

NEW YORK NEUROLOGICAL SOCIETY.

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FACIAL AND RETROBULBAR NEURITIS.—PERIOSTITIS OF OPTIC CANAL.

Dr. William M. Leszynsky presented a woman, thirty-eight years of age, who had come to him first on October 11, 1898. At that time there had been a complete facial paralysis of the left side, which had existed for five weeks. There had been a loss of taste, but the eyes had remained normal. The facial paralysis had been preceded by severe pain and tinnitus in the left ear, lasting several days. She had been treated by galvanism in the usual manner. Two months later, while under treatment, she had complained of severe pain in the left supraorbital and left temporal regions, lasting continuously for over a week. She had then become totally blind in the left eye. The pupil was absolutely immovable, and the ophthalmoscope showed edema of the papilla, but nothing else. Within a week a well-marked papillitis could be detected in the left eye. The vision in the right eye was normal. Under treatment by mercurial inunctions and the internal use of iodide of potassium the vision had improved somewhat, and on November 4 the vision in the right had been 22-100ths. She now had optic atrophy, and the vision had decreased. The faradic irritability had returned, but was still below the normal. A diagnosis had been made of facial neuritis and retrobulbar neuritis arising from periostitis in the optic canal. This was a very unusual condition, and for this reason the patient was presented. Dr. de Schweinitz, of Philadelphia, had recently reported two or three cases, but in them the retrobulbar paralysis had developed nearly a year and a half after the neuritis. The case also showed that a return of faradic irritability did not necessarily mean a return of motility.

Dr. G. M. Hammond asked if uremia could be absolutely excluded in the case just presented.

Dr. Leszynsky replied that there had been a complete absence of all urinary signs and symptoms. The neuritis seemed to have been either of rheumatic or syphilitic origin. There were some elements in the case that had led him to think it possibly of syphilitic origin.

SYRINGOMYELIA.

Dr. J. Collins presented a man whom he had seen with Dr. Powell. The latter had removed a small lipoma from the lumbar region, having no connection with the spinal cord or membranes, contrary to the opinion that had obtained before

operation. The patient was thirty-six years old, and had contracted syphilis nearly five years ago. Since that time he had been under almost constant treatment. He had complained of weakness in the right leg, pain in the back, and slight numbness in the left lower extremity. Careful inquiry had elicited the fact that while still a boy he had experienced difficulty in keeping the right leg in the stirrup when riding horseback, and had noted in that foot what had really been a clonus. The knee-jerk was exaggerated on the right side, and on that side there was also ankle-clonus. There was absolute thermoanesthesia from the fourth intercostal space on the left side down to the tip of the toe. The diagnosis was syringomyelia.

Dr. Richard H. Cunningham said that he remembered having tested the sensation of this patient some time ago, but he did not recall the existence of thermoanesthesia. He had always looked upon the case as one of tumor of the cord, and not as essentially syphilitic.

Dr. Pearce Bailey said that several similar cases had been met with at the Vanderbilt Clinic during the past three or four years. It was the custom there to examine regularly for such anesthesia, and while he did not recall the individual case, it was exceedingly probable if thermoanesthesia had been found it would have been noted and remembered.

Dr. L. Stieglitz was inclined to accept Dr. Collins' view of the pathology, although it was unusual to find such extreme spasticity in such an early stage. He would like to know if there was any muscular atrophy present anywhere in the body, and if there was any change in the sympathetic supply to the eyeball.

Dr. C. L. Dana also looked upon the case as one of syringomyelia. Some of the cases of slowly developing spastic paralysis without sensory symptoms seemed to him to be really examples of early multiple sclerosis. He had seen such cases develop characteristic eye symptoms after a good many years.

Dr. Graeme M. Hammond said he had had an opportunity of examining this man, and would agree entirely with the statement regarding the clinical symptoms. He would accept the diagnosis given by Dr. Collins. This case was certainly not one of recent development. He could not classify it except as one of syringomyelia.

