THE
Journal
OF
Nervous and Mental Disease.

Original Articles.

A STUDY OF LANDRY'S PARALYSIS; WITH A REPORT OF
THREE NON-FATAL CASES.¹

BY THEODORE DILLER, M.D.,
PITTSBURG.

Ever since Landry, in 1859, gave the account of a patient who exhibited a certain set of symptoms and whose illness ran a certain course, there has been an increasing number of more or less similar cases reported bearing his name. The most generally accepted definition of Landry's paralysis seems to have been that given by Leyden and quoted approvingly by Thomas², that it is a rapidly ascending motor paralysis of sudden onset, with loss of knee-jerks, with slight or no sensory symptoms, without atrophy, loss of control of sphincters, electrical changes, or mental involvement and which runs a rapid course without fever and terminates fatally, usually exhibiting bulbar symptoms near the end. Mills and Spiller³, writing more recently (1898), understand Landry's paralysis to be: "A disease in which rapid loss of motor power usually begins in the lower limbs, and the paralysis is flaccid and associated with paresis and loss of tendon reflexes. The upper extremities are soon involved, and bulbar symptoms develop after a few days. Pain is not a prominent symptom. Death occurs within a week or ten days, though in some cases it may be delayed, and in

¹A paper presented at a meeting of the Pittsburg Academy of Medicine, held Sept. 29, 1902.

others, more rare, recovery may ensue. There may be a descending as well as an ascending form of paralysis. The electrical reactions are normal and patients are perfectly conscious of their condition." Thus it will be seen that a descending type is allowed, non-fatal cases are admitted, and paresthesia is considered a constant symptom although severe or persistent pains are not accounted as belonging to the clinical picture. Absence of marked elevation of temperature and non-involvement of the sphincters are by most writers named as distinguishing marks of the affection although not included in Mills and Spiller's definition. Landry's post-mortem findings were negative as were most of those of the earlier investigators.

The literature of Landry's paralysis has now become quite extensive; and in the several papers by Diller and Meyer⁴, Bailey and Ewing⁵, Mills and Spiller⁶, Thomas⁷, Knapp and Thomas⁸, Taylor and Clark⁹, Raymond¹⁰, Oppenheim¹¹, and Schwab¹², will be found references to almost all the reported cases of this disease up to the year 1901, together with critical studies based upon them. A review of this literature, in a most striking manner, bears witness to the strong reverence which exists in the medical mind for a literary tradition, and the great hinderance which it may prove to a proper study of a disease. After forty years, during which many scores of reports of Landry's paralysis have been made, it remained for Taylor and Clark¹³ to entirely break away from the literary tradition of Landry's paralysis in these forcible words: "On the basis of an acute, clinical, and necessarily highly superficial, pathological observation made many years ago by Landry, subsequent observers, led largely by a name, have been attempting to force into the narrow clinical conception outlined by Landry, various symptoms, none of them fundamental and supported by no constant pathological findings, and still less by a definite etiology. The so-called disease has with each new investigation lost something of its coherence. We have persistently clung to a name and demanded that the symptoms which it represented should conform to our preconceived idea." These views of Taylor and Clark are endorsed by a later writer, Schwab¹⁴, and are to my mind none too strong. Although some recent

writers enlarge upon Landry's original definition and recognize "transitional forms," they have practically accepted the older idea that Landry's paralysis is a disease entity. Even such an acute observer as Dejerine¹⁵, in his recent work, follows Landry in his description almost slavishly, accounting rapid ascending course, loss of reflexes, absence of marked sensory symptoms, absence of fever, preservation of sphincters and a fatal termination as marks of the disease. Schmaus¹⁶, in his book published only last year, adopts the same views.

In the various attempts to study Landry's paralysis, the general plan has been to reject all cases which did not pretty closely conform to the type Landry described, or which did not come to autopsy, while but little attention has been paid to the etiology; and the non-fatal and atypical cases have been thrown out of consideration. If this plan were continued, the original Landry's paralysis clinically would, of course, be preserved despite the variable symptomatology and the conflicting post-mortem findings. Indeed, Goebel¹⁷, writing in 1898, practically makes a plea for this method of study and deplores the tendency to bring many types under the head of Landry's paralysis.

While the various post-mortem studies have added much to our knowledge of Landry's paralysis, if not offering a full explanation of it, and are most praiseworthy, it occurs to me that too little attention has been paid to the clinical side, especially to the study of non-fatal and atypical forms of the affection. For it will no longer do to throw out cases which do not terminate fatally, or which depart from the classic type as described by Landry. It would be as reasonable to establish a pathologic basis by throwing out all findings which do not fit a certain type, as to insist upon such a clinical basis; and the pathologic studies have certainly revealed one thing, *viz.*: that there is no constant anatomic basis for the disease, even for the typical cases.

Let us now look at some of the clinical reports with the view of determining the range of symptomatology of the affection, noting especially those clinical manifestations which represent departures from the older definition of the affection.

