diseases. This produced a condition of myxedema which presented the same symptoms as sleeping sickness, the resemblance being not only clinical, but also pathological as regarded the anatomical findings of the central nervous system. He also mentioned that Hessler, in a case of catalepsia published in the *American Journal of Medical Sciences* a few years ago noted rapid improvement after thyroid treatment. Finally, he stated that by giving the serum of goats whose thyroid was extirpated, he could produce marked hypnotic effects in every case of insomnia.

That the sexual glands influenced the nervous system was shown by the frequent occurrence of nervous symptoms, even psychoses, after alterations of the sexual glands, especially the ovaries. This was seen during menstruation and pregnancy, and at the time of puberty and the menopause. In all these conditions, also, the thyroid gland was often swollen.

Dr. Lorand said that in cases of melancholia he had found alterations of the thyroid and ovaries. Recently, in the Pennsylvania Insane Asylum, he had seen two cases of dementia in adult males who had scarcely any growth of hair on the face, and who had small, undeveloped testicles. In another case of imbecility in a young man of eighteen years there was the same absence of hair on the face, associated with infantilismus and cryptorchism.

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## PHILADELPHIA NEUROLOGICAL SOCIETY.

March 27, 1906.

The President, DR. D. J. MCCARTHY, in the Chair.

*Acute Myelitis in a Boy.*—Dr. Ralph Pemberton presented the case and said that acute myelitis in childhood, other than acute anterior poliomyelitis, or that due to compression, tuberculosis or syphilis, has received relatively scant attention from either neurologists or pediatrists.

For example, Gowers in his last edition, dismisses the question with a few lines; Oppenheim barely touches on it, mentioning a case consequent upon slight traumatism in a girl of eight. Sachs, in his text-book on the "Nervous Diseases of Children" is about as brief; while Starr in "Diseases of the Nervous System," and Rotch in the "Diseases of Childhood" barely mention it. More satisfactory accounts, however, are to be had in Holt, Ashby and Wright, and Keating, in their respective works on pediatrics. In the so-called idiopathic variety of myelitis, Holt is inclined to ascribe the etiologic factor to an infectious process; while Oppenheim regards it as an unrecognized trauma, slight enough perhaps to have escaped notice, but of a severity sufficient to produce lesions.

All authorities are apparently agreed that acute myelitis of this variety is very unusual in childhood, particularly under the age of ten; and according to Holt, the prognosis is extremely poor, the majority of cases progressing from bad to worse. Mary Putnam Jacobi, however, in Keating's "Cyclopedia of the Diseases of Children," retails a series of eight cases of which two recovered entirely and two were greatly improved. The onset was gradual in all of these but one, an adolescent of eighteen, and this is the rule, according to Holt; early local symptoms being followed by more diffuse symptoms as the disease progresses.

Ashby and Wright in their work on pediatrics devote rather more space to it, and consider the chances of recovery greater in children than in adults, as the cord seems to recover more readily in early life than in later years. They regard the dorsal region as that part of the cord most commonly affected, and lay some emphasis on cases which have been reported as

occurring during measles and convalescence therefrom; the relation being apparently closer than with other infectious diseases.

There have been no cases of myelitis in children reported in the *Archives of Pediatrics* for the past three or four years. The following case of acute diffuse myelitis in a boy is of interest because of the age of the patient and the acute onset. J. D., white, fourteen years.

*Family History.*—Essentially negative, and patient has escaped all the diseases of childhood except measles some years previously, though he states that every winter he has been subject to pains in his legs, which were attributed to rheumatism. Since twelve years of age he has worked intermittently in a glass factory, where he was exposed to considerable heat from the furnaces.

*History of Present Illness.*—About three months prior to admission, after a full day's work in the glass factory, he was suddenly seized with cramps in his hands while reading a book at his own house. He had felt perfectly well all day, had eaten a hearty supper, was not aware of having become more overheated than usual while at work, or of having become chilled after it.. He waited to cool off as usual before leaving the factory, and then walked about seven squares to his home.

