooner is the loss of power in the inferior extremities completed than the muscles of the trunk are invaded, and, even before the abdominal, the upper extremities become paralyzed, and, like the lower, lie relaxed and motionless—first, the hand in its complicated motions, then the forearm, and ultimately the arm and shoulder.

Not all cases are paralyzed in the order above described, and none

are affected in strictly anatomical order—for in some few instances the paralysis begins in the hand and thence descends to the lower limbs, and in all cases the hands and feet are implicated before the parts most nearly connected with the spinal cord. Rarely a case occurs in which the medulla is first attacked, with the characteristic respiratory and circulatory disturbances resulting. According to Landry, the order in which the paralysis extends in the muscular system is the following: the toes and feet with the muscles acting on them; then the thigh and pelvic muscles, posteriorly, and, after these, the muscles in the front part of the thigh. A similar order is pursued in the upper extremity: first are affected the muscles acting on the fingers and hand; next those which move the arm on the scapula, and then those moving the forearm on the arm. Entirely after the muscles of the extremities, the trunk muscles are paralyzed; then come the muscles engaged in the respiratory movements, and after these, and in the order named, the muscles of the tongue, pharynx, and œsophagus. Attention to the mode in which the paralytic phenomena are developed will facilitate the diagnosis.

Sensibility is somewhat affected in many cases. There are numbness, tingling, formication, and impaired tactile and pain-sense, usually in the inferior extremities, but elsewhere the æsthesiometer discloses no changes. The reflexes are, as a rule, finally abolished, although at first there may be no change. The "knee-jerk" ceases. The bladder and rectum are not affected.

Whether the medulla oblongata be early or late affected, in every fatal case it is invaded by the morbid process, and its injury is the cause of death by asphyxia. The disease is of short duration for the most part, death occurring in a few days, or in a few weeks certainly, the average being ten days. It is not always fatal. The progress of the disease in favorable cases is arrested before the medulla oblongata is reached, although some cases presenting evidences of the functional derangement of this organ have, nevertheless, terminated in recovery. When improvement does take place, the change is early manifested by a cessation of morbid activity, by a return of function to the parts last paralyzed—the hands coming into use, then the arms, muscles of the chest, and abdomen, and finally of the lower extremities.

The rapid progress made by this affection, the preservation of the electric excitability of the muscles and of the functions of the rectum and bladder, the absence of bed-sores, and the prompt extension from below to the medulla oblongata, separate acute ascending paralysis from other acute affections of the cord, and render its diagnosis comparatively easy after the full development of the symptoms.

The prognosis, although grave, is not necessarily fatal. About one half of the cases, apparently, get well.

Treatment.—As some kind of toxic agency is supposed to underlie the morbid process, remedies should be addressed to the elimination of the poison. Syphilis, the minerals, will require large doses of the iodides. The rheumatic diathesis will need the remedies appropriate to that state. When the disease succeeds to some infection, as variola or typhoid, only the general condition can be taken into account. Galvanization of the cord has been apparently of benefit in a few instances. It is probable that the subcutaneous injection of strychnine would do good. It should be tried cautiously, and, if it act favorably, should be pushed, for, in the absence of specific lesions, a loss of power to functionate seems to be the essential condition of the spinal cord.

SOME DISEASES AFFECTING THE BRAIN AND SPINAL CORD.

MULTIPLE SCLEROSIS OF THE BRAIN AND CORD.

Definition.—By the term *multiple sclerosis of the brain and cord* is meant a disease characterized by the formation of isolated patches or nodules of sclerotic tissue in the brain, pons, medulla, cerebellum, and spinal cord. It is sometimes treated of as *cerebral sclerosis* and *spinal sclerosis*, but it becomes more and more apparent that neither organ is separately affected. By Charcot* it is entitled "disseminated sclerosis."

Causes.—In this disease both sexes are about equally affected, and it occurs from youth to middle age, becoming very rare after forty-five and before ten. The most powerful predisposing cause is heredity. Exposure to cold and fatigue, living in damp habitations, and sudden exposure of the body to cold and dampness when in a warm and perspiring state, are alleged to be causes, but doubts may well exist as to their influence unless a predisposition exist. Powerful and prolonged moral emotion, chagrin, anxiety, and other depressing moral causes, may favor the development of this affection. It occurs in the convalescence from acute infectious diseases.

Pathological Anatomy.—The disease in the brain and cord, to the naked eye, appear as glistening nodules underneath the pia. They are distinctly circumscribed, grayish patches, raised a little above the

* "Diseases of the Nervous System," "Sydenham Society Translation," lecture vi, p. 157.