Bailey and Ewing, in their elaborate and painstaking study

in 1896, collected all the cases which had, up to that time, been examined post-mortem, 75 in number. But of these 31 were thrown out of consideration either because post-mortem examinations were incomplete or unsatisfactory, or else because the cases were atypical clinically. Cases of Bernhardt, Pitres and Vaillard, Hayem, Pribytkow and Brochain, because of the presence of marked sensory phenomena; the cases of Vierordt, Strümpell, Eichorst, Diller and Meyer, because the course was prolonged or chronic; while Roth's case is excluded because the paralysis was not ascending in type. After this weeding-out process, Bailey and Ewing consider 44 of the 75 collected cases. In 12 out of 32 where the symptom is recorded, the sphincters were involved *i.e.*, in over one-third of the number analyzed. The knee-jerks were lost in 31 cases, diminished in 2 and increased in 2. Of 20 cases the electrical reactions were normal in 19 and lost in only 1. The temperature was normal in 20, while 14 exhibited moderate fever. As to sex there were 29 men and 10 women. In 3 cases the paralysis affected the arms and legs simultaneously; while in the remaining cases it began in the legs; in 4 cases in one leg before the other. There was sensory involvement in 13 cases.

Thus it will be seen that after excluding a large number of cases because they did not fit into the classic clinical type that certain atypical features are present in those admitted, *e.g.*, sensory involvement in one-third and sphincter involvement in more than a third of the cases. In 3 cases the paralysis was not ascending but began in all four members simultaneously.

Thomas¹⁸ reports 2 cases which came to autopsy in both of which degeneration of the anterior horn cells and peripheral nerves was discovered. Thomas holds that Landry's paralysis is an affection of the lower motor neurone, and that the sensory apparatus is uninvolved. He mentions, however, that there was tenderness on pressure in one of his cases, and of the other that the patient's chief complaint on entering the hospital was numbness of the legs.

In Goebel's case, weakness of the vesical sphincter and diminution of sensation are noted as symptoms which appeared late in the disease.

Of Knapp and Thomas¹⁹ three cases only one was fatal and in this case there was some affection of the sphincters and moderate sensory disturbance. In one of the non-fatal cases, there was loss of control over the anal sphincter, sensory disturbances and muscular atrophy. The second non-fatal case also exhibited muscular atrophy.

In a case reported by Burghart²⁰ electrical reactions of degeneration were observed. This patient recovered somewhat, but finally died at the end of 14 days.

In a fatal case recorded by Robinson²¹ numbness of the feet was the first symptom; pain in the back was complained of and the legs were tender when grasped.

In the fatal case recorded by Taylor and Clark²² pains were present for a month before motor symptoms appeared. Pains in the back and dorsal portions of the thighs were at times extreme. Marked fibrillary tremors formed part of the clinical picture. Pressure over the nerve trunks produced no pain. Clinically this case resembles pretty closely one of my own.

Mills and Spiller state, of the case they record, that some weakness was present for a month before the actual symptoms of the disease appeared. Numbness of both hands with some pain and inability to perform finer movements were noted. Power left the arms first.

Worcester²³ mentions loss of sensation in the legs as a symptom in his case. Numbness in the hands with some loss of the finer movements were premonitory symptoms. The patient was mentally apathetic. There was rapid motor and sensory involvement.

Marie and Marinesco's²⁴ patient suffered intense pain over the entire body although a microscopic examination showed the peripheral nerves to be normal.

Hirtz and Lèsné²⁵ describe a case which was at first acute and then chronic, and which terminated fatally at the end of four months. The muscles of the lower extremities were very tender and the seat of severe pains, increased upon movement.

A. Schultz²⁶ records two fatal cases. In the first case, the symptoms were those of acute ascending neuritis. In the second case the symptoms followed multiple neuritis in an alcoholic subject.

A case described by Oettinger and Marinesco²⁷ presented much objective diminution of all forms of sensation.

In a case recorded by Howard²⁸ paresthesia was the first symptom, and tingling and numbness were very distressing. The muscles were affected in the following order: bulbar, hip, thigh, shoulders, arms, legs, hands, feet, labial, diaphragm.

Spiller²⁹ reports a typical case in which improvement set in after two or three weeks and progressed to a complete recovery. Nearnow³⁰ also reports a case in which recovery occurred. Rowden³¹ reports the case of a boy aged 10 years in which the paralysis was of a rapidly descending type beginning with headache, pains in the neck, a temperature of 103, and terminating fatally in a few days.

In Schwab's³² case, which terminated fatally at the end of seven weeks, there was paresthesia in the fingers of the left hand for a week before the onset of the paralytic symptoms. There was marked hyperesthesia of the abdominal walls.

Wappenschmidt³³ reports a case of sudden onset attended with pain which ran a rapidly fatal course. The peripheral nerves were found normal although the clinical signs had pointed to a peripheral neuritis.

In a fatal case reported by Griffith³⁴ in a man aged 32, there were weakness and malaise for a week, then severe pains in the legs followed by a rapidly ascending paralysis which became complete. The diaphragm was paralyzed. There was some blunting of sensibility. The sphincters remained intact.

Paresthesia of the hands and vertigo were for eight days premonitory symptoms in a fatal case reported by Kapper³⁵. Then headache and pains in the legs appeared, followed by rapidly ascending motor paralysis involving the bulb, laryngeal palsy occurring; and death ensued. The knee-jerks were lost. Consciousness was preserved intact to the end. In the beginning of the attack hysteria was considered. Although the cord and peripheral nerves were examined by the Nissl and osmic acid methods no pathologic changes were discovered in them. A bacteriologic examination was likewise negative.

