A CASE OF LANDRY'S (ACUTE ASCENDING) PARALYSIS: RECOVERY.*

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FOR the following notes I am indebted to Drs. J. R. Edward and W. M. Sadler, house-physician and assistant house-physician, Nottingham General Hospital:

The patient, a collier, aged 23, employed on the "pit bank," was admitted to the Nottingham General Hospital on January 20th, 1908, having been laid up about twelve days.

Previous History.

He had been addicted to an occasional indulgence in alcohol, but was not a constant "soaker." There was no history of syphilis, diphtheria, or lead poisoning. Some months back he had gonorrhoea. He had never been abroad. The family history was unimportant.

Present Illness.

A few days before the attack the patient had a drinking bout, got wet through, and sat for some time in his wet clothes. After this he complained of lassitude and general muscular aching.

On January 8th he felt "starved" and sick, and vomited slightly. The next day he had a numb feeling under the nails of both hands, as if the fingers had gone to sleep. By night the arms were involved as far as the elbows. Next day the arms and feet felt very cold and numb, and there was marked muscular weakness of all four limbs and of the trunk. Face not affected. He just managed to sit up once in bed, but could not dress himself or hold anything in the hands. The limbs twitched a good deal and ached. Drowsy in the day, wakeful at night. Appetite good. Bowels were moved by medicine. There was no loss of control over the bladder, except on one occasion, when he was being lifted on to a stretcher. About a week after the onset the eyelids and mouth became affected. He could only just see through the chink left by the dropped lids. No headache or other pain beyond aching in the limbs. Three days after the onset he began to be troubled with cough and phlegm.

Condition on January 23rd, 1908.

He is a man of healthy appearance, but is perhaps a little cyanosed. Lies on back. Troubled with weak, ineffectual cough. Mouth dry (full doses of belladonna were being given). Mucous membranes of normal colour. Tongue moist, red, a little furred, gums normal. Pulse 100, moderate tension, regular. Respirations 28. Temperature normal. Double ptosis of upper lids, which cannot be raised above $\frac{1}{2}$ in. Slight mucopurulent discharge from conjunctiva. Pupils equal, react to light and accommodation. Internal and external recti on both sides markedly weak. No nystagmus. Double weakness of facial nerve, seen on trying to smile. Palate moves well. Tongue protruded straight. *Upper limbs:* No muscular wasting, but almost complete symmetrical paralysis. All muscles respond to faradism, except flexors of first and second fingers of right hand. No reaction of degeneration. *Lower limbs:* Knee-jerks absent. No ankle clonus or plantar reflex. No wasting of muscles, but great weakness. Some slight movements can be performed, others not at all. All muscles react to faradism and galvanism. No reaction of degeneration. *Sensation:* Everywhere normal. *Trunk:* The intercostal muscles are paralysed and respiration is carried on by the diaphragm. Superficial reflexes absent. The recti abdominis appear almost completely paralysed and the abdominal wall is lax. *Speech* is blurred owing to weakness of the facial muscles; no hoarseness. *Heart:* Sounds normal, apex beat in fourth interspace just external to the nipple. *Lungs:* Slight dullness at bases, but no definite change in breath sounds. *Mental condition:* Bright when he is spoken to, drowsy at other times.

April 23rd.—There is now fair movement of the ribs in inspiration in respect both of expansion and elevation. The umbilicus moves upwards when he attempts to contract the recti. Facial and ocular muscles now normal. Great wasting of all small hand muscles, also forearm muscles, extensors more especially. Biceps wasted, but can be voluntarily contracted. All other muscles of upper arm are wasted and powerless. Sensation normal. *Lower limb:* Cutaneous sensation normal. Muscles tender on pressure or passive movement. Great muscular wasting in thigh, leg, and foot, with plantar flexion. He can slightly bend knee, move toes, and flex ankle. These movements cause pain. Slight glossiness of skin of foot. There is a general tendency to sweating.

July 23rd (six months after admission).—There has been considerable improvement in the arms, which he can raise so as to get hold of the bar at the bed-head. Can sit in a wheel-chair, but cannot propel himself. Patient discharged from hospital.

February 26th, 1909 (seven months after discharge from hospital).—Looks plump and well. The shoulder and arm muscles are in good condition, movements of shoulder and elbow well performed. The extensors of the forearms and the small hand muscles are atrophied, more so on the right side. There is permanent flexion of the two distal phalanges of the fingers, the proximal one being extended. He can propel himself in a

wheel-chair, but can do no useful manual work. There is wasting of extensors of the leg, consequently there is extreme foot-drop and plantar flexion of the toes. Knee-jerks not obtained. Can flex and extend the knee to some extent. Muscles moving the hip are very weak, and he cannot stand even when supported on each side.

Treatment.

