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A CASE OF SO-CALLED LANDRY'S PARALYSIS. WITH  
AUTOPSY.<sup>1</sup>

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Under the name of "Paralysie ascendante aigue" (Acute Ascending Paralysis) Landry in 1859 described a group of symptoms which has ever since been known by his name. Clinically the disease is characterized by a prodromal stage more or less marked, during which paresthesia, headache, malaise, etc., can be noted. Following this there develops an acute flaccid paralysis, involving first, as a rule, the lower extremities, then the upper and trunk muscles, and lastly the muscles supplied from the medulla. Sensation and sphincteric control are usually retained. The paralyzed muscles, although flaccid, show no evidence of atrophy and retain the normal electrical reaction. There is, as a rule, fever, but the sensorium remains clear unless the fever is excessive. The patient usually dies from paralysis of the respiratory muscles under the picture of suffocation. Landry himself could find no anatomical basis for this group of symptoms, and for a long time this negative finding was considered to be characteristic of the disease. In recent years, however, more careful study has shown a variety of pathological conditions. The variation in the clinical picture, to some extent, and the very dissimilar post-mortem findings, have led to considerable confusion in regard to this disease, so that to-day there is a well-marked tendency to do away with the

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<sup>1</sup>Read before the St. Louis Medical Science Club, June 12, 1900.

term Landry's paralysis, as indicative of a definite morbid type with a constant etiology, pathology, and symptomatology; and to use the term, if at all, as descriptive of a symptom-complex, the cause of which is at present unknown, the nature of which is problematical, and the pathology of which varies with almost every investigator.

In spite of this, however, as Strümpell says, the clinical picture of this disease is such a remarkable one that it is worthy of a generalized description, and it appears in all text-books as such. A very excellent summary of the present conception of the disease is given in Mills and Spiller's<sup>2</sup> article, an abstract of which, I think, will make this point clear. They say, first: The usual picture of Landry's paralysis is that of an ascending flaccid paralysis with little sensory disturbance, normal electrical reaction and retention of sphincteric control; its course is rapid and generally fatal. Second, there may be forms which depart from this, which resemble polyneuritis and myelitis to such a degree as to make the diagnosis difficult. Third, in some cases there may be no post-mortem findings, but these cases have probably been insufficiently examined. It may be also that in some cases death takes place so quickly that changes in the nervous system have not had time enough to develop. Fourth, Landry's paralysis may be classed as a myelitis. Fifth, a polyneuritis may exist. Anterior horn cell changes are found. It is difficult to determine whether they are primary or secondary in nature. Sixth, in some cases it is probable that the whole peripheral neurone is affected by the toxic process.

I shall not attempt in this paper to discuss the clinical aspects of the case from the standpoints of diagnosis, differential diagnosis, or treatment. This has been done often before, and very thoroughly. In Oppenheim's and Strümpell's textbooks will be found a very excellent clinical description as well as a summary of the post-mortem findings.

I wish, however, to call attention to a few considerations in regard to the etiology, which are well illustrated in the

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<sup>2</sup>"On Landry's Paralysis with Report of a Case." JOURNAL OF NERVOUS AND MENTAL DISEASE, June, 1898.

case which forms the subject of this paper. From the very beginning, Landry himself believed that the process was toxic in nature, for the reasons that the spleen was found enlarged, albumin was found in the urine, lymphangitis existed, and hemorrhagic foci were found in the lungs and other organs. Cases were then reported where this clinical picture followed, or was coincident with diphtheria, influenza, septicemia, etc. There is little doubt at present that in many instances, if not in every one, the etiological factor that is most significant is a toxin, differing according to the agent in question. Whether the organism penetrates into the spinal cord and medulla, and the toxin originated there produces its effect, or whether there is a generalized toxemia, affecting, for some reason not yet clear, the central nervous system, is as yet undetermined. One other point I wish to emphasize, and that is that there are some cases of peripheral neuritis, or rather multiple neuritis which run a fatal course, and have been confounded with the Landry symptoms. These cases should be classed as a neuritis and not as an ascending paralysis at all, for of course in a neuritis there are usually electrical changes in the muscles and nerves, whereas in Landry's paralysis none are present.