Soltman³⁶ notes a case of Landry's paralysis in an eleven-year-old girl, where recovery occurred following treatment by inunctions.

Stockton³⁷ reports the case of a girl aged 27 (diagnosed acute ascending myelitis), where, following premonitory symptoms consisting of nervousness, insomnia and vague pains, which had existed two weeks, pain in the back and abdomen and anesthesia in the gluteal region developed. Control over the bladder and bowels was lost. Paresthesia in the feet developed along with weakness in the legs which, two days later, were completely paralyzed and anesthetic. The next day the arms were almost completely paralyzed and sensation in the upper part of the body was much impaired. Some movement in the legs now returned. The knee-jerks were absent. The neck muscles became involved and the patient died, being conscious to the end. No autopsy was held. Hematoporphyrinuria was a persistent symptom, although only very little trional had been given.

Maxwell³⁸ records the case of a Chinese woman in which the paralysis was of an ascending type, involving the bulb, and which ran a fatal course in 12 days. Paresthesia in the feet and pains in the legs preceded the motor involvement. The knee-jerks were absent and the urine passed involuntarily. Just before this attack the patient had suffered from diarrhea produced by eating unripe fruit.

In a case examined post-mortem by Wappenschmidt³⁹, the peripheral nerves were found to be normal. The anterior horns and Clarke's columns showed degeneration of increasing intensity from below upwards. Dydynski⁴⁰, on the other hand, holds that Landry's paralysis is a peripheral nerve disease and ascends because it attacks the least vital parts first, these being least resistant to toxic influences. To support his view he points to the effects of curare poisoning upon animals.

Baumstark⁴¹ reports the case of a young man (which he regards as one of ascending acute myelitis) who had contracted malaria in Africa. He suddenly developed weakness in the legs which rapidly increased to complete paralysis. Motor weakness ascended; marked bulbar symptoms appeared. Muscular atrophy, absent knee-jerks, pains along the nerve trunks, produced by pressure, disturbance of sensation, paralysis of the bladder, impaired vision, and peculiar choreiform movements

of the hands were among the symptoms noted. The symptoms all gradually disappeared and recovery occurred. This case is of peculiar interest in that malaria seems to have been the cause, and because of the recovery after such a profound involvement of the nervous system.

At this point I shall introduce the clinical histories of three cases I have observed :

Case I is that of a single man aged twenty-one years, a telegraph operator, of excellent habits. He has never used tobacco or alcohol and denies syphilitic infection. The sisters died of phthisis; otherwise his family history is good. The patient himself had an attack of typhoid fever at the age of 14. Since then, up to the time of his present illness, he has enjoyed excellent health.

Four weeks ago, while telegraphing, he lost some control of his finger-movements in operating his instrument so that he became clumsy and awkward. He thought of writer's cramp. Four or five days later he became weak in the legs so that his knees often gave away in walking. At the same time his thighs and legs, especially the latter, felt numb. A week later the same numbness appeared in his arms, hands, and abdomen. He felt as though a band were about his waist. He now saw double at times—always when looking to the right or left or downwards. He also became dizzy on sudden movement. There had been no pain or fever at any time.

Examination on January 31, 1902. Stripped, he exhibits no atrophy. His gait is slow, labored, one of weakness. The grasp of the hands is feeble. There is no objective disorder of sensation; but he complains of paresthesia in arms, legs and abdomen. There is no pain upon pressure over the nerve trunks. The knee-jerks and Achilles jerks are absent. The abdominal reflex and wrist-jerk are present. The electrical reactions of the muscles of the arms and legs are normal. Eyes: The pupils are 3 mm wide, equal, and respond promptly to light and in accommodation. The eye-grounds are normal. In conjugate movements there is some muscular deficiency noticeable especially in movement to the right. No squint is apparent. The urine is normal.

He was observed only once; but a month later he was seen walking on the street. His sister stated that he took great aversion to some mutton he ate two weeks before the appearance of the first symptoms; said it was tainted and had spoken much about it.

Case II.—A perfectly healthy boy, aged 7½ years, went to

bed in his usual health. The next morning he complained of soreness in his legs and thighs. But little attention was paid his ailment and he attended school as usual. The pains or soreness continued; and three days later appeared in the arms and shoulders. At the same time it was noted that he had difficulty in walking. He was unable to return from school and had to be carried home. Very soon afterwards loss of power appeared in the arms; and three days after the onset of the paralysis he was unable to stand alone and his hand-grasp had become almost nil. He had insufficient strength to shut a door, but was still able to grasp a pencil firmly enough to write his name. He could not move his toes. His condition remained thus for about one week and he then began to improve and has steadily continued to do so ever since. There was no fever at any time; no mental, bulbar or sphincter symptoms. The pains complained of were never severe. He spoke rather of soreness and never cried because of it.

Examination, April 22, 1902 (five weeks after the onset of the trouble). The boy has a waddling, non-spastic gait. The grasp of the hands show about one-half the normal strength. There is no apparent wasting of the muscles (father notes none). The calf and thigh muscles are firm; but the arm and shoulder muscles are flaccid, soft and exhibit R. D. The leg and thigh muscles exhibit partial R. D. Sensation is unimpaired. The knee-jerks are present although somewhat sluggish. On deep pressure there is moderate tenderness in the arms and legs. The boy is quite bright mentally. Since the examination the boy has steadily improved, but is not yet well.