He was at first given liq. strychnin. mv, with tinct. belladonna. mx, every two hours. Both these drugs are respiratory stimulants, and the only chance of recovery appeared to be in stimulating the respiratory centre. The belladonna was given also with the object of checking secretion into the bronchial tubes, where it tended to accumulate owing to lack of expulsive power. The belladonna after a few days' use caused a little noisy delirium at night, which was treated by a bromide and chloral draught. After the first ten days the strychnine alone was given. On March 2nd sodium salicylate was given on account of the muscular pains. To this bromide of potassium and hyoscyamus were afterwards added. Then strychnine was returned to. Massage and galvanism were regularly given during the latter part of the patient's stay, but were not well borne on account of the muscular tenderness.

The group of symptoms known as Landry's paralysis has been known since 1859, when the disease was first described by the observer whose name it bears. It is more correct to speak of a group of symptoms than of a disease, because it is quite possible that the "symptom-complex" in different cases has a different causation and variable pathological basis. By Landry's paralysis we understand an illness usually ushered in by sensory symptoms such as tingling, formication, etc., and then characterized by progressive loss of muscular power, commencing usually in the feet, involving in turn the legs, thighs, pelvic muscles, abdominal wall, arm muscles, respiration, deglutition, facial muscles, etc. There may be considerable loss of sensation, but more commonly this is absent, or confined to slight blunting at the tips of the fingers and toes. Most cases end fatally, especially when the respiration is affected, death occurring usually about the end of the first week, if not earlier, or it may be deferred three or four weeks.¹ In at least two respects this case is remarkable. First, it is distinctly stated in the notes that the arms were affected before the legs. This occurrence certainly runs counter to the commonly accepted notion that the paralysis invariably commences in the legs and ascends the body segment by segment, until its progress is either stayed, or life comes to an end by reason of the importance of the structures involved. But we are told on the authority of the late Dr. Ross² that Landry himself stated that the malady might commence in either pair of limbs, and that the march of the paralysis might be sometimes described as "centripetal" rather than "ascending." Occasionally both the arm muscles and those supplied by the bulbar nerves have been affected before the legs. In the second place, this case is remarkable inasmuch as, notwithstanding the almost complete paralysis, the patient recovered. When I first saw him he lay almost motionless, breathing rapidly, and of somewhat dusky appearance. When spoken to, he threw his head slightly backwards to enable him to see through the fissure left by his drooping lids. In fact, he seemed to be hanging on to life by a thread. Every time I visited the hospital I expected to hear of his death. When asked how he was he always said, "All right," and probably his calmness and pluck had much to do with his recovery. It is impossible to say why certain muscles and nerves were spared and others affected. The muscles of the legs, arms, and trunk, including intercostals, were absolutely powerless. The muscles supplied by the brachial plexus were all involved, yet the phrenics which arise from the cord immediately above were spared. The pneumogastrics, glosso-pharyngeals, and hypoglossals seem to have escaped, while the muscles supplied by the third, sixth, and seventh were affected. The temperature at first was normal. About the sixth week it reached 99.4° in the evening. This, practically afebrile course, conforms to the usual type.

Exposure to cold and excess in alcohol are well-recognized causes of Landry's paralysis, and both of these co-operated in the etiology of the case before us. It has also occurred during convalescence from small-pox, typhoid, influenza, measles, and diphtheria. It has occasionally supervened in connexion with the puerperal state, syphilis, septicaemia, and phthisis.³ It is three times as common in men as women.

As regards diagnosis, there is room for doubt whether the lesions in this case were in the spinal cord or in the

* Read before the Nottingham Medico-Chirurgical Society.

peripheral nerves. At any rate, it was not a case of ascending myelitis leading to rapid and extensive softening of the cord. This is shown by the preservation of faradic excitability, of the functions of the bladder and rectum, and absence of tendency to bedsores, or of anaesthesia. A certain mystery has always hung over Landry's paralysis because it has so frequently happened that no morbid changes in the nervous system have been discovered after death. The disease was capable of abolishing function and of destroying life, without leaving behind any lesion or demonstrable alteration of tissue to indicate in what manner those results had been brought about. There are doubtless cases in which the nerves and spinal cord have been examined by modern methods and no pathological changes have been found. In rapidly fatal cases the action of the poison on the nervous tissues may be sufficient to cause death before characteristic changes of structure have had time to develop. On the other hand, many of the earlier cases occurred before it was customary to examine the peripheral nerves, so it is probable lesions in some cases have been overlooked. When inflammation involves all the elements of a mixed nerve it is usual to find that both motion and sensation are affected. In the case before us we may regard the lesion as principally affecting the whole system of lower motor neurons. The axis cylinders of the motor nerves are outgrowths of the ganglion cells of the spinal cord, medulla, and pons. The chief stress of the destructive process would be found in some cases in the peripheral nerves, and doubtless most often there because of their distance from the centre, and in others in the ganglion cells themselves. And here we can see an explanation why motor power has been recovered in some parts of the body and not in others. So long as the nerve cells themselves are not destroyed, there is possibility of regeneration of axis cylinders and of recovery of muscular tone and motor power, while if the cells themselves undergo degeneration there is permanent muscular atrophy and motor paralysis. This has occurred notably in the cell groups innervating the extensors of the forearm and toes and the small hand muscles. Although the morbid process has exercised a selective action on the motor fibres of mixed nerves, it is probable there has also been interstitial neuritis, which by pressure would cause irritation of sensory fibres. In this manner the tenderness and pain in many of the muscles can be accounted for. Neither hyperaesthesia or anaesthesia of the skin occurred, which appears to show that nerves containing sensory fibres only were only slightly affected, if at all.