In a few minutes after the onset as above, the cramps of the hands extended to his arms and radiated to the shoulders. After being rubbed for a few minutes by some of his family, he felt better and tried to walk, but fainted. He was unconscious about ten minutes, and on "coming to" found he was paralyzed in both arms and the left leg. On the advice of a doctor he was given a hot bath and put to bed. He had a comfortable night, but by the following morning he had lost power in the other leg, and Dr. J. H. Locke, who first attended him, states that he had general anesthesia from the neck down. He seems at this time to have had incontinence of urine and feces, and since then has been confined to bed.

*Examination.*—On admission to Philadelphia Hospital, Feb. 26, 1906, service of Dr. Charles W. Burr, by whom the opportunity of reporting the case was given. Patient is an unusually intelligent boy of about fourteen years, rather undersized and considerably emaciated, with a marked pallor of skin and mucous membranes. Speech is smooth and distinct, and there are no palsies of face or tongue, which is clean and free from tremor. He lies preferably on his right or left hip, with both shoulders touching the bed, the thighs flexed on the abdomen and the legs flexed on thighs. The undermost leg is advanced the more and both legs touch the bed throughout their entire length. There is no deformity of the spine, and it is not tender to palpation, but over the sacrum and lower lumbar region there is a bedsore as large as a man's hand, and extending down to the bone. There are also trophic sores on both heels and over the external malleolus of the right ankle. The arms can be freely though weakly moved in all normal directions, and while the grip is present in both hands, it is greatly impaired. His hands are held usually with a partial flexion of all fingers. The fingers of the left hand can all be extended, the index better than the others, but in the right hand power of extension is lost in all but the index finger, which can be but partially extended. There is more power of flexion of the forearms than of extension, though both are weak and the hands can be well flexed and extended on the forearms. There is some atrophy of the scapula, humeral and forearm muscles, but the wasting is most evident in the hands where the simian hand is present on both sides, with marked atrophy of the adductors of the thumb. No reflexes can anywhere be obtained in the upper extremities. The chest expands not at all with respiration, which is entirely abdominal. The heart and lungs seem normal and the abdomen is protrudent, though soft and yielding to palpation. The liver apparently extends a few cms. below the costal border, the spleen is not palpable and there is flaccidity at all times of the abdominal muscles, though the rectus abdominis reflex is slightly present on both sides. The testicles are partially undescended and the cremasteric reflex cannot be well

tested. The right patellar reflex is absent, but on tapping the patellar tendon on the left, the sartorius muscle contracts. There is no plantar reflex on the right side, but stroking the left sole causes an attempt to withdraw the left leg as a whole. Ankle and patellar clonus and Achilles jerks are absent. Tactile sensation, temperature and pain senses are apparently normal over the entire body, except on the outer aspect of the right leg below the knee, where they are distinctly dulled. He has incontinence of retention of urine and incontinence of feces, though he says his urine formerly dribbled. During the examination he suddenly voided with much force a large quantity of urine.

*Urinalysis* of Feb. 27, 1906, showed: Alkaline reaction; spc. gravity of 10.20; color, deep yellow; albumin, faint trace; microscope, leukocytes and phosphates.

*Examination of Blood,* March 6, 1906, showed: Hb., 62%; R. B. C., 4,800,000; W. B. C., 10,080.

March 27, 1906—Since admission his temperature has three times reached 100° F., but his temperature chart, otherwise shows nothing abnormal. He has gained weight, is considerably brighter, and shows some return of power. It is impossible to completely extend either leg on the thigh as contractures prevent this beyond an angle of about 140°. From this position of partial extension, however, he can entirely flex the left leg on the thigh, but the right not at all. Neither can it be voluntarily extended in the least degree. The trophic sores on his feet have practically healed, the sore on his back is filling up rapidly, and the biceps reflex is now present on both sides to the normal extent.

Dr. Alfred Gordon said the patient had been at the Jefferson Hospital in Dr. Dercum's service for over three months. He entered about five or six weeks after the onset of the disease. When he was brought to the hospital there was absolute paralysis of all four extremities, with marked hyperesthesia and marked tenderness of all the nerve trunks of the four extremities. The symptoms began in the upper extremities, then extended to the lower extremities. The onset was rapid. Dr. Gordon thought at first the case was one of Landry's paralysis. It was atypical in distribution. He was kept under observation and the diagnosis was changed to cervical myelitis. The bedsores the patient presented were very unusual, they extended rapidly, formed, closed and reformed in various places. At one time the sacrum was exposed by the bedsores. The patient developed a sore on the prepuce with suppuration. He also had pus in his urine. The practical point of the case was the treatment. He was put on iodide in gradually increasing doses, and it was remarkable (for it was an experiment), that when he was given the iodides the bedsores healed nicely, and granulations formed, then he would show symptoms of intolerance to iodides. As soon as the iodides were discontinued the bedsores spread and became worse. Again treatment was resumed, there was rapid improvement in the bedsores, but he soon showed intolerance to the iodides and they were discontinued. The patient developed during his stay at Jefferson Hospital very high temperature (105°), had difficulty in breathing several times, but the wonderful resistance the patient presented was astonishing.