Case III.—A single man, aged twenty-five years, born in Ireland, a laborer. Family history negative. The patient had always been a hard worker and enjoyed good health.

The latter part of October, 1901, three weeks after coitus, a single, hard, non-painful sore appeared on the head of the penis. Following this he had a sore throat which lasted seven weeks. He also noted swelling of the glands in the groin. No skin eruption appeared. About the middle of November (three weeks after the appearance of the chancre), he was seized with severe pains in the legs; and 24 hours later with pains in the lower part of the back; and 48 hours later, with pains in the arms. These pains have persisted, night and day ever since. He had no pains in the trunk or head at any time. The pains have been more severe and more persistent in the legs than in the arms. He describes them as "steady," "sharp," "burning" pains. They do not leave or return suddenly, but are more or less constant, but always worse in the mornings. Along with the pains, he complained of tire, weariness, loss of strength.

From the time of the onset of his symptoms up to the date of his admission to the Mercy Hospital, January 23, a period of about nine weeks, he lost 23 pounds in weight. The pains have kept him awake much at night.

Examination, February 1.—There is general emaciation. A dusky reddish color of the skin over the arms and legs is noted. The pallor produced by finger pressure disappears very slowly. Fibrillary twitchings in the face, arms and legs are present. The twitchings are most marked in the face about the mouth and chin. Mentally the patient is quite clear, but gloomy as to the outcome of his trouble. The grasp of his hands is pretty fair, but probably much below what is normal for him. The legs exhibit as much or rather more weakness than the arms; but he can still stand for a short time on one foot alone. There is no swelling or pain about any of his joints. Deep pressure over the great nerve trunks produces no pain. There are no cranial nerve palsies. The pupils are equal (3 mm) and react promptly to light. There is no diplopia. The tongue is clean. Both knee-jerks are very quick, but make a rather limited excursion.

There is general glandular enlargement, the epitroclear, inguinal, and post-cervical glands being clearly palpable. A careful examination of the thoracic and abdominal organs by Dr. Johnson was negative. The pulse is rapid (100). There is no fever; and there has been none since he entered the hospital. A blood examination revealed the following: White cells, 10,720; red cells, 5,100,000; hemoglobin, 85 per cent. The patient has, at all times, had control over his bladder and bowels.

March 3.—The pains are considerably less in severity but still quite troublesome. He is more emaciated than he was a month ago. The bony points about the pelvis have become very prominent; and there are two or three small shallow bed-sores over the sacrum. He walks in a feeble, laborious fashion with his legs partially flexed. The knee-jerks are now practically absent, the right responding but faintly. Achilles jerks are also absent. Deep pressure over the nerve-trunks and passive joint movements elicit no pain. The fibrillary twitchings especially in the legs are more marked than at the previous examination. [He had been walking about the ward just before I came and states that this always increases the twitchings in his legs.] On several occasions during the past month he has been unable to empty his bladder.

By way of treatment he received iodide of potassium, hydrotherapy, and remedies for the relief of pain and to promote diuresis.

All three of these cases are atypical and I dare say there are those who would hesitate or decline to classify them as Landry's paralysis, and yet all of them, I believe, exhibit a sufficient number of the marks of that affection to be so designated. Moreover, the clinical pictures which they present could not well be labeled by the name of any other disease.

These three cases are alike in that they all exhibited a rapidly ascending motor paralysis without elevation of temperature or mental symptoms. In all of them sensory symptoms were present, which in two of the cases preceded the motor symptoms; none had a fatal issue.

Case I is noteworthy inasmuch as the course of the affection was comparatively mild, the patient probably never having been off his feet.

The second case must, I think, be admitted to be a fairly typical one. Here, after three days of soreness in the legs and thighs loss of muscular power suddenly developed in these members, followed very shortly by similar involvement of the arms. This rapidly developed until it became almost complete; remained thus stationary for a week and then rapidly improved. Two interesting features of this case are the preservation of the knee-jerks, although diminished, and the presence of complete and partial reactions of degeneration in the affected muscles five weeks after the onset of the trouble. In neither of these cases was the cause of the disease clear.

The third case, on the other hand, is striking in that it seems almost certain that syphilis was the etiologic factor to which the disease was chargeable. In this case the sensory symptoms at first overshadowed the motor, making it seem like an ascending sensory rather than ascending motor disease. But along with the sensory involvement there were also motor symptoms, although in the background at first. It is to be especially noted that the involvement was rapid and ascending in type. Other points of interest in the case are the great and progressive muscular wasting; marked fibrillary twitchings involving the entire body, including the face; the, at first, exaggerated and afterwards absent knee-jerks; the vaso-motor disturbances; the slight involvement of the

sphincters; absence of fever; apparent absence of blood-changes; absence of mental symptoms; absence of bulbar involvement; the preservation of sufficient strength to walk and give a fairly firm hand-grasp; the absence of pain on pressure; the long and chronic course of the disease.

In one of the cases described by Landry there was a history of syphilis; and the possibility of syphilis as an etiological factor is considered by him. Nonne⁴² believes that it is improbable that syphilis can produce the symptom-complex of Landry's paralysis and leave behind no anatomical changes. Kussmaul, Heubner, Zambaco, Gros and Lancereaux⁴³ describe cases where the Landry symptom-complex developed during the early stages of syphilis without prodromal symptoms or signs of meningitis. Alexander⁴⁴ mentions a case with syphilitic history, where, following ocular palsy and optic atrophy, Landry's paralysis developed and terminated in death.