I am indebted for the material in this case, as also for the very complete clinical history, to Dr. F. Taussig, of the St. Louis Female Hospital, to whom I wish to express my thanks and appreciation.

Julia S., age 21 years, married, was admitted to the Female Hospital on November 16, 1899, with the provisional diagnosis of anemia. She gave the following history of her trouble: Born of healthy parentage, without any hereditary taint of any kind, she had not, previous to the present time, any serious illness except an attack of pneumonia. There was no specific history. Of the three children, however, to whom the patient had given birth, one was still-born, and two died immediately after delivery. The patient had not menstruated for seven months before entering the hospital, and had lost considerable weight. She said that about six weeks ago she noticed a tingling and sense of numbness in the fingers of the left hand; she did not, however, attach any importance to this. About one week later the same sensations were experienced in the fingers of the

right hand, and then spread up both forearms to the elbows. There was at this time no symptom of paralysis. Shortly afterwards the patient, by degrees, found herself unable to use her lower extremities, and for four weeks was confined to bed. Two weeks before entering the hospital, the patient was also partially unable to use her hands and forearms; there was no history of any general constitutional symptoms. When she was examined she appeared rather anemic but only slightly emaciated. She seemed rather dull mentally, answering questions slowly, but without any impediment of speech. Her disposition was however quite cheerful, in spite of her paralysis, and a tendency to insomnia was present. She ate heartily and was never nauseated. Her bowels were usually costive, but of late she had not been able to control fecal movements, so that when her bowels were loose, she had an involuntary passage. The same was true of her urination. Her incontinence was accompanied by burning on micturition, but no other abnormality. Cough had persisted for some time, but was not at all severe; expectoration was scanty. Her pupils reacted to accommodation and light; tongue was flabby and coated white, but without tremor or deviation. Chest, fairly well developed; lungs apparently normal on auscultation and percussion. Apex beat in fifth interspace; sounds clear and distinct. Lungs, spleen, liver, normal. Hyperesthesia of the abdominal wall was very marked, especially in the lower half. Vaginal examination was not made. Extremities: Some inability to move upper extremities, more marked on left side. Grip of both hands very weak, especially left one. Lower extremities absolutely flaccid and paralyzed. Knee-jerk absent on both sides. No sensory disturbance of pain, tactile sense or muscular sense in any of the extremities. No trophic changes in skin or marked atrophy of muscles. Urine analysis: Specific gravity 1023, acid reaction, light amber color, no albumin, no sugar. Treatment: Strychnine, iron and iodide of potash.

During the first nine days that the patient was in the hospital, no marked change was observed. The incontinence of urine and feces persisted, and she complained of some pain in the upper extremities. Her appetite remained good. On the tenth day she said she was feeling worse, saying that she had a spell during the night when she could hardly catch her breath. This attack was rather severe but of short duration. No more attacks appeared during that day, but she complained of feeling weak. The following morning at six o'clock she told the nurse that she was feeling better, and ate

a hearty breakfast. At eight o'clock she was suddenly seized with severe dyspnea. She was found to be cyanotic and gasping for breath, but with a comparatively strong, regular, and infrequent pulse. Atropine 1-120th of a grain was given hypodermically but at 8.40 o'clock she died.

Autopsy: Considerable emaciation; rigor mortis not set in. Left pupil slightly larger than the right. Scaling epidermis about the calves. No edema. Chest: Pleuritic adhesions on right side and laterally, also posteriorly on right side to diaphragm; no adhesions on left side. Right lung showed two nodules, caseous in nature, in the apex, about the size of a large pea; similar nodule in left apex. Lungs otherwise normal. Heart showed liquid blood in cavities, no blood clots. Heart muscle cuts soft and is friable; some fatty degeneration; valves all normal. Aorta shows spots of hyaline degeneration. Abdomen: Very little adipose tissue in abdominal wall and omentum. Spleen slightly enlarged; capsule not tense, but substance very soft. Kidneys show congestion; pyramids prominent; cortical markings pronounced; capsules not adherent. Liver congested with dark blood, but not otherwise abnormal. Intestines show no signs of inflammation externally, but lymph follicles in ileum somewhat enlarged and reddened. Two round worms found in the intestine. Uterus retroflexed, ligaments very lax, no signs of inflammation. Ovaries and tubes normal.

