of the tegmentum (*fontaineartige Haubenkreuzung* of Meynert), and having crossed the raphe lie in a separate bundle (in the cat), which runs just ventral from the fasciculus longitudinalis medialis, and is called by Tschermak the "predorsal longitudinal bundle." In human beings the fibres are probably mixed with those of the fasciculus longitudinalis medialis. Collaterals are given off to the eye muscle nuclei and to the gray matter of the formatio reticularis, but the main bundle runs down in the cord, where it occupies the anterior part of the fissural portion of the anterior funiculus. It becomes exhausted by giving off collaterals and terminals to the anterior horn of the same side, and partly by sending axones through the anterior white commissure to the contralateral anterior horn. Some of the fibres extend down as far as the lower part of the lumbar cord. It seems very probable that a part at least of Loewenthal's marginal fasciculus is identical with this system of fibres.

(4) *Pontospinal or Metencephalospinal Neurone System, and* (5) *Myelencephalospinal Neurone System.*—In the medulla and pons are situated groups of perikaryons which give off axones that run down to the spinal cord. Most of these are as yet very imperfectly understood. There is evidence, however, that some of the cells of the nuclei pontis and of the nuclei arcuati give off such axones. If so, we are justified in speaking of pontospinal neurone systems. A good deal of work has been done upon the descending fibre systems in the formatio reticularis—fibre systems which have their origin in perikaryons of the formatio reticularis grisea, specially in the inferior, middle, and superior central and lateral nuclei. Axones from the cells of the nucleus centralis medius run downward in the fasciculus longitudinalis medialis to reach the anterior funiculus of the cord, where they run close to the fissura mediana anterior. This is an uncrossed descending spinal neurone system from the formatio reticularis. Another fibre system from the central nucleus descends in the opposite lateral funiculus of the spinal cord occupying an area in the dorso-lateral region of the lateral pyramidal tract, medial from the rubrospinal fibre system and the direct cerebellar tract.

(6) *Olivospinal Neurone System.*—I have fully described this system on pages 954–958 of my book on "The Nervous System." The fibre system is usually known as Helweg's path, though it was described earlier by Paul Meyer. Its development has been studied by von Bechterew, who designates it the *Olivenstrang*. The path is seen as a triangular area in cross-section; hence Helweg's term (*Dreikantenbahn*). The cells of origin may be in the inferior olivary nucleus, or they may come from higher regions. Golgi studies are still lacking except for meagre statements in von Kölliker's text-book.

(7) *Vestibulospinal Neurone Systems.*—The cell bodies which give rise to the medullated axones of this path are situated chiefly in the nucleus nervi vestibuli lateralis (Deiters), though some of them may be situated in the nucleus nervi vestibuli superior of von Bechterew. The axones pass ventralward from Deiters' nucleus into the formatio reticularis alba and descend. On their way down they pass through the area situated between the nuclei laterales and the remains of the anterior horn. In the spinal cord they lie in the peripheral parts of the zone of exit of the anterior root fibres, and in the lateral portion of the funiculus anterior. Some of the fibres of this system descend as far as the lumbar cord. The axones appear to terminate by running in to end among the cells of the anterior horn of the same side. This neurone system is under the influence of impulses from the vestibular nerve on the one side and from the cerebellum (nucleus fastigii) on the other. Its influence on the cord is probably concerned with the coordination of the axis of the body and orientation in space.

(8) *Cerebellospinal Neurone System.*—Though a vast amount of work has been done with the idea of determining the fibre systems which throw the spinal cord directly under the domain of the cerebellum without the intermediation of any relay station, it must be confessed that our knowledge upon this subject is still, in reality, most meagre. Marchi early described a fibre system in the spinal cord which degenerated after removal of the cerebellar hemisphere. He thought that the fibres came chiefly from the vermis, partly from the hemisphere, that they passed through the brachium pontis, and then by way of the ground bundles into the anterior and lateral funiculi of the cord. He could follow the degeneration throughout the whole length of the cord. In cross-sections this degeneration occupied two areas, one extending along the periphery of the cord from the fissura mediana anterior to the anterior extremity of the direct cerebellar tract, that is, in an area corresponding to the *zone sulcomarginale* of Marie; the other, a more lateral area, situated just in front of the lateral pyramidal tract. It was his opinion that the fibres ran in to end in the anterior horn of the spinal cord. This degeneration, described by Marchi, probably corresponds, at least in part, to the so-called cerebellar tract of Loewenthal.