All these cases were reported years ago; and Nonne⁴⁵ observes that in recent literature the views of Heubner as to the existence of a syphilitic form of Landry's paralysis are not confirmed, although he admits that here and there a case may be found with a syphilitic history, as, for example, Goebel's⁴⁶, he holds the reverse is the rule. Nonne's contention on this point, may, I think, be allowed, and yet it seems to me that it must be admitted that occasionally syphilis is the etiological factor. In no case I have been able to find does this seem so clear as in the third case I have recorded in this paper. Fr. Schultze, however, reports a fatal case of acute ascending atrophic paralysis⁴⁷ with bulbar symptoms in which the onset occurred five months after a syphilitic infection. Schultze himself does not insist that syphilis was the etiologic factor, although other factors were not apparent.

Brissaud⁴⁸ records the case of a man aged thirty-three who, following violent pains in the lumbar region, was seized with a rapidly ascending motor paralysis, marked bulbar symptoms and loss of control of the sphincters. Sensation was unaltered. Atrophy did not appear. The knee-jerks were lost but afterwards reappeared. The electric reactions were normal. Slight fever was present. The patient made a rapid recovery.

A case reported by Vizioli⁴⁹ most of all resembles my third case. A man aged 65 was infected with syphilis at the age of 21. but not thoroughly treated. The disease began with severe pains followed by motor weakness, first in one hand but afterwards involving the entire musculature, accompanied by pains and fibrillary twitchings and followed by progressive atrophy. Reactions of degeneration were present. The symptoms differed from those seen in the Duchenne-Aran muscular atrophy in that pain was a prominent symptom and that paralysis preceded atrophy.

Nonne⁵⁰ reports two cases of progressive anterior poliomyelitis of a descending type. In both the sphincter function was disturbed and paresthesia was present, points which convinced him that the morbid process was not confined to the anterior gray horns. In both cases the disease was arrested after much atrophy had occurred, following a vigorous anti-syphilitic treatment. In both cases obvious signs of syphilis were present.

Summing up now the evidence afforded by this review of the literature and the study of my own cases, it seems that in the symptomatology of Landry's paralysis sensory symptoms are almost constantly present and generally precede the motor paralysis by several hours or a day, occasionally by much longer periods. The most common sensory disturbance is paresthesia, but hyperesthesia, tenderness and spontaneous pain, which may be severe, are not rare. The motor paralysis is usually of an ascending type, but may be of the descending variety or begin in all four extremities simultaneously. As a rule muscular wasting does not occur and electrical reactions of degeneration are absent, but well-marked exceptions to this rule occur. In a few cases fibrillary twitchings are recorded. The sphincters are involved in about one-third of the cases. The four most constant marks of the disease are rapidly ascending motor paralysis, lost knee-jerks, absence of marked fever and preservation of the mental integrity; but occasional exceptions occur. Oppenheim⁵¹ observes that cases corresponding to the original Landry type are easily separated from other clinical types, but that the only absolute criterion for all cases is a flaccid paralysis

affecting in rapid succession the muscles from below upwards or rarely, from above downwards.

But considering the actual clinical records of reported cases, admitting the atypical and "transitional forms," it would seem that we have in Landry's paralysis a symptomatology which stands midway between multiple neuritis on the one hand and poliomyelitis with bulbar paralysis on the other hand, and which at both extremes of its types touches and indeed overlaps the clinical picture of these two affections. Most of the cases of the disease are, however, clearly distinguishable from either multiple neuritis or poliomyelitis. Moreover, the morbid anatomy of Landry's paralysis, like the clinical picture, reveals features common to both multiple neuritis and poliomyelitis; but it reveals changes not common to either, and in some cases carefully examined by the later histologic methods no morbid changes were found.

The various writers on Landry's paralysis are almost a unit in saying that it is due to an infection or toxemia; and this view seems to be founded upon three facts chiefly: (1) the acute onset and rapid course; (2) the presence of bacteria found in a certain number of cases examined post-mortem; (3) the frequent enlargement of the spleen and lymph glands.

In the earlier reported cases collected by Bailey and Ewing a considerable number showed no post-mortem changes. But in the later reports of autopsies examined by the means of the present day exact histologic technique morbid changes of greater or less extent have been pretty generally recorded. Bacteriologic findings have not been so frequent as histologic changes. In some cases the pathologic changes recorded have been so slight as to scarcely afford a reasonable explanation of the symptoms. Oppenheim⁵³ gives the prudent caution that the finer the technique, the greater should be our caution in accepting the findings revealed by it. But even when examined by the most approved modern methods by competent investigators there are a certain number of cases in which the post-mortem and bacteriologic findings were negative. In a carefully examined case reported by Girardeau and Levi⁵⁴ and A. Schultze⁵⁵ in 1898 and Kapper⁵⁶ in 1900, the post-mor-

tem findings were negative; and in the carefully reported case by Goebel⁵⁷ the peripheral nerves, spinal gray matter, anterior and posterior nerve roots were normal; only a light grade of degeneration of pyramidal tracts high up was discovered, insufficient, in the opinion of the writer, to account for the symptoms.