Dr. Burr said one interesting thing about the case was that the causation was not known. Myelitis as a rule is secondary to something else, at least the disease first appears in some other organ as pneumonia or acute infectious fever, and secondary to that the spinal cord symptoms develop. In this boy the statement of the family physician is positive that he was previously a perfectly healthy boy. Dr. Burr thought that exposure in the glass works and cold afterwards gave opporunity for the myelitis to arise.

*A Patient with Amyotrophy of the Intrinsic Muscles of the Hands Due to Lead Intoxication.*—This case was exhibited by Dr. Alfred Gordon.

Dr. Pickett mentioned that lead poisoning of the ulnar nerves would

give atrophy of the small muscles of the hand, and that this atrophy in Dr. Gordon's case might be peripheral.

Dr. Gordon replied that the ulnar nerves supply the interossei muscles. The patient had also atrophy of the thenar and hypothenar eminences. They were affected first, the interossei next. The thenar and hypothenar muscles involved at the same time remind us rather of progressive muscular atrophy of spinal type. He thought that the lead affected the cells of the anterior cornua, and did not think the atrophy was from degeneration of the ulnar nerve.

Dr. Pickett said that without opposing Dr. Gordon's view of this case he suggested, contrary to the accepted teaching, that possibly an occasional selection of the ulnar nerves by the lead, might account for this semblance of the claw-hand. In a recent case seen with Dr. Pfahler of typical ulnar neuritis, verified by the existence of the proper area of anesthesia, he found the atrophy of the thenar eminence extensive, evidently more than in one head of the short thumb-flexor. Perhaps in such a case disuse of the hand, particularly as regards the thumb, by paralysis of its adductor, increases the thenar atrophy. Not being familiar with the literature he did not know whether this peripheral hypothesis is new or not.

*A Patient with Intermittent Collapse of the Retinal Vessels* was exhibited by Dr. McCarthy and Dr. F. D. Harbridge.

Dr. Harbridge said the patient complained of absolute blindness, sight beginning to leave invariably on the nasal side of the field, and affording the sensation of a thin veil gradually being drawn before the sight, this becoming denser and denser until vision was absolutely lost, the loss of vision lasting from one to five or six minutes, when it gradually returned, always on the temporal side of the field and with a peculiar play of lights—phosphenes.

In 1898 he had several severe attacks of vertigo, and subsequent to this he has been subject at more or less frequent intervals to severe attacks of migrainous headache, possibly every month, so severe that he was compelled to remain absolutely quiet. He did not have neuroretinitis. There was merely a haze of the upper and lower borders of the disk, not in the true sense of the word a neuroretinitis.

Dr. Harbridge had been unable to find any record of exactly a similar case; that is, a case in which repeated opportunity afforded itself to observe a spasm of the retinal vessels in which there was not, sooner or later, a more or less permanent injury.

Dr. Weisenburg did not believe this case to be one of tabes. In tabes even a low grade neuroretinitis such as was described in this case never occurred. Again, why should there be intermittent collapse of the retinal vessels in tabes. Tabes is a disease of the sensory neurones, and he could not see how such ophthalmological changes could be associated with the disease. Again, granting it to be tabes, the symptoms elicited are not convincing of such diagnosis, as the disease is systemic, and unilateral symptoms would be most unusual.

Dr. Camp thought possibly the case was one of those of ophthalmic migraine that have been observed with collapse of retinal arteries and sometimes migrainous attacks and sometimes only an attack of blindness.