This very complete history is typical enough to class the disease under the symptom-complex described by Landry. The diagnosis need not detain us, as the important point at issue is not the name of the disease, but what condition can produce such a group of symptoms as is reported in this history. To be remembered in the post-mortem findings are the condition of the spleen, the old tubercular lesions in the lungs, the pleurisy, and the acute hyperemic kidney. These are directly in line with the assumption of the toxic etiology of the disease, which Oppenheim insists upon very strongly.

The brain and cord, macroscopically, showed nothing abnormal. Upon section of the cord evidences of marked hyperemia could be seen; so intense was this in fact, that it was thought at first that there might be here an acute myelitic process. Upon closer examination it was discovered that this condition depended, as far as could be determined by the naked eye, upon a tremendous capillary engorgement

of the whole cord and meninges. Microscopic examination showed the correctness of this assumption.

The macroscopic section of the brain showed two areas of softening, bilateral in distribution, involving the internal capsule at about the level of the lenticular nucleus. This was regarded as due to an injury to the brain in removing it from the skull. No evidence of meningitis was found, the membranes being somewhat thickened, but everywhere free. No other gross lesions could be demonstrated. The cord and portions of the brain, medulla and cerebellum were placed in 10 per cent. formol and in Müller's fluid. The sections of the cord and medulla were placed alternately in Müller's fluid and formol, so that Nissl's stain could be employed on sections approximately from the same level. A portion of the left sciatic nerve was also placed in Müller's fluid, for examination for possible neuritis. The result of these examinations will be given later.

In examining the literature of Landry's paralysis, I have noted only those cases in which a post-mortem examination is given. I shall not attempt in this paper to give a complete bibliography, but merely to indicate the post-mortem findings in the cases recorded in the last three years and so compare them with the findings in this case.

Boinet,<sup>3</sup> in a typical case of Landry's paralysis, with death, similar to this one, found vacuolization of the nerve cells in the cord and thickened spinal meninges. Sciatic and median nerve showed evidences of a degenerative neuritis. A few scattered bacilli were found in the cord.

Mills and Spiller<sup>4</sup> found evidences of polyneuritis. In many ganglion cells of the anterior horns throughout the whole spinal cord, Nissl stain showed central chromatolysis and migration of the nucleus. The question in this case was whether the pathological process in the cells or in the peripheral nerves was to be regarded as the primary source of the condition.

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<sup>3</sup>"Un cas de paralysie de Landry." *Gazette des Hôpitaux*, 1899. Ref. *Neurologisches Centralblatt*, Feb. 15, 1900. No. 4.

<sup>4</sup>*JOURNAL OF NERVOUS AND MENTAL DISEASE*, 1898, No. 6.

Goebel,<sup>5</sup> in a paper read before the Aertzliche Verein in Hamburg, in January, 1898, entitled "Zur pathologische Anatomie der Landry'sche Paralyse," brings out very clearly the point that was before indicated in regard to neuritis, especially the neuritis acutissima progressiva of Eichorst. He says that our conception of Landry's paralysis should be limited to those cases which conform to the original description of Landry himself. In this case the following pathological changes were noted: Peripheral nerves normal. In the cauda equina some bundles of nerves in the neighborhood of hyperemic blood vessels were found degenerated. There was an increase of interstitial substance without an increase of nuclei. By Marehi's method, slight degeneration was found from the decussation in the medulla to the oculomotor nucleus. The spinal cord was mostly intact. Bacterial examination of the spinal cord and sections stained for organisms were negative. Nissl's stain showed no certain changes.

