ported studies of the first motor neurone in a man born without the left forearm. He now gives the results of his investigations of the other neurones. He found the first and second sensory neurones absent and the second motor neurone probably lacking.

4. *Motor Aphasia*.—(A continued article.)

J. M. MOORE (Beacon, N. Y.).

MISCELLANY

A CLINICALLY AND ANATOMICALLY EXAMINED CASE OF ISOLATED LOSS OF PUPIL-REFLEXES WITH ABSENCE OF PARALYSIS, TABES AND CEREBRO-SPINAL SYPHILIS. M. Nonne and Fr. Wohlwill. (*Neurol. Centralbl.*, 1914, No. 10.)

The authors report here upon a case of isolated loss of pupil reflexes, clinically and anatomically examined, in which the spinal fluid was examined for cell content, increase of globulin, Wassermann's reaction with negative result, and in which the brain and spinal cord were anatomically examined, without discovering a central nervous affection of syphilitic origin; moreover, signs of tabes and paralysis were wanting. Since the true loss of pupillary reflexes is rarely manifested from other causes, especially as the result of chronic alcoholism, and here lues had been present (infection thirteen years before), it could only be accepted that the isolated loss of pupil reflexes represents the clinical remnant of an earlier syphilitic, anatomical process which had spent itself. The authors accept this extinction of the process, since the fluid reactions are negative.

JELLIFFE.

CONTRIBUTION TO VAGOTONIA. W. Lublinski. (*Berl. klin. Wochenschr.*, 1915, No. 20.)

The vagus and the sympathetic act in opposition. If the organs provided with these nerves are to function normally both nerves must maintain an equilibrium. If one nerve overbalances vagotonia or sympathicotonia appears. The first is the more frequent. The author frequently had opportunity to observe: Laryngospasm, asthma, with complaints of cardiac and respiratory difficulties. The diseases of youth are mostly concerned, often through lymphatic symptoms, with glandular swellings, enlarged tonsils, and frequently also enlargement of the thyroid. The bluish, glistening flush on the face, outbreaks of perspiration, cold, bluish hands are striking. The palpebral fissure is narrow, the pupils are small and the eyes lusterless. Frequent swallowing movements are made on account of the excess of saliva. On the upper part of the body may be noticed a mottled redness, dermatographia. Stimuli in the region of the vagus may cause attacks of retarded, temporarily intermittent heart movements. Pressure on the eyeballs may cause that. The respiration is shallow, face pale, Aschner's phenomenon. Also on lying down a marked retardation of the pulse appears. Similarly it comes on in a squatting position or on bending the body forwards. Moreover, arrhythmia of the pulse may be observed, extrasystole. Whether it has to do with heightened irritability of the heart or injury of the heart muscles, the atropin test will decide. With those suffering from vagotonia even slight stimuli suffice to arouse alterations in the pulse, and so also will repeated rising in bed. A characteristic respiratory disturbance is cardiac obstruction in breathing, with laryngospasm and asthma. Automatically there is impulsion to deep breathing with a convulsive sensation in the upper air-passages. Pilocarpin can produce these phenomena artificially. The author looks upon vagotonia as a