Dr. McCarthy, closing, said that he observed this patient during the attack and there was no change in the heart or pulse. The man was pale as if in terror of losing his sight, though this may well have been a condition of sympathetic derangement. The ocular manifestations may be considered as a complication of an aberrant form of tabes dorsalis. In view of the etiology of tabes dorsalis, he saw no reason why a patient with tabes should not have neuroretinitis. The optic nerves, as he understood it, are in the early stage of primary degeneration. There was narrowing of retinal vessels and with the condition of the disk practically typical of primary atrophy. Were it bilateral he would still hesitate to make a positive diagnosis of tabes. The only case in which tabes was diagnosed

from one set of symptoms and later came under his observation was a case presenting ataxia; later the case was diagnosed as posterior lateral sclerosis by another neurologist, and still later as spastic paraplegia by a third; autopsy showed it to be one of multiple sclerosis. He therefore rather hesitated to diagnose a case from one group of symptoms. This case was shown before the ophthalmological section of the college. This is the only case of its kind reported.

*A Large Tumor of the Basal Occipito-Temporal Region, and a Tumor of the Cerebellopontile Angle, with Brief Remarks on the Symptomatology of the Cases.*—These were presented for Dr. Mills by Dr. Weisenburg.

Dr. Lloyd said he was astonished that these tumors ever escaped the knife of the surgeon. He should have supposed that they would have been cut out. He believed it was customary nowadays to cut out tumors even from the cerebellum, although the mortality returns are not yet all in. He thought the cerebellopontile angle must be a favorite seat for brain tumors. He had in his office a specimen of brain tumor taken from a Blockley patient years ago, lying in almost the same region.

*The Distribution of the Sixth Cervical Anterior Root in the Spinal Cord.*—This paper was read by Dr. A. R. Allen.

*The Temporary Disappearance of the Sensory Symptoms in Syringomyelia.*—This paper was read by Dr. Burr.

*Report of Neurofibrillar Changes in a case of Hydrophobia and One of Pernicious Anemia, with Remarks Upon Lesions of the Neurofibrils in Pathological States.*—This paper was read by Dr. G. E. Price.

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April 24, 1906.

The President, Dr. D. J. McCarthy, in the Chair.

*Herpes in the Distribution of the Right Cervical Nerves, Possibly Tabetic in Origin.*—This case was exhibited by Dr. S. D. Ludlum.

Dr. Spiller thought this an interesting case because of the possibility of herpes as an early sign of tabes. There is evidence in the literature that herpes may be a sign of tabes.

Dr. Gordon said trophic disturbances are not uncommon in tabes, but a zoster eruption is quite rare. According to some, herpes zoster is due to a disease of the spinal ganglia, while others believe it is the result of a peripheral neuritis, and others still that the spinal cord is at fault. Head's conception of the origin of herpes zoster is very interesting. He found associated with the usual changes in the cord in tabes inflammatory conditions in the spinal ganglia. Head finds a certain relationship between the localization of the eruption and the inner organs. Rauschke has recently published a case in the *Centralbl. für Nerv. u. Psch.*, No. 188. A woman suffering from tabes presented an eruption of herpes zoster, whose appearance coincided with an attack of gastric crisis. At another time an identical eruption appeared simultaneously with bladder disturbances. The stomach, according to Head, is in relation with the seventh and the ninth thoracic segments. The area of the skin affected with zona was at about this level. The depressor of the bladder is in relation with the eleventh and twelfth thoracic segments. The eruption was also at about this level. Rauschke's case therefore leaves no doubt as to the central origin of herpes zoster.

*Partial Monocular Ophthalmoplegia in which there is Paralysis of the Upward and Downward Movements and of Convergence in One Eye only without Loss of Associated Bilateral Movements.*—This case was presented by Dr. J. H. Lloyd.

Dr. Mills thought there was probably a partial atrophy of the third nerve rather than a unilateral disturbance of convergence.

Dr. Dercum also thought there was partial third nerve atrophy, but thought it must be a nuclear palsy.

*A Patient with Paralysis of Associated Upward Ocular Movements and a Patient with Multiple Sclerosis.*—These were exhibited by Dr. C. S. Potts.