Dr. Joseph Collins, in closing, said that the right lower extremity was one inch and a half smaller than the left. There were no ocular symptoms, and no vasomotor disturbances of the face. There might have been in the beginning an area of gliomatosis which, at first, was connected with the central canal.

TUMOR OF THE AQUEDUCT OF SYLVIVS, OR OF THE CEREBELLUM; DOUBLE JOINTS.

Dr. Collins presented a boy of eighteen, exhibiting all of the cardinal symptoms of tumor of the aqueduct of Sylvius or of the cerebellum. About three years ago it had been first noticed that he was lethargic. Fourteen months ago, while climbing into a window, he had fallen, and shortly after this he had begun to vomit and to suffer from periodical headaches.

These had been followed by increasing sleepiness, and by a disturbance of the gait. His limbs could be thrown around, and the joints bent backward in a remarkable way. The kneejerks were feeble. There was no nystagmus, and no true cranial nerve or ocular palsies. Both eyes showed choked disk and slight pigmentation.

Dr. C. L. Dana thought this peculiar drowsy condition was characteristic of tumors in the aqueduct of Sylvius, extending forward into the third ventricle; yet, in other respects, the symptomatology was that of a cerebellar tumor. If the tumor were in the aqueduct of Sylvius it could not cause much distention or infiltration, or there would be some eye symptoms. The weight of the evidence seemed to favor a tumor in the cerebellum. It was possible that the double-jointed condition had been increased by his present affection.

A CASE OF HEMORRHAGIC ENCEPHALITIS AND MYELITIS.

Dr. Charles L. Dana and Dr. M. G. Schlapp presented this report, which was read by Dr. Dana. The subject was a man of sixty-seven years of age, who had come under observation last June. There was no clear history of intemperance. His previous health had been good. During the last seven years of his life he had been somewhat of an invalid. On June 4, 1899, he had complained of headache, and after taking two cups of tea, he had gone to bed. He had fallen out of bed a few hours later, and had then been discovered to have left hemiplegia. He had been removed to Bellevue Hospital. At that time his temperature had been 104° F., but the temperature had quickly fallen. On examination he could not protrude the tongue, and it was almost immobile. He was paralyzed almost completely on the left side. The lips were markedly affected. The abdomen was somewhat rigid. The patellar reflexes were normal, and ankle-clonus was absent. Sensation to heat and cold was normal. The urine showed nothing abnormal. Four days after admission he had had a chill and the temperature had risen to 105.5° F., and examination of the blood had shown the malarial plasmodium. After having been in the hospital for one week it had been noted that there was no paralysis of the right arm or leg, and the most notable feature was the paralysis of the tongue and lips. He was unable to make any distinct articulate sound. He could swallow and cough, and understood perfectly what had been said to him. Death had taken place on June 25.

At the autopsy made by Dr. Dana, only the brain and cord had been examined. There were slight edema and congestion of the arachnoid. The vessels of the base were not atheromatous. In the right hemisphere was an area of capillary hemorrhage and softening in the lower portion of the pos-

terior central convolution, and superior part of its marginal gyrus. In involved mainly the deep part of the upper lobe of the fissure of Sylvius. In the centrum ovale were two or three smaller areas of hemorrhage. There was no evidence of hemorrhage or softening in any other part of the cerebrum, cerebellum, or pons. The spinal cord showed two foci of softening, one in the upper dorsal and the other in the lower. The brain had been hardened in formalin, and then stained by various methods. The microscopical examination had shown in the affected area much dilatation of the blood vessels, small extravasations of blood, a great proliferation of cells, showing an irritative reaction to the hemorrhage—in other words, the evidence of encephalitis, apparently secondary to the disease of the blood vessels. There were also small areas of softening of the brain in the neighborhood of these hemorrhages, also the result of the breaking and obliteration of the vessels. The walls of the latter were somewhat thickened, but gave no evidence of a syphilitic process. Sections and drawings were exhibited. The examination of the rest of the brain had failed to show any evidence of sclerosis. The medulla showed no evidence of softening or of inflammation. Examination of the spinal cord had not yet been completed. The cord showed a small center of poliomyelitis in the dorsal region. Below this was a more extensive poliomyelitis, involving both the anterior and posterior horns. The appearances very closely resembled those described by other writers on encephalitis hemorrhagica.

