This case is interesting, among other reasons, on account of the peculiar movement in the right upper extremity, which resembles in its behavior somewhat that presented by a patient long under the observation of the late Dr. S. Weir Mitchell, and in whom after death no organic change was found by Dr. Spiller in the central nervous system.

Dr. Alfred Gordon asked as to the condition of the reflexes, sensations and other data in the second case.

Dr. Eshner stated that he could not supply the information asked for by Dr. Gordon in the second case. The first case was clearly one of amyotrophic lateral sclerosis, with bulbar involvement.

A CASE PRESENTING ATROPHY OF THE RIGHT LOWER EXTREMITY, WITH INCREASED REFLEXES AND POSITIVE BABINSKI SIGN.

By Williams B. Cadwalader, M.D.

V. J. S., male, aged 17 years, was admitted to the University Hospital on January 13, 1916, complaining of weakness and wasting of the right lower extremity. The patient's father was said to have had syphilis; his mother, two older brothers and two younger sisters are living and well. The patient states that he had been well until 1913, when he had an attack of what he had been told was rheumatism. This condition was characterized by pain on movement in the joints of both lower extremities and also in the right hand. There was no redness nor swelling. After a few weeks he made a complete recovery. In September, 1914, he again had some pain in the region of his hip joints, which was increased by movement. It disappeared, however, after a few days.

He states that since September, 1914, his right lower limb has been gradually growing weak and the muscles have been wasting. In all other respects he is entirely well.

On examination, his eyes, cranial nerves, upper extremities and internal organs were found to be normal. The muscles of the right lower limb are moderately and uniformly atrophied and there is weakness in proportion to the wasting. The tendon reflexes are exaggerated but on the right side they are distinctly greater than on the left. There is a distinct Babinski sign on the right but not on the left side. Ankle clonus is absent. Sensation is normal. There is no incoördination, ataxia nor tremor.

Examination of the joints by Dr. Edward Martin revealed nothing abnormal and in his opinion the atrophy is not related to joint disease. This was later confirmed by Dr. Pancoast, who made x-ray studies. Dr. Leopold made an electrical examination and reported that the muscles reacted normally to the faradic current.

The Wassermann reaction was negative. Blood count and urine examination were also negative.

The combination of atrophy of gradual onset with increased reflexes and Babinski sign is a difficult one to explain. If it were not for the presence of the Babinski sign and the joint condition could have been demonstrated, then arthritis with muscular atrophy would seem to explain the case, but the presence of the Babinski sign in itself must indicate some disturbance of the upper motor neuron. It would seem possible that this patient had had an infection which had caused pain in his joints simulating rheumatism and that the spinal cord had been involved as in acute anterior poliomyelitis, some of the anterior horn cells being destroyed, causing atrophy, and the morbid process extending laterally to involve the pyramidal tracts, producing the Babinski sign and increased tendon reflexes. The fact that this boy's father

had had syphilis might be important but no evidences of syphilis have been discovered in this patient.

Dr. Dercum said that this was of course not a case of primary neurotic atrophy. The history pointed unmistakably to an infection. However, in some of its aspects it suggests a primary neurotic atrophy. Dr. Dercum had himself placed on record two cases of the latter affection in which the knee jerks were preserved. It is also well known that in autopsies in cases of primary neurotic atrophy, the changes are by no means limited to the peripheral nerves, but also involve the spinal cord. On the whole he was inclined to accept Dr. Cadwalader's explanation. The absence of spasticity is further in keeping with this explanation; there is also no evidence of spastic gait.