Dr. Weisenburg stated that he had opportunity to study Dr. Potts' case a number of times. At first he thought the lesion was a cortical one, but he agreed now with Dr. Potts that the lesion was probably in the thalamus, although the case was by no means clear. In many respects this case was similar to one reported several years ago by Klien, in which, as a result of a traumatic lesion of one parietal lobe, with probable involvement of the posterior portion of the frontal lobe, there resulted ataxic movements of the eyeballs, besides other symptoms. This case showed that a unilateral cortical lesion may cause ataxic movements of both eyeballs, and it could be argued that permanent paralysis of associated ocular movements may result from unilateral cortical lesions. This, however, is improbable. As Klien's case was without necropsy, it looses some value.

Dr. Dercum said he had a case almost similar in character to the case reported by Klien, which Dr. Weisenburg mentioned. In his case there was also a lesion in the second frontal convolution, with ataxia of the eyeball and nystagmus.

*Multiple Motor Neuritis.*—This paper was read by Dr. Wm. G. Spiller and Dr. W. T. Longcope. Cases of rapidly developing paralysis of all four limbs are not common. Undoubtedly many classed as Landry's paralysis are caused by neuritis, but to assume that all have this etiology, as some writers have done, is unjustifiable. The literature of Landry's paralysis has become so extensive that it is inadvisable to review it. The subject has recently been discussed by Hans Lohrisch, and the report of his case among others shows that Landry's paralysis may be spinal in origin.

Rather than to discuss the subject of Landry's paralysis separately, the authors had preferred to include it under the heading of multiple motor neuritis. As already said, they did not ignore the spinal form of this symptom-complex. Believing, as they did, that many cases with the symptom-complex known as Landry's paralysis are cases of multiple motor neuritis, they sought a cause for the first manifestations of the paralysis in the lower limbs. The ingenious theory of Edinger must give us the answer. The nerves of the lower limbs are most employed, and these may be most susceptible to poisons, and we have in Landry's paralysis, as in tabes resulting from another poison, the symptoms first manifested in the lower limbs.

They recently had had an opportunity to observe three cases that might be classed as Landry's paralysis, two with necropsy, but they preferred to regard them as atypical forms of multiple motor neuritis; atypical in the rapidity of their development. Multiple neuritis sometimes develops very rapidly, and one of the authors (Dr. Spiller) had in his service recently at the Philadelphia General Hospital a case in which complete paralysis of all four limbs, with great tenderness of all the muscles of the limbs to pressure developed in about twenty-four hours.

In the first case studied, a man of fifty-two years of age, the paralysis developed rapidly, ascending and involving the upper extremities. Sensory symptoms were present, but of short duration. They consisted of pains in the calves of the legs and tendons of the muscles of the arms and legs. The motor symptoms persisted. In the second case, a man of sixty, there was rapid ascending flaccid paralysis terminating fatally in four days. Reflexes were absent. Sensory symptoms were present at first. At autopsy the spinal cord and brain showed no changes macroscopically or microscopically. The peripheral nerves were not examined. The third case

differs from the other two in that there were no sensory disturbances, and belongs clearly to the group of Landry's paralysis. There was ascending flaccid paralysis without sensory symptoms and with loss of the deep reflexes. The control of bladder and rectum was retained until death, which occurred eight days after onset of the symptoms. The microscopical examination of the nervous system showed that the symptoms were caused by degeneration of the motor cells of the anterior horns, of the motor fibers in the peripheral nerves, and of the muscles. The case is remarkable in that the recent degeneration of the muscles, as shown by the Marchi method, was intense and out of proportion to the alteration of the nerve cells of the anterior horns and to the degeneration of the nerves. To these three cases of motor nuritis, in all of which the paralysis of all four limbs developed within twenty-four or forty-eight hours, the authors added an unpublished case of lead palsy with necropsy, as an example of still another form of multiple motor neuritis.

*A Case of Traumatic Hematomyelia* was exhibited by Dr. F. X. Dercum.

Dr. Lloyd stated that it would appear that this man had an entire absence of the pectoralis major muscles. This reminded him of a case he had at Blockley, where there was congenital absence of the pectoralis major muscle. On looking into the matter he found several such cases reported. Most of these cases preserve a wonderful degree of physical strength, as did Dr. Dercum's patient.