Regarding the occurrence of speech disturbance in a case of this kind, Dr. Dana said that in the great majority of cases of this form of hemiplegia there was no speech disturbance except during a few days after the attack, which might easily be ascribed to shock. In another class of cases of hemiplegia there was a slight uncertainty in speech, lasting for several months. In still another class the hemiplegia was associated with some difficulty of speech, and sometimes true aphasia. He had never observed any sensory aphasia in these cases. When the lesion was well below the cortex, and involved the motor neuraxons and the collaterals going to the opposite hemisphere, there would be some hesitation in speech; when the lesion was still lower down, there would be no disturbance of speech at all. There was a very general opinion prevailing to the effect that pseudo-bulbar palsies of cerebral origin are due to lesions in the lenticular nucleus, but it seemed to him that our present knowledge of the anatomy of the brain made such a view untenable.

Concerning the disturbance of the tongue, the speaker said

that Dr. Collins, who had given a critical description of this subject, placed the tongue center in the anterior and upper part of the foot of the anterior central convolution. Some of the centers of the movement of the tongue were probably deep in the fissure of Sylvius, and not subject to experimental stimulation. In the case under discussion it was interesting to note that mastication and deglutition were not impaired. The case also brought up the subject of non-suppurative encephalitis. It seemed to be established that there was an infectious hemorrhagic encephalitis in infants; also that there was a hemorrhagic poliomyelitis in adults; also an acute hemorrhagic encephalitis of adults due to some acute infection, such as influenza. In the case presented there was a distinct history of malarial infection and of alcoholism, and this combination, it was reasonable to suppose, was responsible for the lesions discovered. Such a case might be classified under the name of degenerative hemorrhagic encephalitis. An examination of the autopsy records of sixty-seven cases of apoplexy showed that 42 of them were hemorrhagic, 11 embolic, and 11 thrombic, and 3 encephalitic.

Dr. E. D. Fisher agreed with the reader of the paper that, in a small percentage of cases, there might be encephalitis, and that the spinal cord might also be involved. He had seen cases affecting the right hemisphere in which the aphasia had been permanent. It was not common, of course, for such persons to lose the memory of names and things, but he could recall a few such cases. He did not believe there were enough fibers passing over from one side to the other to account for that. The right hemisphere he believed was the region in which the center of speech was fixed, and while it was more accentuated on the left, the right, in his opinion, could not be entirely excluded. This belief was based on his own clinical experience.

Dr. B. Onuf thought that, as a rule, dysarthria rarely occurred in connection with lesions of the internal capsule; it was more often met with in lesions of the cortical foci. According to his experience it was most frequently observed with lesions of the left hemisphere. To explain this he assumed that all the articulatory muscles were represented in each hemisphere, and that the center of the right hemisphere was subordinated to that of the left hemisphere through the fibers of the corpus callosum. If there was a lesion of the right hemisphere there might be a temporary dysarthria; if, however, there was a lesion of the left hemisphere the dysarthria would be permanent. On the other hand, if the lesion were in the internal capsule, whether on the right or on the left, dysarthria would occur. If the dysarthria were marked, it could be stated almost certainly that the lesion was in or near the cortex. He believed that there was a double speech center, and that that of the right hemisphere was subordinated to that of the left.

Dr. Fisher asked Dr. Dana whether he had noticed any difference in the character of the articulation in the first few days in a person with right hemiplegia and in one with left hemiplegia. Personally, he had found as much difficulty in the one case as in the other.

