A CASE OF SYRINGOMYELIA

WITH REPORT OF OPERATION *

EDWARD D. FISHER, M.D.
NEW YORK

History.—J. P. M., man, aged 35, American, a clerk by occupation, whose mother died of carcinoma, denied venery and alcoholism, and does not remember of having been ill.

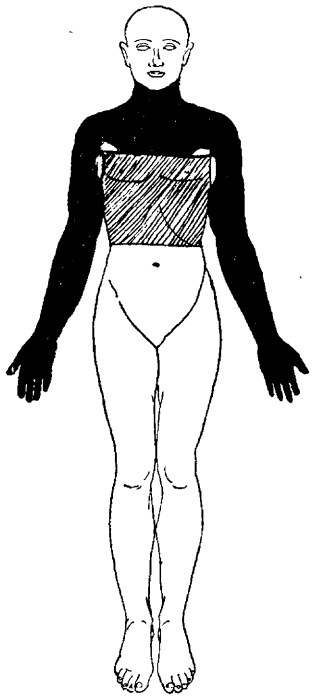

Fig. 1.—Anterior aspect before operation: dark shading, loss of pain and temperature sense; light shading, hypalgesia and therm-hypesthesia.

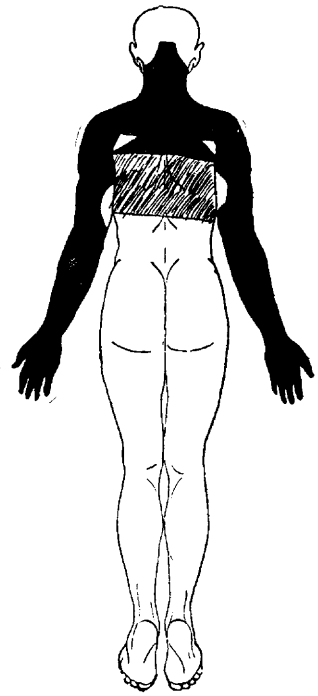

Fig. 2.—Posterior aspect before operation: dark shading, loss of pain and temperature sense; light shading, hypalgesia and therm-hypesthesia.

In 1905 he was thrown heavily to the ground in a football game, being rendered unconscious for sixteen hours and sustaining a fracture of the right femur. There was no escape of arachnoid fluid from the ears, nose or mouth. After having been eight weeks in bed and attempting to get about, he noticed that he could not walk on account of a paretic condition in his legs. At that time there was no subjective sensory disturbance in the lower extremities. Soon, however, a numbness and tingling sensation made their appearance in the upper extremities and back of the left ear. There was no perceptible impairment of the motor power in the upper extremities. The paresis in the legs improved within a few days, and the sensory disturbances in the arms disappeared within two months, while that behind the left ear remained, and still persists. In this fair condition of health he remained until 1910, when paresthesias in the arms appeared, culminating soon in complete loss of sensation, gradual loss of power, with wasting, especially in the muscles of the forearms. During 1911 he had a series of six epileptiform seizures within two days, with complete loss of consciousness and biting of tongue. No aura preceded the attacks. So far as the patient is able to learn, the attacks were generalized throughout the whole body. The convulsions lasted from ten to fifteen minutes. After a week's interval he again had an attack and one week later another one, similar to the original ones. Immediately after these attacks he became hypersensitive to touch and pain between the shoulders. The numbness, wasting and loss of power in his arms gradually grew worse till 1914, when he became completely paralyzed in both upper extremities. Early in 1915, a motor weakness manifested itself in his legs, his gait became somewhat spastic and staggering, especially pronounced when under mental stress as a result of fright. He would then experience great difficulty in walking. Yet there was no subjective sensory disturbance.

Examination.—Feb. 7, 1916, he was admitted to Bellevue Hospital to the service of Dr. George D. Stewart. Examination revealed a complete flaccid paralysis of the upper extremities with considerable wasting of the muscles of the forearms; there were some spasticity and motor weakness in the lower extremities. None of the cranial nerves were involved.

The reflexes of the upper extremities were abolished; those of the abdomen and lower extremities were markedly exaggerated, and there was a double ankle clonus and Babinski's reflex.

Sensation was tested, April 24, 1916, and is outlined in Figures 1 and 2. There was complete loss of touch, pain and temperature sense in both upper extremities and chest and neck up to the fifth ribs, corresponding to the lower six cervical and first, third and fourth dorsal segments of the cord. There was also a zone of hypalgesia and hypesthesia on both aspects of the chest between the fifth and eight ribs, corresponding to the fifth, sixth, seventh and eighth dorsal segments. In addition, there was a line of marked hyperesthesia of about 2 inches in width along the vertebral columns between the sixth cervical and eighth dorsal vertebrae.

