OLIGODENDROGLIOMA OF THE SPINAL CORD*

JOHN R. RUSSELL, M.D., AND PAUL C. BUCY, M.D.

The Chicago Memorial Hospital and the Department of Neurology and Neurological Surgery, The Illinois Neuropsychiatric Institute, University of Illinois College of Medicine, Chicago

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Oligodendrogliomas comprise 3.5 per cent of the gliomas of the brain, according to the analysis of 254 histologically verified gliomas classified by Bailey and Cushing. In their discussion of oligodendroglioma of the brain, Bailey and Bucy pointed out that this variety of glioma should also occur in the spinal cord, since the oligodendroglioma are found in the spinal cord as well as in the brain.

Detailed histological analyses have been made of only a few large series of intramedullary spinal cord tumors. Kernohan, Woltman and Adson in 1931 classified 51 cases of intramedullary spinal cord tumor operated upon at the Mayo Clinic and found 2 (4 per cent) oligodendrogliomas. Up to Jan. 1, 1939, the Mayo Clinic series included 64 intramedullary tumors, 3 of which were oligodendrogliomas, an incidence of 5 per cent. The same group reported 25 gliomas of the conus medullaris and filum terminale, of which 1 was an oligodendroglioma. In 1944 Woods and Pimenta made a study of 30 histologically verified spinal cord gliomas, and classified 1 as an oligodendroglioma. Oljenick has also reported 1 case, giving a total of 6 cases of oligodendroglioma of the spinal cord and filum terminale recorded in the literature.

A careful search of the literature from 1930 to 1947 disclosed an additional 98 intraspinal gliomas, histological classification of which was attempted. The exact pathologic diagnosis is subject to question in some cases, but there was no instance of an oligodendroglioma. Thus oligodendroglioma of the spinal cord is a very rare lesion. For this reason we feel it worth while to report a case in which operation was performed on May 13, 1948. The symptoms were of long duration, and there was an associated scoliosis. This case has been briefly reported by Bucy and Heimburger.

CASE REPORT

History. A LeC., a white male railway clerk, aged 31, was referred by Dr. Harry Hodges and admitted to the Chicago Memorial Hospital on May 11, 1948.

In 1932, at the age of 15 years, the patient was in an automobile accident, but sustained no injury that was noticed at the time. One month later he noticed protrusion of one of the lower left ribs, and was told that it was fractured. Two years after the accident "twitching movements" occurred in the muscles of the back, and his family noticed that his spine was crooked. At about the same time he began to have periods of clumsiness and numbness in both hands, lasting only a few minutes. Following 5 months of naprapathic treatments in 1937, the patient noted no fibrillations in the back muscles, until they returned in 1940. The "twitches" occurred at a rate of about 40 per minute, were most pronounced when the patient was sitting, and caused his entire body to jerk.

During the next 2 years there developed slowly progressive weakness and awkwardness of the legs and hands. He fell twice, due to weakness of the legs. His gait was peculiar and he carried one shoulder lower than the other.

Examination by an orthopedic surgeon in 1942 revealed a lower thoracic scoliosis with

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convexity to the right. The left shoulder was higher than the right. Rhythmical contractions of the left sacrospinalis musculature were palpable. A questionable decrease in pain sensation in the lower extremities was noted, but a detailed neurological examination was not recorded. X-ray films of the spine made at that time showed the scoliosis (Fig. 1).

On May 20, 1942, the orthopedic surgeon performed a spinal fusion with a 6 inch tibial bone graft placed on the denuded laminae from the 10th thoracic to the 3rd lumbar vertebra.

On June 9, 1942, a 2nd operation was done to correct the malposition of the tibial graft. Following this the wound became infected and drained for 4 months. After recovery from the operation the patient was completely relieved of the twitching in the back muscles. He had no weakness or clumsiness of the extremities, except that he considered the right leg a little weak, and favored it because the tibial bone graft had been removed from that side. In due time, he was able to return to work.

In December, 1946, the patient sustained burns on the thighs from a hot radiator, but felt no pain. Following this he had several minor traumata to the legs, without any sensation of pain. In the Spring of 1947, he noticed clonus of the right ankle while playing the organ. After that he had a progressive weakness of the legs, the right being most affected. By the Summer of 1947 he was unable to climb stairs or run, and had great difficulty in walking. In the year before his admission to the hospital, the patient had fecal and urinary urgency, and was incontinent if unable to relieve his urgency at once. He also complained of aching pain in the lower abdomen and back.

Examination. When admitted to the hospital on May 11, 1948, the patient could barely walk with the aid of two canes. His gait was spastic, with a broad base. The Romberg test was positive. He was unable to hop on either foot. There was generalized weakness of both lower extremities, most marked in the hamstring muscles, and greater on the right than on the left. The thighs and calves were markedly atrophic. The tonus of the lower extremities was increased, especially on the right. The knee jerks were hypoactive, but the ankle jerks were hyperactive. Ankle clonus and Babinski's sign were present bilaterally. Spontaneous flexor spasms were noted. The abdominal reflexes were present in the upper quadrants but absent in the lower quadrants. He could not feel pin prick below the 11th thoracic dermatome. Perception of warmth was absent below the 2nd lumbar dermatome, and of cold was present but diminished below the same level. Position sense was diminished in the right foot, and the ability to recognize numbers written on the skin was absent in both legs. Tactile sensibility was normal. No neurological defect was found in the upper extremities or in the cranial nerves. General physical examination revealed a lower thoracic scoliosis with convexity to the right, and a surgical scar from the 9th thoracic to the 3rd lumbar spinous process.

On lumbar puncture the initial CSF pressure was 160 mm. fluid. Bilateral jugular compression gave a rapid rise in pressure to 300 mm., but on release of the compression the pressure fell slowly and hesitantly back to the original level. After removal of 7 cc. of fluid, the pressure