

Society Proceedings

PHILADELPHIA NEUROLOGICAL SOCIETY.

October 23, 1906.

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

MOTOR PARALYSIS AS AN EARLY SYMPTOM OF TABES DORSALIS.

By Dr. C. D. Camp.

First Case: Male, 56 years old, history of syphilis thirty years before and alcoholism for past three years. The first symptom was a unilateral paresis of the extensors of the foot. Examination showed loss of the knee jerks and of the Achilles jerks, sensory symptoms, Romberg's sign and myotic pupils with loss of the reaction to light. There was no pain or tenderness over nerve trunks.

Second Case: Clerk, 65 years old, no history of syphilis or alcoholism, but he had been a painter 20 years previous to the onset of his symptoms. First symptoms sharp, shooting pains in the legs followed by gastric and vesical crises, unilateral foot drop of recent development and ataxia. Examination showed Westphal's sign, Romberg's sign, ataxia of all four extremities, sensory changes, and optic atrophy. Dr. Camp said that the motor nerves of the eyes are frequently affected in the beginning of a case of tabes, but that other motor nerves may be affected is not well recognized. The pathogenesis is the same as in other cases of tabes, but the toxin acts more extensively. The affection may be due to degeneration of the anterior horn cells, owing to a default of the habitual excitations upon which the vitality of the cells depend.

Dr. F. X. Dereum said that it is a well known fact that motor involvement may occur in well advanced tabes, but it is not, as Dr. Camp notes, a well known fact that these palsies may occur early in tabes. It is a very common thing to see palsies in connection with the eye muscles in tabes; there is nothing more common than a transient diplopia, nothing more common than a ptosis. Sometimes these palsies are temporary, at other times permanent and persistent. The speaker said that one thought was suggested to him in both of Dr. Camp's cases, namely, that the ordinary etiology of tabes was wanting. Both syphilis and alcohol were present in one case, and the part possibly played by alcohol had to be considered; in the second case there was no history of specific disease. Dr. Dereum also called to mind the palsies met with in primary neurotic atrophy, in which disease we may also have organic changes in the cord, such for instance as degeneration of the posterior columns of the cord. Dr. Dereum, however, agreed with Dr. Camp that the cases shown were true tabes.

Dr. Alfred Gordon stated that a year and a half ago he reported a case in *American Medicine* in which he discussed the pathogenesis of lead intoxication. The patient presented during his life the picture of tabes, but questioning closer he found the patient had been a painter. He examined the peripheral nerves of all the extremities, also the cord. He found posterior sclerosis as well as degeneration of the nerve trunks.

Dr. J. Hendrie Lloyd thought it hardly fair to criticize these cases after only a brief reference to the notes. He was skeptical about the diagnosis of locomotor ataxia in the first case presented by Dr. Camp, and considered it more suggestive of alcoholic neuritis. One point that raised a doubt, however, was the condition of the eyes. Dr. Lloyd stated that his ideas have been considerably enlarged and modified on the subject of the possibility of the ravages of alcohol on the nervous system. He had seen a number of cases in which the classical symptoms were not all present, but a clear and distinct history of alcohol

was obtained. Two years ago he had a young man under his care, an undoubted case of alcoholic multiple neuritis, in which the symptoms were confined entirely to the lower extremities. In such a case it might have been difficult for some observers to differentiate it from a beginning case of locomotor ataxia. The patient made a good recovery.

The second case of Dr. Camp's he thought had a suspicious history of exposure to lead, although he had not been thus exposed for a good many years. One of the most marked cases of pseudo-tabs that Dr. Lloyd had ever seen, was reported from his Blockley clinic ten or twelve years ago. On making a careful microscopic examination he found no trace of degenerative changes in the cord. The man had followed painting for many years, but also had a history of alcoholism. This he thinks is the only matter of doubt about such cases, and raises a very interesting question. Another interesting question, is the possibility of locomotor ataxia beginning as a multiple neuritis. He had seen cases which suggested this possibility. There is nothing inconceivable in it.

In closing Dr. Camp replied that in reference to the case in which there was a history of alcoholism being one of alcoholic neuritis, the patient had only been drinking for the last two or three years, and the symptoms dated back nine years.

As regards the etiology in the second case where there was no history of syphilis nor alcoholism, there was only the one fact of the man having been a painter 20 years before. That is a long time to go back, but he supposed that if we have to theorize, there might be some connection between the two. Lead intoxication has been considered to be one of the causes of tabs.

As to Dr. Spiller's reference to a statement of Cole's, that he (Dr. Spiller) did not believe the posterior columns of the cord are degenerated in every case of multiple neuritis, Cole's exact words are, that he "could find no record of any case of multiple neuritis in which the spinal cord was examined by the Marchi method, in which it was stated that the posterior columns were free from degeneration."

TRAUMA PRECEDING PROBABLE SYRINGOMYELIA AND TABES.

Dr. S. D. Ludlum presented these cases:

Dr. Dercum thought one important point should be insisted on in these cases; that is, the relation of trauma should be clearly defined. He did not think too much stress should be laid on the fact of trauma in either of the cases presented by Dr. Ludlum. The relation of trauma to tabs has been gone over a great many times and none has ever been shown. He said that he understood that Dr. Ludlum disclaimed an etiological relationship with trauma in the cases presented.

Dr. Gordon said that the cases did not impress him as in any way extraordinary. Traumatic syringomyelia, as understood in its broadest sense, that is a hemorrhage or anything which involves in the spinal cord Gowers' tract, is a possibility. Dr. Gordon recalled that he presented before the Neurological Society a few years ago a woman who had a distinct history of trauma and developed distinct syringomyelic symptoms and atrophic disturbances. As far as the tabs is concerned, he thought this a question of some importance. At the last Congress of the French Neurologists and Alienists the question of traumatic paresis came up and observations were brought forward showing that the symptoms of paresis developed immediately after trauma. The general opinion nevertheless was that the trauma was simply an accidental cause. We know nothing of the previous condition of the patient in regard to his knee jerks, his ocular symptoms, so it is difficult to draw even the slightest inference as to trauma as a factor in the causation of tabs.

Dr. Spiller said he did not believe trauma had been the cause in