*A Case of Hematomyelia Not of Traumatic Origin.*—Dr. Spiller reported the following case that had been in his service in the Philadelphia General Hospital: About four years previously the patient had what was supposed to be spinal meningitis. He recovered, but probably had thickened blood vessels of the spinal cord. He had been in excellent health and was employed at the time the hematomyelia occurred (December, 1905) in an ice house, and with another man had been lifting blocks of ice weighing 100 pounds. He had lifted four of these blocks before any symptoms were noticed. Fifteen or twenty minutes after lifting the last block he began to have pain between the shoulders, and felt numb and weak in the upper extremities. Ten or fifteen minutes later he felt weak and numb in the lower limbs, and was then brought to the hospital. Dr. Spiller examined the man first in January, 1906. He had then recovered largely the use of the lower limbs, but these limbs were somewhat spastic. He had normal sensation for touch everywhere. The patellar reflexes were exaggerated and Babinski's reflex was present on the right side. He had much impairment of temperature and pain sensations in the upper part of the thighs, over the trunk as high as the first or second rib, and in both upper limbs. He had incontinence of the sphincters of the bladder and rectum. He was almost completely paralysed in the upper limbs, and these limbs were much wasted. Dr. Spiller thought if hematomyelia could be diagnosed clinically surely this was a case.

Dr. Lloyd said that spontaneous hematomyelia was a very rare condition. So very rare, indeed, that a few years ago it was generally denied. He recollected that when Dr. Dercum published his book on "Diseases of the Nervous System" he did him the honor to ask him to write the chapter on hematomyelia, and Dr. Lloyd found it extremely difficult to obtain anything in the literature about it. A few years before that time Dr. Kindred had put on record a case in the *Medical News* which was nearest to a case of true spontaneous hematomyelia that Dr. Lloyd had ever seen recorded. A man of fifty-seven years of age, in perfect health, was seized suddenly with pains between the shoulders and in the chest, simulating angina pectoris, and died in a few hours with all the symptoms of paralysis of the spinal cord. A large clot in his cord was found, causing the cord to bulge and completely disintegrating the interior of it. In a recent work on this subject, a monograph by a French author, Lépine, a few cases of socalled spontaneous hematomyelia had been collected.

The question of causation is from a scientific standpoint most interesting. It has always seemed strange that the pathology of the vascular system which plays so large a part in the brain has so little field in the spinal cord. It has been stated by Gowers that emboli are never found in the spinal cord below the level of the medulla. Dr. Lloyd thought this an extreme statement, but the fact remained that we do not have alterations taking place in the blood vessels of the spinal cord and subsequent vascular lesions, the condition which the Germans call Rückenmarkapoplexie, nearly so frequently as in the brain. It is, as has been said, an extremely rare condition. He thought most of the cases reported had been very rapidly fatal. This was a fine scientific question. Dr. Lloyd felt there was doubt about the diagnosis in the present case. The man claimed that his condition came on him suddenly, but we know we have to take with a grain of salt the statements of patients. He presents symptoms of marked degeneration. The paralysis is mostly confined to the upper limbs with atrophy; he could walk fairly well, and he had exaggerated reflexes of the lower limbs. His case presents some resemblance to syringomyelia or to amyotrophic lateral sclerosis. If he had spontaneous hematomyelia it must be in a region of the cord where it catches both anterior horns, makes some pressure on both descending lateral tracts, and practically leaves the rest of the cord without much change. It would be hard to locate one hemorrhage in the spinal cord which would exactly answer for all these lesions.

The question has been raised elsewhere whether in some cases of syringomyelia we did not have the inception of the disease in a condition of hemorrhage. There may be, it was suggested, a hematomyelia in the cord followed by degeneration and cavity formation. That is a point yet to be decided. There were one or two conditions we should always bear in mind in regard to the differential diagnosis of hematomyelia; for instance, there was the possibility of the rapid onset of hemorrhagic myelitis that might be mistaken post-mortem for hemorrhage in the spinal cord.

In the caisson disease, caused by working in compressed air, hemorrhages have not, as a rule, been found, but there is a necrotic process, which apparently has some mechanical cause, possibly thrombi in the blood vessels.

Dr. Hawke said the patient was admitted to the alcoholic ward of the Philadelphia Hospital in 1902. He had then delirium tremens. This was followed by typical symptoms of cerebrospinal meningits. He was in a delirious and semi-conscious state for several weeks. After possibly three months he convalesced and remained around Blockley ever since. Dr. Hawke thought this previous condition might be a factor in determining his present diagnosis.

