

die very quickly, no curare was employed. Their muscle substance remains irritable for a sufficiently long time to enable excellent fatigue records to be obtained. At the temperature of the body there is a progressive diminution in the height of the curves, with no lengthening of either the ascending or the descending portion. If, instead, the muscle be cooled to 12 C., and then fatigued, essentially the same result is obtained. There is, however, a broadening of the upper portion of the curves, indicating a tendency of the cooled tissue to linger in the contracted state; and this phenomenon, besides those above mentioned, is the most marked feature of the record of fatigue of the cooled mammalian muscle. While constantly present in the muscles studied, it is not sufficient to amount to a lengthening of the contraction process as a whole, with the single exception of the gastrocnemius of the white rat. In eight of the ten experiments performed with that muscle, careful measurement showed a slight lengthening, amounting in the maximum case to barely 0.7 of the original curve. This is almost a negligible quantity in comparison with the much greater lengthening exhibited by the muscles of both the frog and the turtle.

In view of the above facts, the following conclusions seem to be justified:

1. There is a physiologic difference between cold-blooded and warm-blooded animals in the mode of fatigue of their excised, voluntary muscles.
2. One of the characteristic phenomena of fatigue of the excised voluntary muscles of cold-blooded animals is a pronounced slowing of the whole contraction process manifested by a lengthening of the muscle curve. This is most pronounced at low temperatures. It is diminished, but not obliterated, at high temperatures.
3. The excised voluntary muscles of most warm-blooded animals fatigue without a slowing of the contraction process as a whole. At low temperatures there is observable a broadening of the muscle curve, caused by fatigue, which, in individual cases, may result in a minute lengthening of the curve as a whole.
4. The above facts suggest that the general physiologic differences between the muscles of cold-blooded and of warm-blooded animals are not due to immediate differences of temperature. The existence of a homo-thermal condition and the constant subjection of the skeletal muscle system to a uniform temperature, seem to impress on that system distinctive physiologic peculiarities.

College of Physicians and Surgeons.

LANDRY'S PARALYSIS.

WITH REPORT OF CASE.*

ROBERT MCGREGOR, M.D.
SAGINAW, MICH.

To the pathologist and clinical observer, acute ascending paralysis is a malady of more than ordinary interest. For nearly fifty years it has been known and studied, yet little light has been thrown on the essential pathologic process which is the basis of this remarkable disorder. Since Landry, in 1859, wrote his classic treatise on the subject, many efforts have been made to work out a solution of the perplexing problem, but with far from uniform results. Reasoning from the association of a flaccid paralysis, with preserved electric irritability and absence of trophic symptoms, Gowers ad-

vanced the theory of a toxin acting on the arborizations of the pyramidal fibers in the anterior horns of the spinal cord in a manner analogous to the effect of curare on motor-nerve terminations in muscle. This hypothesis, however, has never been reinforced by the conclusions of other investigators.

There are good grounds for believing that bacteria or the products of bacteria or toxic elements elaborated within the body having a selective action on motor neurons play an important rôle in the causation of this disease. Streptococci, staphylococci, diplococci and rod-like bodies resembling those of anthrax and typhoid have been noted by competent observers in the spinal cord in typical cases. It is quite conceivable that a variety of toxins may have in common the property of exerting a more or less profound influence on motor neurons, just as there are several well-known substances, organic and inorganic, which possess this singular power. The precise way in which these toxins affect the integrity of the motor elements has not been definitely determined. Considerable difference of opinion has always existed as to whether the motor structures in the cord or in the peripheral nerves were the parts primarily subjected to the morbid change. Some authorities, notably Ross, have maintained that the affection is a peculiar form of multiple neuritis. This view is based, not on the clinical manifestations, but on the finding of degenerative changes in the peripheral nerves in certain cases. Such findings, however, are by no means frequent, and in many cases the most careful scrutiny has failed to disclose any histologic alterations in either the peripheral nerves or spinal cord. Perhaps the most constant features found postmortem in typical cases are acute enlargement of the spleen and engorgement of the lymphatic glands, especially those of the mesentery, conditions which clearly indicate the invasion of the system by a toxic principle. As far as the nervous system is concerned, wherever the primary point of attack may be, it is evident that the pathologic change is an extremely subtle one; and of late the assumption has been gaining ground that in all typical cases this change affects the gray matter in the anterior horns of the spinal cord. It is probable, also, that in cases in which the toxicant is exceptionally virulent involvement of the anterior roots may follow, and a neuritic condition be induced in the peripheral nerves as a later and secondary consequence.