Ferrier and Turner dispute Marchi's conclusions, inasmuch as they found that when one hemisphere of the cerebellum is extirpated without injury to neighboring parts, no degeneration can be traced to the cord, though degenerated fibres can be followed through the corpus restiforme to the inferior olivary nuclei and the nuclei of the posterior funiculi. When the vermis is extirpated alone, the fibres which run to the nucleus nervi vestibuli lateralis of Deiters degenerate. It is their opinion that the degeneration, described by Marchi, occurs only when Deiters' nucleus is injured in the attempt at cerebellar extirpation. This view is supported also by the experimental work of Risien Russell and by that of Mott.

Marchi has, however, found a supporter in Biedl, who reproduced a degeneration of the fibre systems, described by Marchi, by cutting the corpus restiforme. He thinks, however, that Marchi's tract reaches the cord by way of the inferior cerebellar peduncle rather than by way of the middle cerebellar peduncle, as Marchi asserted.

The experimental work of Thomas tends to support Marchi's observation as far as the fibres in the anterior funiculus are concerned. He thinks that the fibres arise from cells in the nucleus dentatus, and that they pass through the superior vestibular nucleus of von Bechterew and the lateral vestibular nucleus of Deiters into the formatio reticularis, and thence into the anterior and lateral funiculus of the cord. His work was done chiefly upon dogs. Extirpation of the cortex of the cerebellum alone does not cause the degeneration; it is necessary to injure the nucleus dentatus cerebelli. If Deiters' and Bechterew's nuclei are also injured, the degeneration in the cord is much more extensive.

Ramón y Cajal has made an important contribution to the neurone system under discussion by the application of Golgi's method. He finds that the axones of the cells in the nucleus dentatus, passing out in the brachium conjunctivum, give off descending limbs of bifurcation which run to the anterolateral fasciculus of the cord. In his opinion it is these axones which correspond to the descending cerebellar path of Marchi. Ramón y Cajal emphasizes the fact that in frontal sections of the guinea-pig's brain, stained by the method of Weigert-Pal, this cerebellospinal path from the brachium conjunctivum can be clearly seen as isolated bundles. (See Fig. 439 on page 430 of his "Histologia del Sistema Nervioso de los Vertebrados.")

It may be regarded as certain, therefore, that a direct uncrossed cerebrospinal neurone system exists, the cell bodies of which are situated in the nucleus dentatus (and perhaps in the neighboring gray nuclei), the medullated axones of which go through the anterolateral white matter to the anterior horn of the cord. It would appear, too, that the fibre system, making up the spinal portion of this path, or a part of it, is really an offshoot or by-path from the main ascending fibre system of the brachium conjunctivum. The function of the path is doubtless coordinative. It seems probable that it carries to the anterior horn cells at least a part of those

cerebellar impulses which maintain the equilibrium of the body.

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See also references under preceding heading.

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SPINAL-CORD DISEASES: ACUTE ASCENDING PARALYSIS.—(Synonym: Landry's Paralysis.) In 1859 Landry described the symptom complex which has since then been known both as Landry's paralysis and as acute ascending palsy. The clinical picture he portrayed was the following: The sudden development in a previously healthy individual of a flaccid paralysis of the legs, which generally began first in one and then within a few hours or a day affected the other. When the paralysis of the legs had become complete, the trunk muscles became paralyzed, and then in the space of a few days the arms were involved in like manner. Next there was involvement of the deglutition, articulation, and respiratory musculature, and in a short time death followed from asphyxiation. There are milder cases, however, in which there is restitution of function. Landry described prodromes, such as general malaise and paræsthesia in the extremities, and during the course of the illness slight disturbances of sensation. He laid particular stress on the absence of muscular atrophy, the presence of normal electrical reactions, and negative findings at the autopsies.