Dr. J. Hendrie Lloyd said he thought the subject of the relation of muscular atrophy to syphilis was an interesting and rather novel one. He did not know to what extent this case brought it up, because, as he understood, there was no evidence of specific infection. We do know, however, that there are cases of marked muscular atrophy in patients who have suffered from a primary sore. Only that afternoon Dr. Lloyd had been going over one of the cases at the Philadelphia Hospital of extreme muscular atrophy in all four limbs in a young man. The symptoms came on two years after the primary sore. The patient had a positive Wassermann reaction. As in all cases of nervous syphilis, the type was what we see in poliomyelitis, except that the symptoms came on without the evidence of an acute infection. There was not an abrupt onset with fever and pain, such as we see in poliomyelitis. The progress was slow, first in one set of muscles and then in another. Dr. Lloyd said he had another case in a man at Blockley which had impressed that fact upon him. This man was a very robust, perfectly developed fellow, over six feet tall, who had atrophy of all the muscles of one thigh from his hip to his knee. There was nothing to account for it, except that the patient had had a syphilitic infection a few years before the onset of the trouble. He had no pain, no alteration of his cranial nerves or reflexes, no evidence of syphilis in the action of his eye muscles, but he had had syphilis; and as a result some years later he began to have very marked muscular atrophy of the muscles of his thigh. It was of the type seen in poliomyelitis. This subject is referred to by comparatively few authors. McDonagh, however, in his recent work on the Biology of Syphilis, includes muscular atrophy as one of the symptoms. Nor should there be any surprise that muscular atrophy is one of the symptoms of syphilis. If the spirochete gets into the parenchyma of the posterior tracts it causes locomotor ataxia; if in the brain cortex, it causes paresis. In these cases of muscular atrophy, the spirochete probably gets into the parenchyma of the anterior horns. In these cases also the type of amyotrophic lateral sclerosis is sometimes seen. Or, again, there may be a primary lateral sclerosis, without muscular atrophy. Some years ago Dr. Lloyd, with Dr. Ludlum, had put on record a series of cases of primary, or essential, lateral sclerosis, all occurring in syphilitic subjects. In these cases doubtless the organism of syphilis finds lodgment in the lateral tracts, just as it does in tabes in the posterior tracts.

Dr. Cadwalader said that he had considered this case from exactly the same point of view Dr. Lloyd had spoken of, but there seemed too little evidence of syphilitic infection to warrant drawing such conclusions. Dr. Cadwalader said that he believed many cases of amyotrophic lateral sclerosis were syphilitic in origin. Also some cases of muscular dystrophy and some cases of spinal muscular atrophy must be syphilitic.

Dr. S. F. Gilpin showed a case of paralysis agitans in a negro.

Dr. Dercum said this was the second case of this kind he had seen. Several years ago Dr. Burr showed a case of this affection in a negro before

the Society. Dr. Gilpin's case is one of typical paralysis agitans and occurring in the negro, it is very rare.

Dr. N. S. Yawger presented a case of a lesion probably confined to one posterior horn of the spinal cord throughout the thoracic region.

Dr. J. S. Rodman and Dr. W. B. Cadwalader presented a case of cerebral abscess apparently cured by operation.

Dr. A. A. Eshner asked whether the abscess was merely intracranial or subdural or really cerebral. Its superficial character, as well as its mode of origin and the results of operation would seem to suggest the former rather than the latter.

Dr. George E. Price said the high leukocytosis was interesting. In uncomplicated intracranial abscess it was apt to be below 14,000; while in meningitis it ran from 14,000 upwards. In this case the meninges were involved.

Dr. Rodman, in closing, said if he had failed to make it clear that this was a real cortical abscess he had missed the whole point in presenting the case. There was a superficial abscess as well. The superficial abscess was between the skull and the scalp. The abscess beneath the dura was in the brain substance itself, about 2 cm. below the surface of the cortex.

Dr. Charles K. Mills and Dr. George Wilson presented a case of Sprengel's deformity: congenital elevation of the scapula.

Dr. Wilson said the patient walked with difficulty because she probably had tabes, she had sharp pains and lost reflexes, also incontinence of urine. The lack of motion of the upper limb was due to lack of motion of all the shoulder muscles, atrophy of the trapezius and deltoid. The scapula fails to descend. It is an embryological condition.

Dr. William G. Spiller read a paper on tabetic ocular crises.¹

Dr. Dercum said that Dr. Spiller's paper called to mind the intense ophthalmic migraine so-called which we every now and then meet with in paresis, especially in the early stages. Every now and then, among the early symptoms presented by paresis we meet with intense pain referred to the eyeball or to the orbit or immediately adjacent regions. These attacks are usually of short duration. They are put down in the older textbooks as attacks of ophthalmic migraine. It is not impossible that they are really attacks analogous to the tabetic crises which Dr. Spiller has described.