The various post-mortem findings recorded need not at this time be rehearsed since they have been sufficiently analyzed by recent writers. Suffice it to say that they have been most manifold both in degree, situation and kind. Disseminated inflammatory areas in the bulb were found by Ormerod, Boinet, Courmont, while others (Eisenlohr, Schultz, Ketli, Halva and Immerman) have found similar changes in the cord. A great many cases are recorded in which the changes were in the anterior horns of the cord. (Bickel, Taylor and Clark, Worcester, Hirtz and Lesne, Wappenschmidt.) In many others they were in the peripheral nerves (Barth, Dejerine, Bois, Dydynski, Eisenlohr); while in a still larger number both the anterior horns and the peripheral nerves were involved (Centanni, Eisenlohr, Pribykow, Thomas, Knapp and Thomas, Boinet, Mills and Spiller, Krewer, Schultz). In Goebel's case some fresh degeneration of the muscles was detected, although the peripheral nerves and the gray matter of the cord were normal. In a few cases (Diller and Meyer, Goebel, Bickel), changes were found in the white matter of the cord.

A most interesting seat of morbid changes, in view of the constant presence of sensory symptoms, is the posterior nerve roots. Changes in these structures are recorded by Knapp and Thomas, and Boinet. In view of the prominence of sensory symptoms in Landry's paralysis it is much to be regretted that there are very few records of examination of the posterior spinal nerve ganglia. Finally the frequent records of enlargement of the spleen and lymphatic glands and pulmonary hemorrhages must be borne in mind.

The nature of the morbid changes are manifold, representing many forms and degrees of degeneration, extravasation, hemorrhage and inflammation.

While bacteriologic examinations have not so frequently as

histologic examinations yielded positive results, still there are now on record a considerable number of cases in which micro-organisms have been found post-mortem. The streptococcus, a diplococcus and staphylococcus have been found in the central nervous system. Piccinno found bacteria in the perivascular lymph spaces; Courmont and Bonne a diplococcus in the cerebro-spinal fluid. Remlinger⁵⁹ produced acute ascending paralysis in rabbits by injections of the streptococcus and recovered the organisms from the cord.

The etiology is one of the least studied, least understood, and at the same time most important phases of the subject. Bailey and Ewing mention as etiologic factors typhoid fever, syphilis, diphtheria, exposure and over-exertion; while in the great majority of cases analyzed by them (26), there was no definite infection. Various authors state that the disease has followed pneumonia, whooping-cough, small-pox, influenza and septicemia. In several cases alcohol was thought to have been the cause. Oppenheim⁵⁸ mentions one case in which the disease followed the kick of a horse who was affected by septicemia. In no case does the etiology seem quite so clear as in one of my own cases recorded in this paper where the disease began a few weeks after the patient had contracted a chancre. In Baumstark's⁵⁹ cases malaria was the apparent cause.

There are three views regarding Landry's paralysis at which it may be worth while to glance.

One set of observers have regarded it as nothing more than a form of multiple neuritis. (Dejerine, Barth, Ross, Putnam, Walton, Dydynski, Krewer.) These observers point to the frequently reported changes in the peripheral nerves, and explain the changes occurring in the spinal gray matter on the theory that these are merely parts of the same trophic and anatomic unit, and liken them to the changes which occur in typical multiple neuritis. Against this view may be urged the inconstancy of these morbid changes, the rather frequent cases in which the peripheral nerves exhibited no changes, the fact that the peripheral nerve changes found consist usually of degeneration, seldom of inflammation; and finally that the clinical course of the disease differs considerably from that of true multiple neuritis.

Bernhardt and Westphal concluded that Landry's paralysis is very like acute poliomyelitis, differing from it chiefly because it is more generalized over the gray matter of the cord. Bailey and Ewing include six cases of so-called poliomyelitis in their list of analyzed cases, because of the wide extent of involvement and the presence of bulbar involvement. This view does not sufficiently account for the ascending character of the disease or the frequent sensory symptoms. Cases followed by complete recovery also afford an argument against it.

The third and most recent view of Landry's paralysis, is that supported by Thomas and Knapp, and Thomas, and Mills and Spiller: that it is primarily an affection of the motor neurone of the first order, a parenchymatous degeneration of toxic or infectious origin. There is much to support this view of the morbid anatomy of the disease in the consideration that the most constant pathologic changes are those found in the anterior horns of the cord, and that such a theory offers a reasonable explanation of the clinical symptoms recorded in many of the cases. But arguing against this view is the post-mortem record made by Knapp and Thomas themselves, of degeneration in the posterior nerve roots, a similar record made by Boinet, the case of Schwab where the only change was great vascular congestion, the cord and peripheral nerves being normal, and the cases with negative post-mortem findings.

To these three views might be added the view maintained by Taylor and Clark⁶⁰ and supported by Schwab, that there is no such "disease" as Landry's paralysis; that the cases described as such exhibit "no essential constancy in the clinical symptoms nor in the pathologic findings, and the etiology is wholly vague."

While it is quite true that the symptomatology, morbid anatomy and etiology of Landry's paralysis are inconstant and variable, and that it is not a disease in the sense in which tetanus or small-pox or exophthalmic goiter are diseases, it is at least as much of a "disease" as some other so-called "diseases," *e.g.*, cerebral palsy of childhood.