Since Landry's original cases were published, many cases, either similar to his or varying from the type in one essential or another, have been reported, until to-day our conception of the disease has broadened so as to include cases exhibiting a wider range of symptoms; so that, as Oppenheim says, the only essential symptom necessary to the disease is the rapid development of a flaccid paralysis, commencing in the lower extremities and affecting successively the arm and the bulbar nerves, though in rare cases the order of involvement is reversed.* In the literature under this heading have been included many cases which undoubtedly belong to other diseases, principally acute anterior poliomyelitis and multiple neuritis.

ETIOLOGY.—The disease is rather uncommon in its occurrence. Men are affected more often than women, and as a rule between the ages of twenty and forty, though instances of its occurrence in children and old people have been observed.

The view most generally held as to the etiological factor of the disease is that it is due either to some form of toxæmia or to bacterial infection. Landry himself was under the impression that it was caused by poisoning, and this opinion was accepted by many subsequent observers, but especially by Westphal. The following facts Diller regards as pointing in this direction: First, the presence in some cases of enlargement of the spleen and lymph glands, hemorrhagic spots in the lungs and intestines, and albuminuria; second, the finding of bacteria in some of the cases. The varieties found include anthrax, typhoid, and other unclassified bacilli, streptococcus, varieties of staphylococci, diplococci, and the Fränkel pneumococcus. No specific form of microorganism has been isolated, but the disease seems to be capable of development after diphtheria, typhoid, variola, anthrax, pneumonia, pertussis, the puerperium, malaria, and probably also upon septicæmia as a basis. Oppenheim thinks it doubtful whether the entrance of the micro-organisms themselves into the spinal cord, medulla, and peripheral nerves can produce the disease, but he believes that probably their toxins injure the motor tracts in such a manner as to cause paralysis, without as a rule producing recognizable lesions. Thoinot and Masseline and Remmlinger have produced in rabbits symptoms similar to Landry's paralysis by the injection of micro-organisms into the circulation.

*In recent years some authors have regarded Landry's paralysis as being merely a symptom complex due to various causes and not a disease or clinical entity with a definite pathological basis.

One case has been reported in which the disease followed a kick from a horse affected with septicæmia.

Alcohol has been mentioned as an etiological factor in some cases, and exposure to cold and overexertion likewise. Syphilis was formerly regarded as being a not uncommon cause of the disease, though if the question be looked at in the light of our present knowledge of syphilis and its pathology, it seems highly improbable, as Nonné points out, that it could cause the paralysis and yet leave no evident lesion.

Pathological Anatomy.—Landry postulated as one of the characteristics of acute ascending paralysis the absence of any lesion in the spinal cord, and as a matter of fact in many of the earlier reported cases no changes were found. Since then, however, through our better technique and in the light of our greater knowledge of the nervous system and its pathology, morbid changes in the spinal cord have been found in many cases. The lesions reported are very numerous and varied in their nature, and do not show any considerable uniformity. Disseminated areas of inflammation have been observed in the medulla oblongata by Omerod and Prince, while Eisenlohr, Schultze, Ketli, and others have found similar changes in the spinal cord. In some cases the axis-cylinder processes in the antero-lateral columns of the cord were swollen; in others there were changes of varying intensity in the anterior horns, similar to those found in poliomyelitis. One case showed only a degeneration in the anterior roots, while in another there was a focus of myelitis.