The Wassermann test of the blood serum gave five units, February 12, and negative, March 23, 1916; that of the spinal fluid was negative, March 28, 1916. The cell count of the spinal fluid was: seven cells per cubic millimeter, all mononuclears; globulin, faintly positive.

In view of these findings, a tentative diagnosis of a cord tumor was made, probably a glioma, and operation was decided on.

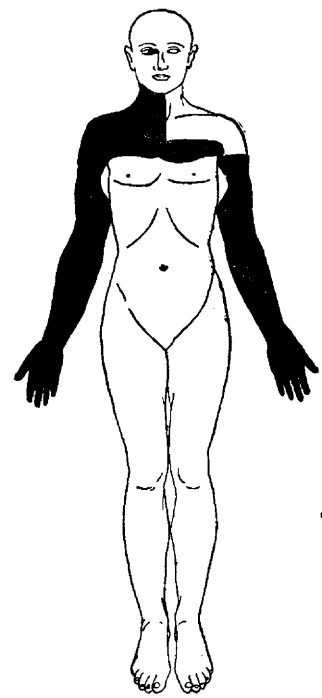

Fig. 3.—Anterior aspect after operation: dark shading, loss of pain and temperature sense.

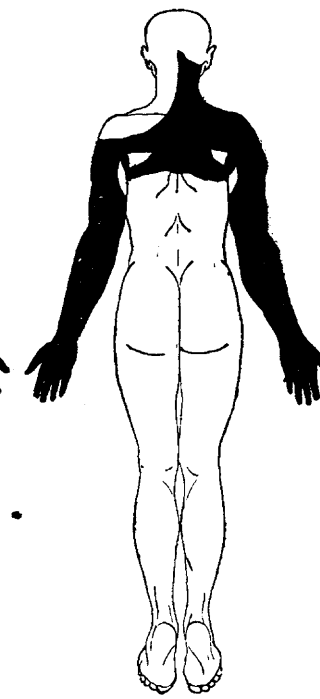

Fig. 4.—Posterior aspect after operation: dark shading, loss of pain and temperature sense.

Operation and Result.—The patient was operated on by Dr. Stewart, May 4, 1916. The central canal was found enlarged and a probe could be passed upward and downward.

The flaccid paralysis of the upper extremities still persists, but the patient is able to flex the fingers somewhat. The wasting is quite marked in the entire extremities. Otherwise the patient is physically improved. The eyegrounds are normal.

* Read before the Section on Nervous and Mental Diseases at the Sixty-Eighth Annual Session of the American Medical Association, New York, June, 1917.

The reflexes of the upper extremities are completely abolished. The abdominal reflexes and those of the lower extremities are exaggerated. There is a double ankle clonus and Babinski's reflex. The pupils are small, equal and regular; the response to light is sluggish, especially in the right pupil; accommodation is good in both.

Sensation of the anterior and posterior aspects on the right side is as shown in Figures 3 and 4. There is complete loss of touch, pain sense in the arm and the chest corresponding to the lower six cervical and first, third and fourth dorsal segments of the cord. On the left side there are complete anesthesia and analgesia in the arm and part of the chest corresponding to the lower four cervical and first, third and fourth dorsal segments. There is also a hypesthesia on the face corresponding to part of the right second cervical segment. The thermanesthesia is complete on both aspects of the neck and chest as far down as the sixth ribs.

The left pronator radii responds to a strong faradic current. All other muscles of both upper extremities do not respond to faradic current, and show a complete reaction of degeneration.

46 East Fifty-Second Street.

ABSTRACT OF DISCUSSION

DR. I. ABRAHAMSON, New York: We had a similar case at the Montefiore Home. The provisional diagnosis was a central tumor of the cord. An unusual feature of our case was the existence at the level of the lesion of an action myotonia, that is, a spinal type of myotonia. At the operation a myelomalacia was found. The cord was incised and improvement followed. The occurrence at the level of the lesion in Dr. Fisher's case of involvement of tactile and pain and temperature sensibility indicated that the lesion was at or very close to the posterior roots. Below this level the pain and temperature sensibilities alone were involved.

DR. E. D. FISHER, New York: A possible origin of this disease was hemorrhage into the cord at the time of injury, and at the site of the hemorrhage we found the beginning of the glioma later. I think without question the nerves were affected.