Dr. George E. Price inquired as to the length of the individual attacks. He thought the duration of the attacks would have a bearing on the differential diagnosis between ophthalmic migraine and tabetic ocular crises.

Dr. Alfred Gordon said tabetic ocular crises as well as crises of other cranial nerves are rare. In the last edition of Gowers no mention is made of them. In connection with this a brief history of a case of tabes under Dr. Gordon's care will be of interest. The patient had attacks of the following nature: all of a sudden she would be taken with a deafness of the left ear with dizziness. There was no pain. The attack came on suddenly and disappeared suddenly. She had for a number of months attacks of this character. Evidently it was very likely a tabetic crisis consisting of a disturbance of function of the eighth nerve. Sudden attacks of this character lasting a fraction of a minute and consisting of deafness with vertigo and without pain are exceedingly rare. He examined a patient for objective sensory disturbance of the ear in which there was no trace of involvement of the fifth nerve, but this striking symptom of sudden deafness of the left ear and vertigo, although without pain, reminds one of tabetic crises.

¹ Published in the Journal of the American Medical Association, March 18, 1916.

Dr. Charles K. Mills said the case was interesting and had been well presented by Dr. Spiller. There was only one point about this patient to which he would refer. So far as his description of what he sees is concerned, Dr. Mills was inclined to attribute that to his emotional and imaginative tendencies. He is a well known case at Blockley. Almost every member of the staff has at times heard him discoursing on the Lord, the stars, salvation and damnation, etc., much after the manner of his description of his visions in the attacks described.

Dr. Spiller said the man he presented has attacks at various times during the day and they disappear as suddenly as they come. He did not believe the visual phenomena could be attributed to vivid imagination, because they were always associated with agonizing pain and intense lachrymation.

Dr. Charles K. Mills said he did not mean to infer that the case was not such as Dr. Spiller described. He had seen the man in one of his visual attacks a short time since. He merely wished to indicate that the patient's imagination would tend to expand a real visual appearance. If he saw a lizard or a lobster in his optic crises his imagination was sufficient to call up a train of other visions.

Professor Ulric Dahlgren of Princeton University read a paper on the primitive balancing apparatus in vertebrates.

FEBRUARY 25, 1916

The President, DR. FRANCIS X. DERCUM, in the Chair

Drs. S. D. Ingham and William E. Robertson presented a case in which spinal tumor had been removed ten months previously.

Dr. John H. W. Rhein asked if there had been at any time any abdominal pain, and what the present reflexes were.

Dr. Ingham said the reflexes of both legs were absent constantly from the time of the first examination.

Dr. P. de Long showed a case of tabes with paralysis agitans.

Dr. Theodore Weisenburg said he had asked Dr. de Long to show this case because he thought it was very unusual. He had known this man for sixteen or seventeen years. He always thought him a typical case of tabes dorsalis. Six or seven years ago Dr. Weisenburg noticed that the man had a tremor of the right upper limb, then in the head and now in both lower limbs. It is a very unusual complication.

Dr. Rhein said that he had presented before the Section on Nervous and Mental Diseases of the American Medical Association, in June, 1904, a case of locomotor ataxia in which there was a tremor resembling paralysis agitans. The pill-rolling position of the fingers was well illustrated, the tremor being fine and rhythmical and bilateral. Voluntary effort quieted the movements temporarily, after which they became more intense. The pathological study of the case showed the usual findings of well-advanced tabes. Arterial change could not even be compared with that found in cases of paralysis agitans in which it has been claimed that the lesions resemble those of old age. No degeneration of the muscle spindles was found. This case was one of tabes associated with paralysis agitans.

CEREBRO-CEREBELLAR ATAXIA

By Alfred Gordon, M.D.

Girl of 17, made her first attempt to walk at 18 months. Began to speak only at 2 years of age. From that time her speech remained deficient. She