In addition to these changes in the cord itself the peripheral nerves have been found to be affected. Sometimes the peripheral neuritis has been the only lesion found, and this has led one set of observers (Dejerine, Barth, Ross, Putnam, and others) to regard it as the characteristic morbid condition of Landry's palsy. They explain the alterations in the anterior horn cells, when they occur in conjunction with peripheral neuritis, as due to trophic disturbances. Dejerine and Thomas, in their recent work on spinal-cord diseases, go so far as to say that it is a disease of the peripheral neurones, and is poliomyelitis when the cell body is affected and polyneuritis when the axis-cylinder processes are implicated. Diller states, as an argument against this theory, that the cases in which there is no peripheral neuritis are frequent, and that the changes found in the nerves are usually those of degeneration, not of inflammation.

The spleen is usually acutely enlarged, the lymphatic glands swollen, and in some cases there is acute degeneration of the viscera.

Oppenheim sums up our knowledge of the pathology of Landry's paralysis by saying that the changes in the spinal cord, especially in its gray matter, are the predominant ones; and that they sometimes consist of inflammatory conditions and vascular disturbances (disease of vessels, hemorrhage, exudation, thrombosis, softening, and infiltration); sometimes of changes in the nerve cells, as described by Marinesco and by Bailey and Ewing.

Symptomatology.—The disease commences suddenly with paralysis of the extremities, or it is preceded by prodromes. These latter consist of a chill or chilly sensations, slight fever, pain in the back and in the extremities, and paræsthesia, more rarely hyperæsthesia and general malaise.

Pain and general weakness may occur fully a month before the actual onset of the disease. The paræsthesia is usually in the fingers or toes, and may be very marked and distressing.

The *paralysis* as a rule marks the beginning of the disease. It is generally ascending in type, that is, it affects the lower extremities first. The feet are first affected, either both at once or successively. Then the legs, thighs, the lumbar, abdominal, and chest muscles are successively involved; next in order are the muscles of the hand, forearm, and arm, then those of the neck, and finally there occurs paralysis of the muscles innervated by the bulbar nerves, viz., those of speech, deglutition, and respiration.

The paralysis manifests itself first as a weakness in one or both legs, then becomes complete and flaccid, there being no spastic phenomena. The paraplegia can develop in a day, and the involvement of the other muscles takes place within eight or ten days, rarely in three or four.

Cases have been described in which there has been a descending paralysis, and also all four extremities have been affected at the same time. These cases are rare. Westphal reported a case in which only bulbar symptoms appeared. Cases have been reported in which the cranial nerves were involved, the paralysis affecting the muscles controlled by the facial, the eye muscles and those of accommodation; as a rule, however, these nerves are not involved. The pulse rate is usually increased in frequency because of the affection of the vagus, and attacks of asphyxia due to paralysis of the respiratory muscles cause death.

The *electrical reaction* is generally prompt and normal, especially in those cases which run a short course. Sometimes there are quantitative variations in the electrical reaction, and the reaction of degeneration has also occurred. Oppenheim noted the following peculiar electric reaction in one of his cases: it was impossible to increase the intensity of contractions by increasing the strength of the current used. Examination of a piece of this muscle showed waxy degeneration.

The absence of muscular atrophy * was regarded as one of the characteristics of the disease, and in the majority of cases this holds true. Some authors have regarded its development in the later stages of the disease, when it runs a protracted course, as being due to inactivity. But there have been undoubted cases of Landry's paralysis in which the wasting of the muscles was an early occurrence. Fibrillary twitchings are sometimes seen.

Sensibility is usually but slightly disturbed. Landry described the presence of slight changes. Bailey and Ewing describe thirteen cases of sensory involvement out of the forty-four genuine cases which they found in literature up to 1896. As a rule, marked disturbance is absent. Hypæsthesia in the extremities, and more rarely hyperæsthesia, have been described. In some instances delayed transmission of pain or temperature sense, or failure to discriminate between pain and touch sense, and diminution in the acuteness of the muscular sense, have been observed. Trophic disturbances are not present.

Patellar and superficial reflexes are lost, though they may be present for some time in the early stages of the disease. As a rule the bladder and rectum are unaffected. Cases have been reported in which either one of the sphincters has been weakened or paralyzed. Sometimes at the onset there may be temporary retention of urine, or later, when the abdominal muscles are paralyzed, there may be constipation.