Giraudeau et Leopold Lévi.<sup>6</sup> Careful examination of the spinal cord with Nissl's stain showed absolute normal cells. Peripheral nerves and anterior roots were also normal. Medulla cells also intact.

F. von Reucz.<sup>7</sup> Hyperemia of the whole central nervous system. Nissl's stain: degeneration of the motor cells of the anterior horns and cells of the motor nuclei of the medulla. Slight degeneration of the spinal cord fibers. The vessels of the pia showed round-cell infiltration and hypertrophy of the intima. No bacteria. The vessels seemed similar to those found in endarteritis syphilitica.

L. B. Wilson and J. L. Rothrock.<sup>8</sup> Bacteriological examination negative. Slight congestion of the blood vessels of the cerebellum and cerebrum. Walls of the large vessels of the medulla infiltrated with leucocytes. Many nerve cells were found swollen, with absence of granular structure and migration of nucleus. No degeneration of the fibers in the

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<sup>5</sup>Ref. *Neurologisches Centralblatt*, April 1, 1898.

<sup>6</sup>*Revue Neurologique*, p. 669, 1898.

<sup>7</sup>*Charité Annalen*, Vol. 23, p. 317.

<sup>8</sup>*Phil. Medical Journal*, p. 1181, 1898.

medulla or cord. Median, intercostal, and sciatic nerves degenerated. The process was to be regarded as a parenchymatous ascending poliomyelitis, which originated centrally and extra-vascularly. The peripheral nerve changes were to be regarded as secondary.

W. L. Worcester<sup>9</sup> found in the large cortical cells migration of the nucleus and swelling. The medulla was normal. A few cells in the lumbar region were pathologically changed; the majority of cells normal. Slight varicose swelling of the peripheral nerves.

J. W. Thomas.<sup>10</sup> Acute inflammatory exudate of the anterior horns; parenchymatous degeneration of the ganglion cells with their dendrites; infiltration of the perivascular spaces of the anterior horns; slight infiltration of the posterior horns around the blood vessels and in the white matter; slight degeneration of the fibers. Anterior and posterior nerve roots were degenerated. Micro-organisms were not demonstrated either in sections or by culture. Second case: Degeneration of all peripheral nerves examined; motor cells of the anterior horns stained by Nissl show degeneration; other cells intact; no micro-organisms.

Mills and Spiller.<sup>11</sup> Marked hyperemia of the whole central nervous system; myeline degeneration of the peripheral nerves; nerve cells of the anterior horn swollen, with migration of the nucleus and chromatolysis; Clarke cells normal; no micro-organisms.

Piccinno F.<sup>12</sup> Vesicular degeneration involving the nucleus and cell protoplasm; bacteria found, not only in the vessels and perivascular spaces, but also in the protoplasm of the nerve cells. These organisms were oval cocci, found mostly in groups, and sometimes in short chains.

Hertz and Lesne.<sup>13</sup> Thickening and infiltration of vessel walls; peripheral nerves intact; ganglion cell changes dependent upon the condition of the vessels.

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<sup>9</sup>JOURNAL OF NERVOUS AND MENTAL DISEASE, Vol. XXV, No. 5.

<sup>10</sup>Am. Journal of Medical Science, viii, 1898.

<sup>11</sup>*Loc. cit.*

<sup>12</sup>"Su di un caso de Paralisi Landry." Ann. d Neurologia, vol. 15, No. 1, 1897.

<sup>13</sup>"Paralysie ascendante aigue experimentale." Société biolog., 23, No. x.



Renlinger.<sup>14</sup> A coccus isolated from a case of septi-cemia; pure culture obtained and injected into rabbits. Acute paralysis developed, followed by death in five days, from paralysis of respiration. Cultures from the cord gave the original cocci; hyperemia of the cord, especially in the lumbar region and the gray substance.

Courmont et Bonne.<sup>15</sup> Peripheral nerves normal; vacuolization of the anterior cells; hyaline degeneration; nucleus of the hypoglossus affected, bacteriological examination of the cord and cerebro-spinal fluid showed the presence of a diplococcus, similar to the streptococcus.