Dr. Michell Clarke, in an interesting article in the *BRITISH MEDICAL JOURNAL* for September 12th, 1908, would limit the term "Landry's paralysis" to cases in which the lesion is confined to the anterior cornual cells, degenerative changes being nowadays demonstrated by Nissl's method, but which would have been passed over by earlier pathologists, thus giving rise to the notion that Landry's paralysis caused no morbid changes of tissue. This proposition apparently assumes that true Landry's paralysis is always fatal, for the morbid anatomy of a patient who recovers must always be more or less a matter of speculation. In the clinical sense I think the case I have related is sufficiently near the type of Landry's paralysis to be included under that name. Dr. Clarke goes on to describe a case of ascending paralysis due to toxic polyneuritis. There was great muscular wasting, the reaction of degeneration was present, and the patient died after an illness of about three months. At the *post-mortem* examination the peripheral nerves showed a very advanced degree of neuritis. But this was not all, for many anterior horn cells, especially in the cervical and lumbar enlargements, showed degenerative changes, such as disappearance of the Nissl bodies, vacuolation, and disappearance of the nucleus.

The physical and chemical properties of the poison causing acute ascending paralysis probably vary, and its nature in different cases is but little known. In one published case⁴ micrococci have been found in the ganglion cells of the cord. Whatever the nature of the poison, it probably circulates in the blood like alcohol and the diphtheritic poison, and exercises a selective action on the lower motor neurons, the peripheral portion of which frequently suffers most. Another example of the selective action of a poison is that of the late effect of the syphilitic poison on the sensory protoneurons or on the pyramidal

cells of the cortex cerebri, producing the diseases known as tabes dorsalis and general paralysis respectively. The notion that Landry's paralysis is associated with no morbid tissue changes of permanent character can no longer be maintained, or that no muscular wasting occurs in cases that recover. This question was fully inquired into by the late Dr. Ross, and he "clearly showed that many cases have been reported which in the early stages were clinically indistinguishable from cases of rapidly fatal ascending paralysis, in which, when convalescence was protracted, some of the muscles became atrophied, and gave partial or complete reactions of degeneration to electrical tests,"⁵ *main en griffe*, and double ankle-drop being not uncommon distortions.

REFERENCES.

¹ For an account of a rapidly fatal case see *BRITISH MEDICAL JOURNAL*, February 27th, 1909. ² *Allbutt's System*, first edition, vol. vi, p. 701. ³ *Allbutt*, op. cit., p. 697-8. ⁴ *Allbutt*, op. cit., p. 702. ⁵ *Allbutt*, op. cit., p. 700.

ON THE USE OF CERTAIN NEW CHEMICAL TESTS IN THE DIAGNOSIS OF GENERAL PARALYSIS AND TABES.

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THE diagnosis of general paralysis of the insane is for two reasons a more important matter than the diagnosis of most forms of insanity. On the one hand, thanks to the clinical observations of Pilcz, Kraepelin, and a host of other workers, with the pathological researches of Nissl and Alzheimer, we have a clearer picture and more exact knowledge of the course and nature of the disease than about almost any other psychosis, so that we can foretell what events are likely to occur in it, and take measures to guard against them. On the other hand, the prognosis is more fatal and the lethal termination more rapidly reached than in any other form of insanity, so that it is important to recognize the condition as soon as possible, and to get the patient's affairs arranged on the basis of that knowledge. Although in a pronounced case the clinical picture is one of the sharpest in the whole of medicine, yet in an early or non-typical case the difficulties in diagnosis are often exceedingly great. So much is this the case, that it is found in asylum practice that the majority of the patients are admitted either with an erroneous diagnosis, or else comparatively late in the course of the disease. Hence the additions to our knowledge that of late years have accrued from a study of the cerebro-spinal fluid in the disease have been welcome and valuable aids to the practitioner. These newer laboratory methods of course can never replace accurate observation of the well-known physical signs of the condition, any more than Vidal's test has replaced the employment of physical examination in the case of typhoid fever, but a knowledge of them is of the utmost importance when we have to deal with obscure and doubtful cases.

Before detailing some personal researches it will be well to review very briefly present knowledge concerning the cerebro-spinal fluid in general paralysis, and this is best done, perhaps, by mentioning the various points in the chronological order of their discovery.

The first step in this connexion, and still one of the most important, was the discovery, made in 1900 by Ravault, Vidal, and Sicard, that lymphocytosis in the cerebro-spinal fluid is one of the most constant accompaniments of both general paralysis and tabes. Many forms of cells are found, the most significant being large mononuclear lymphocytes and plasma cells. When in a non-febrile malady the lymphocytosis is very pronounced, then it may be regarded as practically pathognomonic of general paralysis. The cell increase is greater at the onset of the disease—a fact that obviously enhances its value in diagnosis.