In the last score of years many important advances have been made in our knowledge of the physiology of the nervous system. In the light of this knowledge we are often enabled to reason from symptoms to the exact site of the pathologic process. The light gleaned from such knowledge in typical cases of acute ascending paralysis leads us to suspect that the motor cells of the anterior cornua of the spinal cord are primarily, and perhaps in most cases solely, concerned in the morbid action, and that with improved staining technic it will yet be possible to render apparent cellular changes which can not now be made manifest. It is probable that whatever changes the motor cells undergo are of a transitory character and of a nature to affect temporarily their function only, since the majority of patients who recover do so completely. Etiologic facts support in a striking way the theory of a toxic state or dyscrasia as an essential and antecedent condition. By far the greater number of recorded cases have followed in the wake of sundry infectious diseases, local or systemic disturbances of a septic character, or have been

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preceded by wounds, more or less trifling, which had served as points of infection. Some cases have been ascribed by certain authors to exposure to wet and cold, especially when this exposure was associated with alcoholism, but the precise way such causes operate is difficult to determine if we are to believe Landry's paralysis to be separate and distinct from multiple neuritis. If such causes were potent factors, it would be quite easily understood why most cases have been observed in males in the third decade of life.

So closely do many cases parallel multiple neuritis that it is not always easy to distinguish between the two disorders. In typical cases the diagnosis is based largely on negative evidence. Thus, in Landry's paralysis there is generally an absence of pain or tenderness along nerve trunks and of sensory impairment, trophic symptoms or electric alterations. The mode of onset of paralysis, too, is distinctive, being, as a rule, more abrupt, speedily completed and more profound, a distinguishing characteristic being the progressive implication of groups of muscles, commonly from below upward, and never beginning in both upper and lower extremities at the same time. Perhaps the cases which offer the greatest difficulty are those in which peripheral neuritis, to a greater or less degree, coexists with the spinal lesions. Such cases may have so much in common with multiple neuritis as to render a positive diagnosis practically impossible. Landry's paralysis rivals acute anterior poliomyelitis in the rapidity of its onset, although in the latter malady the affected areas, as a rule, are more circumscribed and the muscles are paralyzed *en masse* rather than in successive groups. The nature of the paralysis has a close correspondence in the two conditions, being in both flaccid, purely motor and with abolished reflexes, the sole distinguishing point of difference being the presence or absence of changes in electromuscular excitability.

A remarkable feature of this strange malady is its extremely high death rate, contrasting sharply in this respect with both multiple neuritis and acute anterior poliomyelitis. The recorded results of several groups of cases occurring in this country and in Europe give a mortality varying from 50 to 80 per cent. In the majority of cases a fatal result follows within a week or ten days. The incidence of bulbar symptoms is of grave significance, death, as a rule, occurring from interference with the cardiac or respiratory centers. The occurrence of mental symptoms is of evil omen, as indicating a system especially susceptible to toxic influence or a high grade of virulence of the toxic agent. Recissions of mental or bulbar symptoms are hopeful indications. The degree of rapidity of onset and the degree of completeness of the paralytic condition may be taken as a criterion of the intensity or the severity of the pathologic process, and consequently of the comparative gravity of a given case. Recovery in favorable cases is always slow, but frequently complete. In protracted cases, the paralyzed limbs may or may not undergo atrophy. Considerable uncertainty exists on this point; doubtless because of the rarity of the disorder so few opportunities have presented for studying the disease in its later stages. In two rather protracted cases I have seen, the patients had become quite emaciated, but the muscles had preserved, unchanged, their electric irritability.

The following case which had been under my observation for a period of two years well illustrates the characteristic onset and symptoms of Landry's paralysis, be-

sides presenting some peculiar phases of the affection which are interesting and suggestive:

Patient.—J. B., a young man, aged 23, was born and reared on a farm near Saginaw, Mich.

History.—There is nothing particularly noteworthy in either his family or personal history. He had been a moderate smoker, but never addicted to intoxicating liquors in any form. At the age of 10 he had been operated on for appendicitis. Early in the summer of 1901 he was employed as fireman on the steamer *Mecosta*, sailing between Cleveland and Lake Superior ports. On June 26, 1901, while at Fort William, he, in common with the rest of the crew, was vaccinated by the health officer of that port. A very sore arm followed, with considerable swelling of the glands in axilla.