Knapp and Thomas.<sup>16</sup> Marked dilatation of the blood vessels, but without hemorrhagic extravasation; degeneration of the nerve roots by Marchi's method; diffuse degeneration of the white substance of the cord; anterior cells by Nissl show few normal ones, and in some of them no nucleus or nucleolus is found; posterior cells show few changes; fatty degeneration of the sciatic nerve; bacteriological examination negative.

I shall not attempt to critically analyze in any detail these findings. Knapp and Thomas in the article referred to have done this very successfully. On account of the multiplicity of the microscopic reports the task of presenting a clear résumé of them is very difficult. In a general way they may be summed up as follows:

In a few cases organisms have been demonstrated in the cord and have been cultivated from it, or from the peripheral nerves or spinal fluid. These organisms show no constancy, and from an etiological standpoint they can have at present no definite significance. In a considerable number of cases the peripheral nerves have shown evidence of a neuritis. The general opinion seems to be that this neuritis is secondary to the process in the cord, and bears no definite similarity to a polyneuritis, or not enough, at any rate, to

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<sup>14</sup> "Paralysie ascendante aigue." *Presse médicale*, 12, iv, p. 209.

<sup>15</sup> *Archives de Neurologie*, Nov., 1899, pp. 354-373.

<sup>16</sup> "Landry's Paralysis." *JOURNAL OF NERVOUS AND MENTAL DISEASE*. Feb., 1900, No. 2.

regard Landry's paralysis as a variety of neuritis. In a majority of all cases very definite and significant changes have been found in the nerve cells of the cord, mostly localized in the anterior horns. These changes point, without doubt, to a pathological process in these cells. The cells of the posterior horn have, as a rule, been little affected. The changes in the nerve cell are the changes that we see from acute inflammatory processes affecting the central nervous system, such as are found in acute myelitis, epidemic meningitis, etc. The most constant finding is perhaps the hyperemic condition of the cord and membranes.

Evidences of degeneration of the white matter of the cord are not constant enough to be important. A few cases have shown no abnormal changes at all, and in a few cases, together with abnormal changes in the cord elsewhere, the nerve cells have been found normal. The absence of acute inflammatory processes, in the way of cell infiltration or softening, is very significant. The most definite statement in regard to the pathogenesis and pathology of the disease is found in Knapp and Thomas's<sup>17</sup> article, which is that the process is an acute parenchymatous degeneration involving the peripheral motor neurones, arising from some toxic or infectious cause. Mills and Spiller came independently to the same conclusion.

This is certainly very strong evidence, and it is with considerable hesitation that I am forced, as a result of a study of my specimens, to adopt another view, and to regard the process, in this case at least, as primarily an interstitial one, which finds its expression principally in the abnormal condition of the blood vessels.

Sections of the cord, medulla, cerebellum, and of various portions of the brain cortex were stained by the methods of Nissl, Weigert, Pal, Van Gieson and with picro-carmin, hemalaun, etc. The sciatic nerve by Van Gieson, carmine and Weigert. It is to be regretted that it was impossible to make use of Marchi's method. The result of my findings may be tabulated as follows:

<sup>17</sup> *Loc. cit.*

Nerve Cells; Anterior horns; Nissl staining: For the most part normal in staining qualities and in shape. Nissl bodies show the usual character. Nucleus and nucleolus normal. Cell outline regular and dendrites can be followed the usual distance. The number and size of the cells appear normal. Some cells show pigment. Here and there possibly one cell in five shows slight chromatolysis, mostly central in variety. Vacuolization of the nucleolus occasionally observed. No irregularities of nuclear membrane. In a very few scattered instances migration of nucleus and total disappearance of the same were found. It is to be noted that the Nissl stain was made as freshly as possible and studied at once. After a lapse of almost two months, they were again studied, and it was noted that the pigment was much increased.

Cells of the posterior horns including Clarke's cells: Nissl staining. Most of the cells show the usual normal picture; in a few instances the usual accidental chromatolysis or slight variations from the normal was observed.