It is characteristic of Landry's paralysis that there is either no rise of temperature, or only a very slight rise, throughout the course of the disease. Regarding this, again, exceptions have been reported. Sweating is said by some to occur frequently. Albuminuria generally occurs, and in one case there was hæmatoporphyruria.

The sensorium remains almost invariably clear until the end. Delirium has been present in those cases which have fever.

Prognosis.—The prognosis is grave. Most of the cases end fatally. The disease may terminate abruptly at the end of two or three days, death being due to bulbar paralysis; as a rule, however, the duration of the disease is prolonged to eight days before the onset of bulbar symptoms. The disease may assume a subacute character and last for two months or more. Some of the cases have terminated in complete or partial recovery. Oppenheim regards it as a sign of favorable outcome when there is a remission in the bulbar symptoms. Generally the paralysis first disappears in the part which was

* What has been said of the electrical reaction can with equal truth be said of the occurrence of muscular atrophy.

first affected. Convalescence may last for many months. Remissions and relapses with a fatal termination are possible.

Therapy.—There is no therapeutic agent which is specific in its action. The application of the actual cautery to the back has been recommended. In cases in which syphilis is suspected vigorous mercurial and iodide treatment should be used. Electricity, especially galvanism, has been used. The chief attention of the physician should be directed to the nourishment and stimulation of the patient during the acute stage; and then, if he survive, an effort should be made to restore the functions of the paralyzed parts by means of hydrotherapy, massage, electricity, nutritious food, change of scenery, etc.

Israel Strauss.

SPINAL-CORD DISEASES: ACUTE SPINAL MENINGITIS.—The membranes of the spinal cord consist of an outer dense fibrous sheath, the dura mater, and an inner delicate vascular sheath, the pia mater. The web-like fibres connecting these two, and known as the arachnoid, may be conveniently reckoned together with the pia mater. The spinal dura is not adherent to the inner surface of the bones forming the vertebral canal. Between the periosteum and this membrane is a loose connective tissue of interlaced bundles with delicate slender elastic fibres and spindle-shaped connective-tissue cells, and within the meshes of this tissue there is a greater or less amount of fat, which is often gelatinous in appearance. The dura mater itself is a dense white sheath composed of bundles of white fibrous and of elastic tissue in about equal numbers, running lengthwise. Both outer and inner surfaces are covered with endothelium, and between the meshes are lymph spaces which open upon these surfaces, thus establishing a connection between the outer surface of the dura and the space between dura and pia. Binding the dura to the pia mater are the delicate fibres which form the arachnoid, fibres of connective tissue bound together into lamellæ surrounded by very fine elastic fibres and covered with endothelial cells. The outer layer of the pia is of the same structure as the arachnoid, the inner is separated from this by the pial capillaries and vessels, which, surrounded by prolongations of the fibrous and elastic reticulum composing the inner layer, pass into the substance of the cord. Golden-brown pigment cells with branching processes are found in this layer, especially in the cervical region, where they may lend a brown color to the membrane. Both membranes are supplied with nerves, the pia more abundantly. The lymphatics of the pia are continuous with those of the cord.

The inflammation of the membranes of the cord may affect the outer membrane, the inner, or both. Internal meningitis, or leptomeningitis, always extends to the arachnoid also, although an effort has been made by some authors to distinguish a pure arachnitis, so called, without involvement of the pia. External meningitis or pachymeningitis also usually extends to the pia-arachnoid, at least in acute inflammations; the chronic processes may remain limited to one or the other. As the symptoms in all forms of acute meningitis are very similar and certain symptoms are common to all, it will simplify matters to describe first in full detail the symptoms of the typical variety, acute purulent leptomeningitis, and then, in the less important varieties, the symptoms will not need to be described in full again.