The term Landry's paralysis cannot be dropped. It has been used too long to be erased from our nomenclature. Nor

is it, in my opinion, desirable that it should be dropped; but it is most desirable that the older conception of its meaning be enlarged to include this highly striking group of cases, variable though they be, standing both in symptomatology and pathology midway between multiple neuritis and poliomyelitis, and which cannot be placed under either of these headings. It must be borne in mind that these cases exist, and if the name of Landry's paralysis were discarded a name would have to be invented under which to include them. No harm can come of the continued use of the term Landry's paralysis if the variability in the etiology, symptomatology and morbid changes and bacteriologic findings be borne in mind, and if we at the same time freely admit the contention of Taylor and Clark, that in the strict sense of the word it is not a "disease" but rather a group of symptoms, overlapping each other more or less, and which are toxic infections in origin, produced by a great variety of poisonous agents.

The accumulated evidence points very strongly to the toxic origin of Landry's paralysis; and with equal certainty it indicates that it is produced by no one single agent but by many. Multiple neuritis may be here briefly glanced at. This disease is produced by traumata, toxic agents, and slow degeneration changes. Among the toxic agents may be mentioned lead, arsenic, copper, carbon monoxid, the toxins of diphtheria, influenza, malaria, typhoid fever, rheumatism, etc. The symptomatology of multiple neuritis varies greatly, depending upon the nature and potency of the toxic agent, and the resistance or vulnerability of the tissues. The poisons of lead and diphtheria possess peculiar selective action. Similarly the symptomatology of cerebro-spinal meningitis varies enormously; and the disease is doubtless susceptible of being produced by several varieties of toxins, perhaps by many. Putnam⁶¹ has well said: "When the nervous system is working in a perfectly normal manner and not under strain it seems physiologically a unit. When the reverse is true it becomes evident it is comprised of many parts of varying powers of resistance to disease."

For a definition of Landry's paralysis I would suggest the

following: Landry's paralysis is an infection or toxemia produced by various poisons in which the spinal cord (including the bulb) and the peripheral nerves, and especially the lower motor neurone, are attacked, and in which parenchymatous degeneration, hemorrhagic and inflammatory exudates of various kinds and degree, are usually found post-mortem in this portion of the nervous system. But in a few cases no post-mortem changes are discoverable, and in still others changes which are inadequate to explain the symptoms. The spleen and lymph glands are usually enlarged. Bacteria of various kinds have, in a considerable number of cases, been found in the central nervous system; but in the great majority of cases a careful examination has failed to discover any microorganisms. The male sex is affected much more commonly than the female. No age is exempt.

The first symptom of the disease is usually paresthesia, sometimes accompanied by pain in the members which are subsequently paralyzed, and this may exist from a few hours to several days or even longer as a premonitory symptom. General malaise, neurasthenic symptoms and ataxic movements may also be premonitory symptoms. Widespread flaccid paralysis rapidly develops involving legs, trunk and arms, and, in fatal cases, the muscles innervated from the bulb. The paralysis is usually of an ascending type involving the legs first, but it is occasionally of a descending type and may involve all extremities simultaneously or attack the various muscles in an irregular order. In the majority of cases the sphincters are intact. Their involvement however is not rare. The knee-jerks are absent. As a rule atrophy, fibrillary twitchings and electrical changes in the muscles are absent; but any or all of these phenomena may be present. Pain and tenderness to pressure may be prominent symptoms, but as a rule are not very marked, although they are present in some degree in the majority of cases. As a rule the temperature is normal or but slightly elevated. Consciousness is almost invariably preserved unimpaired throughout the attack.

Cases which terminate fatally run a course of from a few days to one or two weeks, and almost always exhibit bulbar

symptoms. In cases which do not terminate fatally the course may extend over many weeks or even months, and recovery may be incomplete. Often premonitory symptoms exist for several days or even weeks.

While the prognosis is grave, a considerable number of cases terminate in recovery or partial recovery.

The treatment should be directed to the accomplishment of two ends: the support and stimulation of the vitality and the production of elimination. To this end hypodermic injections of strychnia and the use of whiskey, digitalis, mild diuretics and cathartics are to be recommended. Anodynes may be required when pain is severe. In cases in which syphilis has been recently contracted, vigorous specific treatment should at once be instituted. Later, after a turn for the better has occurred, massage, electricity and hydrotherapy may aid the tendency to recovery.

Note.—Since the above was written an important article on "Poliomyelitis in the Adult," by Dr. E. W. Taylor, has appeared in the August (1902) number of this journal, in which the clinical histories of six cases are given. In Taylor's first case in which the clinical manifestations pretty closely corresponded to the Landry symptom-complex, a careful post-mortem examination showed evidence of primary inflammation of the ventral horns of the cord, and secondary destruction of the nerve cells, and marked degeneration of the peripheral nerves.

Taylor reiterates his objection to the term Landry's paralysis and observes that "Poliomyelitis is none the less poliomyelitis because it simulates in its clinical course what Landry, many years ago, described as acute ascending spinal paralysis." This contention may be readily allowed. But all cases of so-called Landry's paralysis can not be shown to be cases of poliomyelitis; many more closely correspond to multiple neuritis. I can see in Taylor's argument no reason for changing the views I have expressed in this paper.