Course of the Disease.—In a few days he became subject to chilly sensations, alternating with transitory spells of fever. He suffered, too, from occasional attacks of vertigo, and from rather profuse sweatings. In spite of these symptoms, he continued at his work all the way down to Cleveland, which was reached ten days later. Here he began to experience in the right foot a peculiar feeling of weakness, which gradually ascended the limb to the hip. In a few hours a like sensation was felt in the left foot, and steadily progressed upward. Recognizing that something was seriously wrong, he entered the Marine Hospital in Cleveland. Next day the condition of weakness in the legs had deepened into absolute paralysis. The arms soon began to suffer in a similar fashion, the right being first affected from the fingers upward. Within a period of eighty-four hours all four extremities were rendered powerless, with flaccid muscles and abolished reflexes. The paralysis of the arms was slightly less profound than that of the lower limbs, but he was unable to feed himself or to use them for any purposes whatever. Up till this time, and during the further progress of the case, there was an entire absence of pain, of nerve-tenderness in any degree and of sensory symptoms of every kind. There was no headache or mental disturbance. The functions of the bladder were not interfered with. At no time were there any edema, glossy skin or epithelial changes; nor were the coldness and lividity of the limbs, so often seen in anterior poliomyelitis, ever noticed. Before the end of the first week symptoms appeared indicating extension to the medulla. The lips became paretic, and the tongue, palate and throat were likewise affected, so that speech and swallowing became seriously impaired. Diplopia appeared and eventually convergent strabismus and ptosis. The heart, too, began to manifest disturbance of its rhythm, with slight dyspnea and occasional spells of tachycardia. So deplorable was the condition of things that a telegram was sent to the patient's father, urging him to come at once if he wished to see his son alive. On the father's arrival, he decided to take the boy home, and accordingly a journey of nearly 300 miles was made by rail, with the patient on a cot in the baggage car. He stood the trip very well, and after being for a time at home entered the Saginaw Medical College Hospital as one of my clinic patients.

During the period of several weeks the case exhibited no material change in any respect. Except at the stage of onset, the temperature was not elevated, but occasionally showed a subnormal tendency. He had no pain and no particular distress aside from the difficulty in swallowing, and the utter inability to move a limb or to express himself by the use of speech. He slept well and took a fair amount of nourishment, but his muscles had markedly diminished in volume, more, perhaps, than could be accounted for by mere disuse, yet a response, more or less prompt, could everywhere be obtained to the faradic current. After two months the bulbar symptoms began slowly to recede, and two weeks later the dysphagia, dyspnea, difficulty of articulation and ocular derangements had wholly passed away. The paralytic condition of the limbs, however, remained unchanged. No contractures or pedal deformities had occurred. The first sign of motor activity appeared three months after onset, and consisted of slight flexor movements of the left arm, the limb latest to become affected. Similar movements were soon pos-

sible in the right arm, and a little later in the left leg; the right lower limb—the first to become paralyzed—lagged considerably behind its fellow. The flexor groups of muscles, it was observed, manifested evidence of returning power very largely in advance of the extensors, which had to be reinforced by mechanical and electric stimulation. This lack of correspondence between flexors and extensors produced a condition of wrist drop and foot drop highly suggestive of multiple neuritis.

Treatment.—In the general treatment of the case reliance was placed on strychnia, iron, arsenic, the hypophosphites and glycerophosphate compounds.

Result.—The most gratifying results seemed to follow from a simple solution of the glycerophosphate of iron, together with moderate doses of strychnia. The return of muscular power, however, was an extremely protracted process, nearly eight months having elapsed from the time of onset before the patient was able to stand unaided, and fully ten months before he could walk more than a few steps without fatigue. After a year, his strength improved more rapidly, but nearly all of the second year was required in regaining something like his former vigor. In June, 1903, after two years of illness, he entered on the duties of a mail carrier over an R.F.D. route, a vocation he has since followed satisfactorily in all weathers without the loss of a single day.

Previous to his illness his usual weight was 165 pounds; since his illness his weight has never exceeded 135 pounds. He manifests at times a previously unusual tendency to trip over words in speaking. He still exhibits slight weakness of ankle on both sides, most marked on right. The knee jerks have thus far failed to return.

MULTIPLE NEURITIS.

WITH REPORT OF FOUR CASES.*

DAVID I. WOLFSTEIN, M.D.
CINCINNATI.

