Society Proceedings.

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The President, DR. ALFRED GORDON, in the Chair.

A CASE OF CEREBRAL MONOPLÉGIA.

By Horace Carncross, M.D.

In April, 1906, this patient (a woman of 39 or 40 years), after a period of overwork and worry, became paralyzed on the left side while in bed at night. She could not be awakened in the morning and remained unconscious for three weeks. No history of convulsions was obtained. At the beginning of the attack the mouth was drawn and the eyes were turned to the right. For a week after gaining consciousness the patient states that she was irrational. Then her mind became clear, and she also became able to draw up the left leg, which, as well as the left arm, had been utterly powerless. About two months after the onset of the attack she began to walk. The arm improved little until the next winter.

At the time of examination (October, 1907) the power of the right side was good. The left arm could be raised slightly, but the forearm could not be flexed unless the arm was raised so as to be assisted by gravity. There was no power of pronation or supination, there was complete wrist drop and an extremely faint grip. The left lower limb showed power practically equal to that of the right side. The face, which on casual glance appeared symmetrical, showed slight traces of the former paralysis in the lower part of the left side, with an extremely slight deviation of the tongue to the left.

The gait, station, coordination of right arm, cranial nerves, electrical reactions, sensations for pain and touch, and superficial reflexes were all normal. But the deep reflexes were universally increased, and those of the left arm were markedly exaggerated. There had been a clonus of the fingers of the left hand, which could not at this time be developed. There was practically no wasting.

The heart, which was enlarged to the left, presented a systolic murmur at the apex, with a relatively accentuated second pulmonic sound, and the pulse was irregular in force and rhythm. There was a history of two attacks of rheumatism in years gone by. The urine was negative.

The cerebral monoplegia which followed the original complete left-sided hemiplegia, was diagnosed as due to an embolism of the ascending frontal branch of the middle cerebral artery of the right side.

Dr. C. K. Mills stated that the case though simple in its aspects, is interesting because cortical monoplegia is comparatively rare. In regard to the loss of sensation, he thought it possible that the patient might have had a temporary loss of sensation and yet the lesion have been

largely restricted to the pre-central convolution. It is well known that in embolism we sometimes have an effect beyond the locality of the obstructive lesion, so that the post- and pre-central convolutions may have both been involved in the acute condition of the lesion. The impairment of sensation in such cases from purely motor lesion, may be due to disruption of the neuronal associations between the sensory and the motor area.

Dr. A. A. Eshner said he wondered whether it was perfectly precise to designate this case as one of monoplegia. Although Dr. Carncross demonstrated that the paralysis predominates in one upper extremity, he also pointed out that the reflexes in the lower extremities are exaggerated, which must indicate some involvement of the motor fibers passing to these parts. With respect to the situation of the lesion, the question naturally arises whether in the presence of a cortical lesion one would not have a right to expect some convulsive tendency, either a convulsion at the inception of the disease, or epileptic seizures since then.

Dr. Spiller said there are a few cases of monoplegia from lesion of the internal capsule on record. The case presented by Dr. Carncross, he thought, must be regarded as cortical in origin. The middle cerebral supplies the whole of the motor cortex of the brain except the extreme upper part. Dr. Spiller has a brain in which there was complete destruction of the middle cerebral and of the whole Sylvian distribution, yet the uppermost part of the pre-central convolution and the whole of the para-central lobule were preserved, as that portion of the brain is nourished from the anterior cerebral artery. In that case the fibers coming from the para-central lobule were destroyed within the white matter of the brain by the sclerotic lesion and there was hemiplegia. Dr. Spiller had another brain in which there is destruction of the cortex in the central convolutions only in the upper part, in the center for the upper limb. In the case presented by Dr. Carncross, we may suppose there was an embolus of the terminal portion of the middle cerebral artery, and that the cortex of the upper limb center was destroyed; it is possible that the anterior cerebral artery extended further downward on the lateral surface of the brain than in most persons, and that the center for the lower limb has been chiefly supplied by this artery.

Dr. Spiller doubted whether anyone can find in the literature a case of cerebral monoplegia of the upper limb without some involvement of the face. Monoplegia is to this extent a misnomer. He did not observe any weakness of the orbicularis palpebrarum muscle or of the flexors in the lower limb in this case that Dr. Carncross had shown.

Dr. Carncross did not know whether convulsive seizures had ever occurred. The increased reflexes are in the right and left leg and right arm, and especially in the left arm.

A CASE OF UNILATERAL HEMORRHAGE IN THE MEDULLA FOLLOWED BY SYRINGOMYELIC SENSORY DISTURBANCES.

By Alfred Gordon, M.D.

A man of 48 was suddenly seized with an intense vertigo, objects turning to the right. He soon lost consciousness. An hour later there was inability to swallow and expectorate, also shortness of breath. Upon examination were found right facial palsy, narrowness of the right palpebral fissure, with marked tremor of the tongue, difficulty in moving the