I. *Acute purulent leptomeningitis, acute cerebro-spinal meningitis, acute cerebro-spinal fever, epidemic cerebro-spinal meningitis.*

A. Hirsch has given an excellent historical survey of this disease ("Die Meningitis Cerebro-spin. epidemica," Berlin, 1866). The first well-authenticated epidemic appeared in France among the soldiers of the army in 1805, and was followed by sixty-two epidemics in that country, forty-eight of which began in the army. The disease first appeared in epidemic form in the United States probably in 1806, and from then on until 1830 there were frequent outbreaks in this country, and seven years later in France again, then throughout Europe. The latest epidemic which has been carefully studied was that in Boston in 1896–97, to the report of which by Councilman, Mallory, and Wright we shall have occasion to refer frequently. The later epidemics have usually been less severe than the earlier, and show less tendency to develop among the standing armies.

Winter and spring seem the seasons most favorable for the development of an epidemic. It is not a disease of cities; some of the severest epidemics have been in country districts. Children are not as susceptible as young adults; the disease is rare before the first year. The mode in which infection takes place is not known; but the nasopharyngeal passage is supposed to be the place of entrance for the micro-organisms. Apparently it is not contagious, for it is rare to find more than one case in the same family. Insanitary surroundings, overcrowding, exhausting exertion, exposure, etc., seem to aid in its spread. There are isolated sporadic cases, probably of the same character as those occurring in the course of epidemics, which are scattered along in the intervals between outbreaks.

Bacteriology.—The causative agent of cerebro-spinal meningitis is still in question. The diplococcus intracellularis meningitidis or meningococcus intracellularis of Weichselbaum, the pneumococcus of Fraenkel, the streptococcus meningitidis of Bonome, the meningococcus of Foà, the streptococcus pyogenes and the staphylococcus pyogenes, have all been isolated by different observers. The colon bacillus has been stated to be the etiological factor in cerebro-spinal meningitis of the new-born.

Weichselbaum found the organism which bears his name in six out of eight cases examined by him in 1887. These six cases were all primary. In the other two cases, which were secondary to pneumonia, he isolated the pneumococcus only. His results, corroborated by Goldschmidt and by Edler, were more strongly confirmed by Jäger, who, in 1895, found the organism in twelve cases during an epidemic. Councilman also looks upon the diplococcus intracellularis as the cause of the epidemic form, and lays great stress on this fact from the point of view of both diagnosis and prognosis, inasmuch as, in his opinion, all cases are fatal except those caused by this diplococcus. When the pneumococcus or the streptococcus was found there was associated with the meningitis some other lesion, in lungs or heart usually, and none of these cases recovered. Sporadic cases are probably not caused by the Weichselbaum organism. Councilman's conclusions are based upon the largest number of bacteriological examinations ever made by one observer —thirty-eight positive results from fifty-five lumbar punctures, thirty-one positive results from thirty-five autopsies.

On the other hand, Fraenkel isolated the pneumococcus which bears his name from the pia-arachnoid in spinal meningitis, and his results have been confirmed many times. Wolff claims that it is present in forty-two per cent. of all cases. Flexner and Barker found this organism during the Maryland epidemic of 1892.

The streptococcus has been isolated, and in other cases the staphylococcus pyogenes also. Among some bacteriologists (as Bordoni-Uffreduzzi) the opinion prevails that all of these different cocci are varieties of the same organism—the pneumococcus of Fraenkel.

The pathology of cerebro-spinal meningitis is that of an extensive purulent inflammation, with infiltration of the subarachnoid space. The cerebro-spinal fluid is increased and usually turbid with floccules of fibrin in the early stages; later on, it becomes purulent. This increase in the fluid causes bulging of the dura, especially posteriorly, perhaps because of the patient's recumbent position. The fluid, according to Councilman, contains many leucocytes, but not abundant fibrin except in the cases caused by the pneumococcus. Flexner found numerous large phagocytic cells in the fluid from one of his cases. The small pial vessels are enormously distended, the leucocytes being collected along their walls and in the perivascular lymph spaces. The inner surface of the