Medulla, cortex, cerebellum. Nissl staining: Cells show no definite pathological changes, being very similar to the condition found in the cord. The cells of the nuclei of the cranial nerves seem normal. It was observed, however, that the cells of the hypoglossus nucleus seemed to show more variation from the normal than the others. Nerve cells from all these regions were also studied in carmine and in Van Gieson preparations. The impression of their normal condition, as demonstrated by the Nissl stain, was strengthened.

Peripheral nerve: The only peripheral nerve that was saved for examination was a portion of the left sciatic. This was found to be normal in every particular.

Anterior and posterior nerve roots: No pathological changes were found in the nerve fibers.

Meninges: They were found everywhere very hyperemic and, in places, thickened, though not adherent. The blood vessels from the pia as they entered the white substance of the cord were much more tortuous and more numerous than normal. In many places the vessels were congested with blood, which was shown very clearly by the Van Gieson method.

Cord, as a whole: No evidence of degeneration in the white matter of the cord was found either by the Weigert stain or by its various modifications, all of which were used. One exception to this was found in a few sections in the upper dorsal region, where slight V-shaped degeneration, with the point of the V anteriorly, could be followed for a short distance. This was regarded as of little importance, being probably due to faulty sectioning. No evidence of degeneration was found in the white matter of the medulla, pons, cruræ, cortex, or cerebellum. I wish to mention here that, as the Marchi method was not used, this point cannot be regarded as definitely settled in this case, as very possibly there may have been degenerated areas in the cord too delicate to be demonstrated by the methods employed.

Blood vessels: As it appears to me that the primary and most important changes in this case were found in the blood vessels, I shall attempt to describe their condition in some detail. The intense and generalized hyperemia of the whole cord was touched upon in the macroscopic description of the specimens in the beginning of the paper. The reason for this is apparent in the sections studied under the microscope. The blood vessels are thickened and tortuous, and their ramifications can be followed out to an extraordinary degree. At first sight their number seemed to be increased (*Gefässwucherung*), but it is very probable that, owing to the enormously increased injection of the vessels, more of the minute capillary branches are made visible by the stain than would otherwise be the case. The injection of the blood vessels is as marked in the posterior as in the anterior portion of the cord, and between the white and the gray matter no general difference could be observed. The vessels in the cerebellum, medulla, pons, and to a very slight extent the vessels of the cerebrum, showed the same condition of active hyperemia. It was very marked in the medulla and cerebellum. The vessels cut crosswise or longitudinally were found filled with blood, the same condition being found also in meningeal vessels and the vessels of the anterior and posterior nerve roots. The caliber of many of the vessels was enormously increased, so

that in the posterior nerve root, for example, a blood vessel occupied one-third of the space in a section of the root. In many places, apparently anomalous vessels were to be found, for instance in a number of sections from the dorsal cord the vessel running into the posterior commissure gave off a large-size branch about one-third of the way down anteriorly, the branch taking a diagonal direction towards the lateral column. Vessels of large caliber were seen in the neighborhood of the central canal surrounded by a clear space. Many of these vessels were of striking size.

**Hemorrhage:** Free blood was found everywhere in the cord for the most part in the form of small punctate hemorrhages, but sometimes of considerable size. The hemorrhagic foci were of greater size in the gray matter than in the white, and were more frequent in the upper regions of the cord than in the lower. The medulla, in the neighborhood of the hypoglossus and vagus nuclei, showed the presence of numerous hemorrhages. Most of the hemorrhages were of quite recent date, the form and arrangement of the red corpuscles being retained. Others were evidently older, and in a few places pigment granules from degenerated corpuscles were present.

**Vessel walls:** A careful examination of the vessel walls was made for the purpose of determining the presence of degeneration. Although some vessels, especially those in the neighborhood of the anterior or posterior fissures, showed slightly thickened walls, the majority showed the reverse condition; the walls being thinned by the tremendous pressure of the overfilled blood vessels. No evidence of degeneration of the vessel walls could be found. No increase of nuclei and no endothelial infiltration could be observed. The perivascular lymph spaces were found filled with blood, and slight evidence here of an endothelial proliferation was seen.