Dr. Gordon said in regard to a possible hematomyelia: If we look at the so-called spontaneous diseases of the cerebrum, we will find a condition similar to this; for instance, internal hemorrhagic pachymeningitis without apparent cause. They had lately a case at the Jefferson Hospital, which Dr. Dercum mentioned at the last meeting, a man apparently perfectly healthy and well, was taken with somnolence, mental hebetude and so on. He finally died, and internal pachymeningitis was found. The man was about sixty-four or sixty-six years of age, without trauma or history of intoxication.

In regard to this present case, do we know about the syringomyelia disturbances before he came under observation of a physician. A number of patients walk around never suspecting any sensory disturbances, any sensory disassociation. Perhaps the patient had a syringomyelia before. Then collapse took place, and, whatever it may be, affected the anterior cornua and produced the present condition. Dr. Gordon did not say it was not a primary hematomyelia. He did say it was possible it was a plain case of syringomyelia, because we do not know anything about the sensory condition before the trouble came on. Dr. Gordon had read of one or two cases of hematomyelia in the spinal cord. Can we be positive in a

case like this when it presents so many features analogous to other affections of the spinal cord.

Dr. Weisenburg believed that the case was one of non-traumatic hematomyelia and not one of syringomyelia, for the following reasons: If this case were one of syringomyelia, the clinical symptoms were at variance with the pathogenesis of the disease. Syringomyelia is a disease of maldevelopment, the symptoms of which come on early in life, and it would be rare indeed to have a patient with syringomyelia in whom the symptoms did not appear until the forty-eighth year.

The criticism had been made that to explain the sensory symptoms there would have to be a large hemorrhage or a cavity throughout the whole length of the spinal cord. Dr. Weisenburg did not believe that there was such a lesion, but thought a single hemorrhage involving both the gray matter and the column of Gowers of each side would easily explain the sensory symptoms, and that this lesion was limited between the fourth cervical and first thoracic segments. Again Dr. Weisenburg did not believe that the state of the sensation was such as is usually found in syringomyelia, for here he would expect sensory changes of a more or less segmental type.

Dr. Spiller said that some of the objections to the diagnosis were that it was a case of syringomyelia, that possibly the man had had these sensory disturbances before the accident occurred, that it could not be a hemorrhage involving a large portion of the gray matter, that spontaneous hematomyelia was a very rare disease, and therefore it could not be hematomyelia. That hematomyelia does occur we know, and had the case not been unusual he would not have presented it. As for this being a case of syringomyelia, he had never heard of syringomyelia developing within a half hour. There was no reason to doubt the man's statements, and it was very clear that paralysis developed while he was doing heavy lifting, and he was positive he had been in good health previously. As for its being a case of hemorrhage into a cavity, there is no way of diagnosing that condition clinically. Hemorrhage is more likely to invade the gray matter, and a hemorrhage in the lower cervical and upper thoracic regions in the gray matter and anterior portion of the cord would explain all the symptoms. The sensory changes made a diagnosis of amyotrophic lateral sclerosis improbable. Dr. Spiller referred to the fact that tubular hemorrhage extending through a great distance in the gray matter of the cord is well known. The objection of one of the speakers that the man may have had the syringomyelic disturbance of sensation before the sudden development of his palsy may be answered by saying that the existence of such pronounced sensory symptoms without motor disturbance would be improbable. That motor weakness did not exist is shown by the fact that the man was employed in heavy lifting. By this method of reasoning not a man present in the audience could exclude the existence in his own person of a latent brain tumor, but it was not probable that such lesions existed. Again, it is not customary to find that persons previously in good health suddenly developing paralysis have been subjected to a careful examination shortly before the paralysis occurred.

Dr. C. D. Camp read a paper on hemianesthesia.

Dr. Alfred Gordon exhibited a brain showing sclerosis on the left side.

Dr. Spiller reported two cases in which the Gasserian ganglion had been removed, the pressure sense being preserved, although all other forms of sensation were lost.

Dr. C. F. Martin read a paper on the "Sphincter Reflexes in Tabes and Paresis."

Dr. Charles H. Muschlitz read a paper on "Overflow Reflex Manifestations in Dementia Præcox."