Dr. Collins thought the paper was most interesting because adequately proving that a single lesion was capable of producing diffuse

and multiple lesions in the central gray matter. This was the first communication of the kind ever presented in this country. The paper called attention to the possibility of an acute malarial infection giving rise to such a process. The changes discovered in this examination were those which might be expected theoretically. He did not see how this report could have much effect on the question of aphasia, because serial sections of the entire length of the medulla oblongata had not been presented—indeed, he was doubtful if the patient had had true aphasia. There might have been an affection of the central allocation of the tongue not implicating the muscles of the lips and palate, in part due to the very small focus of inflammation in the medulla oblongata. The explanation given by Dr. Onuff was the one that he had himself long held, and which was the same as that given by Dr. Dana in this report.

Dr. M. G. Schlapp said that the inflammation in the medulla seemed to be a very recent one. There was a small-round-cell infiltration, but the large proliferating cells found in the cortex were not present. The infiltrating stage and the proliferating stage were both represented in the cortex.

Dr. Dana, in replying to Dr. Fisher, stated that his experience had been that dysarthria occurred about as often with right hemiplegia as with left, and that dysarthria was practically due to the size of the lesion. If the lesion were very large and the hemiplegia complete there was almost always dysarthria. Where there was only slight hemiplegia associated with dysarthria it was probable that the lesion was high up in the cortex, or involved a part of it. He had not thought that this case illustrated aphasia, but rather dysarthria. The weight of evidence in this case seemed to indicate that a paralysis of the tongue was the result of a cortical lesion—a sudden apoplectic lesion. There had been absolutely no hemorrhagic process in the medulla.

REPORT OF AN UNUSUAL CASE OF LEAD PARALYSIS WITH AUTOPSY.

Dr. B. Onuf reported this case. The patient was a man, thirty-seven years of age, having a good family and personal history. He had been a painter for many years, and had had one attack of lead colic some years ago. A short time ago he had had a severe fall, and for some days afterward had appeared in a dazed state. From March until August he had done no painting; then he had begun painting again with colors containing a large percentage of lead. He had kept this up during the month of August, but during this time had suffered frequently and severely from colic. At the end of August he had become quite ill, and in three or four days had become almost helpless in the lower extremities and in the left arm. On admission to St. Catharine's Hospital in September, 1899, there had been complete flaccid paralysis of both lower extremities; absence of both knee-jerks; marked tenderness of the nerve trunks and muscles of the lower limbs. There was also paralysis of the flexor muscles of the left arm and of the extensors of the fingers. The gum showed a "lead line." Under diaphoretic treatment the spontaneous pain

had ceased. On October 4 he had had an attack of severe dyspnea, and on the following morning he died in a second and similar attack. The extensors and flexors of the right arm were found, at autopsy, wasted, as were also the peroneal muscles and the flexors of the thigh. The left lung was the seat of a broncho-pneumonia. Microscopical examination had shown what appeared to be poliomyelitis of the anterior horns. The parts examined so far had been the second and fifth lumbar, and there was an enormous infiltration of the walls of the blood vessels with round cells. This infiltration of cells had been so extensive as to lead to very general distention of the nerve cells. The anterior roots were markedly affected, but the posterior roots of the lumbar region were normal. The plantar nerve showed increase of connective tissue and endarteritis obliterans. The liver had been examined by Dr. Bookman for lead, but none had been found. As five weeks had elapsed such evidence was not of much importance.

Dr. L. Stieglitz said that this case showed in the human being exactly what he had found in his case of experimental lead poisoning. In experiments on 36 animals he had only obtained in one guinea-pig an acute poliomyelitis at the necropsy, and also cell infiltration and more or less destruction of the nerve cells around the foci. This guinea-pig had been paralyzed acutely. Death had occurred within twenty-four hours. In his experimental cases he had obtained, in every instance, changes in the ganglionic cells themselves. The case presented in the paper was, of course, an atypical one of lead poisoning, and hence would not justify any conclusions regarding the ordinary pathology of lead poisoning. The cases reported by Oppenheim and by Herter seemed to him the most typical.