I would suggest that the term Landry's paralysis be employed precisely as most neurologists employ the term apoplexy. Under this term is included both cerebral embolism and hemorrhage, and also those rarer and more or less obscure conditions

which may produce apoplectic symptoms. When we feel sure that an apoplexy is hemorrhagic in character, we speak of cerebral hemorrhage; but since we see many cases in which the differential diagnosis between cerebral hemorrhage and embolism is uncertain, the more general term apoplexy becomes convenient.

Similarly the term Landry's paralysis may be conveniently retained to designate cases of a certain clinical type, some of which can be made out to be cases of poliomyelitis, others of multiple neuritis. But the term, like apoplexy, is especially useful to designate those cases which cannot be made out to be either cases of multiple neuritis or poliomyelitis and those cases in which the post-mortem findings are negative.

With advancing knowledge the terms apoplexy and Landry's paralysis may be discarded, but that day is not yet.

²Amer. Jour. Med. Sc., August, 1898.

³JR. NERV. AND MENT. DIS., 1898, p. 369.

⁴Amer. Jr. Med. Sc., April, 1896.

⁵N. Y. Med. Jr., July 4-11, 1896.

⁶JR. NERV. AND MENT. DIS., June, 1898.

⁷Amer. Jr. Med. Sc., Aug., 1898.

⁸JR. NERV. AND MENT. DIS., Feb., 1900.

⁹*Ibid.*, April, 1900.

¹⁰"Leçons sur les maladies du système nerveux," Deuxieme Serie.

¹¹"Lehrbuch der Nervenkrankheiten," Zweite Auflage p. 397, et seq.

¹²JR. NERV. AND MENT. DIS., Dec., 1900.

¹³*Op. Cit.* p. 192.

¹⁴*Op. Cit.*

¹⁵"Sémiologie du système nerveux," p. 559.

¹⁶"Vorlesungen über die path. Anat. des Rückenmarks," 1901, p. 295.

¹⁷Neurolog. Centralblatt, April 1, 1898.

¹⁸*Op. Cit.*

¹⁹*Op. Cit.*

²⁰Quoted by Knapp and Thomas, *op. cit.*

²¹Med. Rec. Vol. XLVII., p. 534.

²²*Op. Cit.*

²³JR. NERV. AND MENT. DIS., May, 1898.

²⁴Bull et Mémoires de la Société Méd. des Hôpitaux de Paris, 1895, p. 659.

²⁵La Presse Médicale, 5, 1897, p. 269.

²⁶Abstracted in JR. NERV. AND MENT. DIS., Vol. 26, p. 239.

²⁷Bull. et Mémoires de la Société Méd. des Hôpitaux de Paris, 1895,

p. 63.

²⁸Brit. Med. Jr. I, 1898, p. 1,654.

²⁹JR. NERV. AND MENT. DIS., 1901.

³⁰Obozrenjepish, 1899, No. 2, p. 151 (Abstracted in JR. NERV. AND MENT. DIS.).

³¹Brit. Med. Jr., May 4, 1901.

- ⁸²Jr. NERV. AND MENT. DIS., Dec., 1900.
- ⁸³Deutsche Zeitschr. f. Nervenhk. Bd. 16, H 3-4, p. 305.
- ⁸⁴Lancet, 1900, Vol. II., p. 1490.
- ⁸⁵Wiener klin. Wochenschr., 1900, No. 1.
- ⁸⁶Jahr. f. Kinderhk. Bd. 51, H. I., p. 67.
- ⁸⁷Amer. Jr. Med. Sc. Vol. 120, p. 36.
- ⁸⁸St. Bartholomew Hospital reports. Vol. 36, 1900, p. 137.
- ⁸⁹Inaug. Dissert. München.
- ⁹⁰Jahresbericht über Neurolog. Psych. 1900, p. 406.
- ⁹¹Berlin klin. Woch. 1900. No. 37 and 38.
- ⁹²"Syphilis und Nervensystem," p. 297.
- ⁹³Quoted by Nonne. *Op. Cit.*
- ⁹⁴"Syphilis und Auge." Wiesbaden, 1889. Quoted by Nonne.
- ⁹⁵*Op. Cit.*
- ⁹⁶*Op. Cit.*
- ⁹⁷Berlin klin. Wochenschr., 1883. No. 39. Quoted by Nonne.
- ⁹⁸Rev. Neurologique. Apr. 30, 1902, p. 355.
- ⁹⁹Ann. di. nevrol. Abstracted in Neurolog. Centralbl., 1900, p. 79.
- ¹⁰⁰*Op. Cit.*
- ¹⁰¹"Lehrbuch der Nervenkrankheiten," p. 401.
- ¹⁰²"Lehrbuch der Nervenkrankheiten," p. 400.
- ¹⁰³Rev. Neurologique, Oct. 1898.
- ¹⁰⁴*Op. Cit.*
- ¹⁰⁵Wien. klin. Wochenschr. 1900, No. 7.
- ¹⁰⁶*Op. Cit.*
- ¹⁰⁷La Semaine Méd., July 15, 1897.
- ¹⁰⁸*Op. Cit.*
- ¹⁰⁹Berl. klin. Woch., 1900. No. 37 and 38.
- ¹¹⁰*Op. Cit.*
- ¹¹¹*Op. Cit.*, p. 230.