In cerebral softening, after the loss of a greater or less number of teeth, and in organic disease of the tongue, a thickening of speech occurs, which does not, however, at all resemble dysphasia. In stuttering, speech is too rapid, in dysphasia it is slow. In the difficulty of speech which sometimes accompanies chorea, there is some resemblance to dysphasia, but in chorea the ataxy may be observed during repose of the parts, while in general paralysis it only accompanies voluntary movements. The trembling of the lips seen in anger, indignation, and fear, disappears when the patient becomes calm. In multiple cerebral sclerosis the difficulty of speech closely resembles that of general paralysis, and the other symptoms presented must determine the diagnosis. When a patient maintains absolute silence, the characteristic trembling of the lips may be observed, if the face is made to assume the expression of suffering.

Conclusions.—In general paralysis there are always lesions of the floor of the fourth ventricle, consisting chiefly of proliferation of the cells of the interstitial tissue, leading to destruction of the higher elements, notably of the cells of the grey matter of the nuclei of the facial and hypoglossal nerves.

These lesions manifest themselves symptomatically by labial and lingual ataxy, that is, any movement of determined extent and direction is preceded, and, still more, followed by a series of movements in the same direction but of less extent.

The repetition of movements direct and opposed, which precede and follow a given movement, varies with the extent and rapidity of that movement.

Dysphasia is the natural and necessary result of the labial and lingual ataxy.

Dysphasia may be manifested in the eight forms already enumerated.

The general paralytic tries to lessen the dysphasia by speaking slowly and closing his jaws.

The intimate connection between the labial and glossal ataxy and dysphasia stamps the latter as altogether peculiar to and pathognomonic of general paralysis.

On General Paralysis as a Predisposing Cause of Fractures.

René R., an advanced general paralytic, fifty years of age, while walking in the airing-court, fell on his right shoulder and fractured his humerus. Five days later he died, and on post-mortem examination, in addition to a long oblique fracture of the shaft of the bone, three fissures, more or less gaping, were detected. Microscopic examination of the bones revealed proliferation of the fatty elements in the medullary, spongy and hard portions. The denser portions were often much thinned, and from all parts a fatty oozing might be observed.

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