To sum up these findings: Nerve cells were found normal, or at any rate the slight variation found in them could be explained by preagonal or post-mortem chromatolysis. Absence of degeneration in cord. Absence of neuritis. Absence of myelitic process, of softening or purulent inflammation, and of meningitic process. The positive findings were limited to the

blood vessels and the perivascular lymph spaces alone; these consisted in a tremendous vascular congestion, a thinning of the vessel walls, due to this increased pressure; and hemorrhages with escape of free blood into the nervous structure, and with the possible increase in the number of vessels. All this with an absence of inflammatory product, and an absence of positively demonstrable diseased condition of the vessel walls other than the thinning before alluded to.

I wish to call attention to one possible source of error in regard to the hemorrhages, which is worthy of consideration. Is it possible that these hemorrhages are artefacts, produced by a mechanical destruction of the overfilled blood vessels in the process of sectioning? Could the blood, so to speak, have been pressed out of the blood vessels by the mechanical force of the microtome knife? This might be possible if it were not plainly demonstrable that there was often a break in the continuity of the vessel where the hemorrhage had taken place; further the evidence of slight compression of the nerve sheath in the neighborhood of the hemorrhages could not be explained in this way. Again, the hemorrhages are found too frequently and are quite independent of the plane in which the sections were cut, and there is evidence of the long duration of some of the hemorrhages.

In the face of these findings then, there seem to be two theories, both of which, it is true, only partially account for the symptoms in the case, and both of which must be regarded as unsatisfactory: first, a mechanical theory. The symptoms may be due to the effect of the pressure of the free blood upon the nervous structures which lay in their immediate neighborhood. In favor of this idea is the number of these hemorrhages, their distribution, and their greater prevalence in the upper portion of the cord. Against it are the greater number of hemorrhages of recent date, and an absence in a marked degree of the effects of old hemorrhages, as fibrin, blood crystals, pigmentation of surrounding structures, etc.

Second, the toxic process, whatever its nature, produced in the cord a condition very similar to that of acute non-purulent encephalitis. The nerve cells of the anterior horn were affected,

but to such a minute degree that their morphologic appearance could not be regarded as differing from the normal when studied by the Nissl stain. In other words, they are to be regarded as functionally affected. The absence of neuritis or degeneration in the anterior or posterior nerve roots would appear to strengthen this view. To explain the purely motor character of the symptoms, it is to be remembered that the anterior horns are more freely supplied with blood than the posterior, and that the purely posterior sensory cells are removed by their position from the direct effect of either a toxic or mechanical influence. The fever and constitutional symptoms are, without doubt, due to the original toxic process, which may have had its origin in the old tubercular foci in the lungs.

While not in any sense doubting either the possibility or the correctness of Knapp and Thomas' assumption of the parenchymatous nature of this process, yet in my case of Landry's paralysis the primary change must have been an interstitial one. I am strongly of the opinion expressed by Taylor and Clark,<sup>18</sup> in a recent article on the subject, that Landry's paralysis is not a definite clinical type, but that it is an acute ascending paralysis comprising a group of symptoms depending upon the most varied causes, and showing such variety of pathological appearances that the term has merely a clinically descriptive value, in much the same way that hemiplegia, paraplegia, or any other term used to describe a clinical picture may have.

138 UEBER EIN NEUES SCHLAFMITTEL AUS DER GRUPPE DER URETHANE (A New Hypnotic Belonging to the Urethane Group). Paul Schuster (Therapeutische Beilage der Deutschen med. Wochenschrift, June 7, 1900, p. 19).

Schuster finds hedonal very useful in cases of functional nervous disease with insomnia. He employs it in doses of two grams, and in the thirty-eight cases in which he has used it has seen no bad results. Sleep follows the administration of the drug within a quarter to a half hour, and lasts from five to seven hours. None of his patients had to urinate more frequently than they did before they had taken the drug.

SPILLER.

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<sup>18</sup>JOURNAL OF NERVOUS AND MENTAL DISEASE, 1900, No. 4.