Dr. F. D. Fisher thought the clinical history of this case corresponded more with that of alcohol than of lead poisoning. As a rule, in cases of lead poisoning the lower extremities were the parts first involved. He had written on this subject some years ago, and had called attention to the occurrence of these degenerative changes in the brain and cord in cases of alcoholism. It, therefore, seemed to him that the case reported combined both alcoholism and lead poisoning. The changes in the central nervous system had been pretty well established.

Dr. W. M. Leszynsky said that it also seemed to him that the reader of the paper had not proved the connection between the lead poisoning and the post-mortem findings. Certainly the case appeared to be one of alcoholic toxemia, or of some acute infectious process.

Dr. Hammond said that while it was very probable that the man had suffered from lead poisoning, he doubted very much if the lead had had anything to do with the condition of the spinal cord; it was much more probable that it was the result of some acute infectious process. He did not believe a case of true lead toxemia had been reported in which such changes in the nervous system had been observed.

Dr. Cunningham said that some weeks ago he had met with a case of lead palsy in a man who at the same time had developed a mild form of influenza. The phenomena observed had been quite similar to those reported in the paper.

Dr. Collins also took the ground that in addition to the lead poisoning there had been an alcoholic neuritis, or some acute infection giving rise to the changes in the spinal cord.

Dr. Onuf said that the frequent attacks of colic during the last few weeks that the man had been able to work seemed to show the close connection between the lead poisoning and the other symptoms. Cases of alcoholic neuritis usually present quite a different picture from that exhibited by his case. The autopsy had revealed evidence of a certain amount of alcoholism, it was true, but the man had not been drinking for a considerable time previously. There had been no history of an acute infectious process.

THE LATE DR. WILLIAM A. HAMMOND.

The New York Neurological Society desires to record its regret at the death of Dr. William A. Hammond, one of the founders of this society and for years one of its most active and distinguished members. Dr. Hammond was one of the pioneers of neurology in this country. His work was always suggestive and most lucidly presented, and it was often original and of permanent value. It has indissolubly linked his name with the history and growth of neurological science. It was before this society that many of his papers were presented. We record our appreciation of his work as well as of his brilliant mental gifts. These were most helpful to this society in its beginnings, and led to enduring results in the advancement of clinical neurology and psychiatry and the development of neurology in America.

CHARLES L. DANA,
LONDON CARTER GRAY,
GEORGE W. JACOBY.

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20. ZUR KENNTNISS DER SYPHILITISCHEN PSEUDOPARALYSE (Contribution to Syphilitic Pseudoparalysis). Oberwarth (Jahrbuch für Kinderheilkunde, 49, 1899, Heft 4).

Twelve cases of syphilitic pseudoparalysis of childhood are here reported. In four of the cases a careful electrical examination by Kalischer gave normal reactions. This fact, together with the rapid recovery, speaks against an involvement of the central nervous system, such as Zappert found in his case. In three of the cases some contractures or spastic symptoms were present. This was probably due to reflex spasm, caused by the great sensitiveness of the bones (periostitis). In most of the cases distinct evidence of periostitis was present, either in the presence of swelling or in tenderness along the course of the bones. This process gave rise to the rigidity of the muscles and the childish instinct keeping voluntary motion in abeyance. Evidence of organic disease of the nervous system; headache, vomiting, incontinence of urine or feces, or changes in the reflexes were not present. He draws the following conclusions: That pseudoparalysis syphilitica disappears in a few weeks under antisymphilitic treatment, leaving no evidence of organic nervous disease; that the clinical phenomena can be explained by an osteochondritis specifica; that the clinical picture is that of a more or less painful and complete paralysis of the extremities.

MCCARTHY.