TUMORS OF THE FORAMEN MAGNUM OF SPINAL ORIGIN

EDMUND A. SMOLIK, M.D.,* AND ERNEST SACHS, M.D.†
St. Louis, Missouri

(Received for publication August 10, 1958)

TUMORS in the region of the foramen magnum may be divided into two groups: (1) those arising primarily in the posterior fossa and extending downward into the foramen magnum and (2) those arising primarily in the upper cervical cord and extending upward into the foramen magnum. The former are essentially brain tumors and the symptoms and signs in this group are primarily those of brain tumors; the latter are more strictly spinal tumors and these are spoken of as spinocranial and cause the symptoms of a spinal tumor.

The occurrence of such spinal tumors is relatively rare. In the series of 234 verified spinal cord tumors from the Sachs collection, there were 6 verified cases or an incidence of 2.5 per cent. It seemed worth while to call attention to their occurrence and as far as possible to correlate some of the common characteristics of their symptomatology.

Other case reports and collected series indicate their relatively rare occurrence but this may suggest a higher unrecognized incidence. Elsberg and Strauss,6 in a report of 185 cases of spinal cord tumors, found 3.7 per cent in the foramen magnum. Love and Adson10 reported 23 cases, 12 of which were definitely craniocaudal. Elsberg,5 Abrahamson1 and Symonds and Meadows,16 attempted to describe the findings in foramen magnum compression. Strully, et al.19 called attention to the protean and insidious neurologic manifestations of high cervical lesions caused by compressions other than neoplasms. Piehl, Reese and Steelman12 called attention to the spinocranial syndrome as described by Bogorodinski. Until recently, the diagnosis has been infrequently made ante mortem and still less often has surgical treatment been performed. That the actual incidence of these lesions may, however, be greater than heretofore suspected, is suggested in a report by Bennett and Fortes.2

Study of the cases recorded in the literature,4,7,11,13,17 as well as the cases reported here, indicates that the capricious and bizarre picture frequently diagnosed as demyelinating disease or infectious disease of the central nervous system may, in fact, be caused by a tumor at the foramen magnum.

* Associate Professor of Neurological Surgery, St. Louis University School of Medicine, St. Louis, Missouri.
† Research Associate, Departments of Surgery and History of Medicine, Yale University School of Medicine, New Haven, Connecticut. Formerly Professor of Clinical Neurological Surgery, Washington University School of Medicine, St. Louis, Missouri.
The true diagnosis can be arrived at only by careful correlation of symptoms, signs, spinal fluid dynamics, chemistry and use of contrast myelography.

The following 6 cases of spinocranial tumors describe the train of symptoms and signs encountered in our series. In 5 instances the diagnosis was made and operative procedure was carried out, and in 1 case the diagnosis was not established until post mortem. These cases also illustrate the gravity of surgical interference when the disease is far advanced.

**CASE REPORTS**

*Case 1.* #9507. H.V.D., a 46-year-old white female, was admitted to Barnes Hospital on Feb. 12, 1942. Her illness had begun in the Fall of 1941 when her neck became stiff and ached a good deal, at times so severely as to make turning of the head impossible. This persisted in spite of osteopathic treatments.

Seven weeks before admission, numbness and tingling developed in the fingers of both hands, more marked on the left. These sensations spread progressively up her arms, involving her chest. She stated that her lungs felt tight and heavy. The sensation of heaviness spread through the abdomen and finally to her lower extremities. As the numb, dead feeling became more severe, the patient lost the use of her extremities except for a slight movement in the right hand and right leg. Throughout this period, the symptoms were more marked in the early morning, improving during the day. She was hospitalized at another institution, where, 4 nights before entry into Barnes Hospital, another woman, who was mentally deranged, attempted to choke her. From that time on the patient was unable to speak above a whisper. No change was noted in function of her bowels or bladder until 8 days before entry, when constipation became extreme. On the night before entry, she had incontinence of urine. There had been a gain of 25 lbs. in weight in the last 6 months.

*Examination.* Temperature was 37.5°C., respiratory rate 24 per min., pulse rate 110, and B.P. 120/75. The patient was a well developed, obese woman, apparently not suffering from any pain and quite adjusted to her environment. She was alert, cheerful, cooperative and showed no signs of apprehension.

There was no involvement of the cranial nerves. There was slight tenderness at the base of the skull on passive movement of the neck but no rigidity. At the 2nd or 3rd cervical dermatomes there was a disturbance in the temperature sense forming a collar type of deficit, but this was not constant. Deep reflexes were hyperactive and equal but superficial reflexes were absent. There were bilateral Babinski and Hoffmann signs. The tone of the rectal sphincter was poor.

*Laboratory Data.* Blood and urine were normal. Kahn was negative. Lumbar puncture and Queckenstedt test were performed on Feb. 13, 1942. The initial pressure was 190 mm. of water. When the left jugular was compressed there was good response but not on compressing the right. Lumbar puncture on Feb. 16, 1942, showed a rise when both jugulars were compressed. The fluid was turbid and pinkish in color. The cell count, with acid, was 9; without acid 234, nearly all fresh red cells. Pandy was 4 plus; Wassermann negative; colloidal gold curve 0001111223; and total protein 1,173 mg. per cent. A second lumbar puncture performed on the same day showed 3,700 red cells without acid, 17 cells with acid; total protein was 1,043 mg. per cent. A provisional diagnosis of Guillain-Barré neuronitis was made.

*Course.* On Feb. 17, 1942, her temperature suddenly rose to 41.0°C. Her pulse rate was 130, and respiratory rate 30. Respirations gradually increased in depth, with apneic pauses of 30 to 40 sec. occurring every 5 to 10 min. Pulmonary edema de-