The principal interest in neuritis centers in the fact that, on the one hand, it resembles mere functional affections like neuralgia, which is a disease of the sensory nerve characterized by pain in the course of the nerve, or in its end distribution; on the other hand, it may be so extreme and grave in its manifestations as to be confounded with even the most serious affection of the cord itself. The term neuritis implies that there is an inflammatory condition of the nerve affected, though the diseased condition is not necessarily inflammation in its usually accepted sense, but may also be a degeneration, though with a tendency toward repair.

A neuritis, of course, may be confined to a single nerve when its symptoms will be those of the nerve affected.

As a matter of experience, however, we are more interested in that form of neuritis which affects many nerves simultaneously. This form we call multiple neuritis or polyneuritis. As regards causation, multiple neuritis follows various factors, which may be grouped as follows:

First. Toxic cases, due to the action of the poison derived from within the body. The poisons that produce most of the cases that we meet with are alcohol and mercury and lead, and occasionally arsenic; of course, there are many other toxic substances which produce this condition, but which will not be discussed here, as we see them less frequently.

Second. The second group is due to some agent acquired or developed within the body, that is, infectious forms which may accompany or follow most of the acute

infections. As an agent which can give rise to this condition besides many other acute infections, especial attention should be directed to influenza, gonorrhea and the puerperal state. As a matter of knowledge, it is well known that the disease sometimes takes an epidemic form, beriberi.

Third. A large class of cases is due to general diseased states of the body, of whose origin we are not informed, such as rheumatism, acute diabetes, anemia, tuberculosis, syphilis, etc.

Fourth. There is another group of cases which followed exposure to cold or developed apparently without determinable cause.

As a type of neuritis due to alcohol, let me give the history of the following case:

CASE 1.—Mrs. M., a young woman, aged about 30 years, who had been suffering for three or four months previously with very severe pains and with growing weakness in the extremities, called me to see her at a time when she was bedridden and unable to move the feet. She was very emaciated, her digestion was very much impaired, examination of the viscera revealed nothing abnormal except the heart action, which was very much accelerated, with very weak pulse. The pupils reacted promptly. There was no involvement of any of the cranial nerves. She complained greatly of extreme sensitiveness, but there was little numbness and tingling, though previously she had complained much of these symptoms. Pressure along the course of the large nerve trunks was painful. The muscles were relaxed and flabby, both of the arms and legs. The wrists and feet presented the well-known drop condition. The wrist drop was very marked, as was also the foot drop. As is well known, this wrist drop is due to a paralysis of the extensor muscles. The legs were very much emaciated and power was practically abolished. The paralysis was so complete in the lower extremities, with an inflammation of the spinal cord, that myelitis might have been suspected. A negative symptom of great importance was the absence of any interference with the function of the bladder or rectum, and another negative symptom, to which Star calls attention, was the absence of pain or anesthesia about the trunk. As is well known in the various forms of disease of the cord, like myelitis, or locomotor ataxia, such disturbance of sensation on the trunk is present frequently.

In making the diagnosis, the family history was taken into consideration, and a most shocking state of affairs was elicited. The husband was a confirmed drunkard and had induced his wife to follow in his evil tendency, and, in fact, a little child of this couple had also been taught to drink now and then. The patient had been drinking steadily for two or three years previous to the time I saw her. She finally developed a condition of mental confusion and uncertainty as to her whereabouts, and exhibited a certain hebetude of mind which is a very frequent accompaniment of severe forms of multiple neuritis, especially of the alcoholic type, and is known as Korsakoff's insanity.

There was great loss of memory of recent occurrences and much insomnia; later there were hallucinations of sight and hearing. The knee jerks were gone, as were also the ankle jerks. The paralysis began in the feet and legs, extending upward rapidly, and soon involved the arms and forearm. The muscles did not respond to the Faradic current, and when tested with the galvanic current showed the presence of degeneration.

Only a strong current elicited a contraction. There was great sensitiveness to manipulation, and the tactile sense was very much blunted. This case was so extreme and the general condition of the patient so depraved that all attempts at treatment were futile and death ensued, due to collapse.

Let me now give you an example of the milder form, in which the toxic agent was the poison of influenza, under the head of Group 2:

CASE 2.—Mr. B. came to his physician's office complaining of great pain in the lower extremities, which had not yet become severe enough to prevent him from walking. This pain in the course of two or three days increased and there was progressive

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