INTERESTING REACTIONS INCIDENTAL TO THE TREATMENT OF TWO CASES OF BRONCHIAL ASTHMA *

FRANCIS M. RACKEMANN, M.D.
Assistant in Medicine, Massachusetts General Hospital; Alumni Assistant in Medicine, Medical School of Harvard University
BOSTON

It is now well known that naturally sensitive individuals give a skin test to the proteins to which they are sensitive and thus presumably have many antibodies fixed in the cells of their skin. In connection with other work done in New York in 1915-1916, Dr. Longcope and I attempted to demonstrate circulating antibodies either as precipitins or as so-called anaphylactins in the blood serum of these naturally sensitive individuals, but always with a negative result. Cooke[1] could find no circulating antibodies in the blood of hay-fever patients. Schloss[2] and also Koessler[3] succeeded in finding circulating antibodies in a case of egg susceptibility and hay-fever, respectively, and recently Walker[4] has reported the irregular appearance of complement fixing antibodies and precipitins in patients naturally sensitive to horse hair and cat hair proteins.

In view of the fact that, in the case of artificial sensitization, circulating antibodies appear immediately after the subsidence of the anaphylactic symptoms, as Dr. Longcope and I were able to show,[5] it has seemed worth while to study and report two interesting cases of natural sensitiveness in patients who, during treatment for their asthma, showed anaphylactic symptoms after injection of the offending protein, since circulating antibodies should be expected at this time if ever.

One patient was a woman of 33, who had had asthma for fifteen years. The other was a man of 39, with asthma for thirty years. In both patients the asthma came on in attacks which were often associated with contact with horses or other animals. Both patients gave markedly positive skin tests to an extract of horsehair as well as to extracts of other animal proteins. In both patients there seemed to be no other cause of asthma and they were, except for asthma, apparently normal persons. In view of the fact that their troubles were presumably due to a great excess of antibody in the cells, which fixed the offending protein to the cells and there split it to its poisonous elements,[6] it semed justifiable to attempt to reduce the number of fixed antibodies by small repeated doses of the antigen — in these cases horsehair.

This antigen was a Ringer solution extract of crude horsehair, which had been previously treated with an alcohol and ether mixture to dissolve the fat and dirt. The extract was sterilized by passage through a Berkefeld filter and was standardized by determining the total nitrogen content by the ordinary Kjeldahl method.

The woman received subcutaneously, on successive days (in terms of nitrogen content), 0.01, 0.01 and 0.02 mg. On the fourth day the dose (0.03 mg.) was introduced into the outside of the left thigh a little above the knee. Immediately following the injection a little blood appeared in the syringe so that presumably the dose had been injected into a vein, although no vein showed under the skin at that point.

Within two minutes she complained of being full and hot; within three minutes she had a definite coryza with red running eyes and flushed face, and complained of a burning, prickling sensation, especially over the head and upper trunk.

Within five minutes definite asthma began, associated with a choking sensation in the throat and an anxious, worried expression. Twelve minims of a 1:1,000 solution of epinephrin (adrenalin chlorid, Parke, Davis & Co.) was injected subcutaneously at this time, and in ten minutes afterward the asthma was much relieved.

In thirty minutes after the dose of extract the severe asthma was practically over, though there was still a little wheeze. The redness, heat and burning of the arms had become a general urticaria which spread over the arms, chest and back. In one hour the asthma had gone, the urticaria was clearing, and in an hour and a half the patient was practically normal again.

The man was given eight desensitizing doses of horsehair extract at intervals of three or four days, the doses gradually increasing from 0.006 to 0.65 mg. During this time his asthma was perhaps a little better,

* From the Medical Clinic of the Massachusetts General Hospital.

1. Cooke, R. A.: Jour. Immunol., 1917, **2**, 217.
2. Schloss, O. M.: A Case of Allergy to Common Foods, Am. Jour. Dis. Child., June, 1912, p. 341.
3. Koessler, G. L.: The Specific Treatment of Hay-Fever (Pollen Disease), in Forchheimer, Therapeusis of Internal Diseases, **5**, 671.
4. Walker, I. C.: Jour. Med. Research, 1917, **31**, 243.
5. Longcope and Rackemann: Proc. Soc. Exper. Biol. and Med., 1916, **13**, 101.
6. Weil, Richard: Jour. Med. Research, 1915, **32**, 107. Vaughan: Protein Split Products, Lea and Febiger, 1913, p. 324.