

While in the lower thoracic cord the area of gliomatosis involves the left posterior horn and causes atrophy of Clark's column and the posterior columns, on the right side at this level there appears sharply limited to Goll's column a complete degeneration and an accompanying atrophy of the fibres of the medial half of Clark's column. Farther down, that is in the upper lumbar cord, this degeneration of Goll's columns becomes diffuse, especially toward the right posterior root zone and right posterior column.

In the lumbar cord, the gliosis in the left posterior horn is confined to a very small area. The degeneration of the right posterior column is, however, very apparent.

The question which the author puts to himself: Is this a case of tabes dorsalis with syringomyelia, or a special form of gliosis which by its spreading out into the posterior columns has produced the symptoms of tabes?—he does not answer definitely, but leans to the latter supposition.

J. C.

Cortical Localization of the Movement of the Face.—Brissand. (*Le Progres Medical*, Dec. 31, 1893).

A man, eighty years old, who had for long suffered from myocarditis and a dilated heart secondary to emphysema, was stricken with an apoplectic attack in April, 1889. Loss of consciousness for about an hour, paralysis of the entire right side, and aphasia. Little by little, in the course of a few days, speech returned, the hemiplegia gradually passed away, and finally there was left but slight numbness and clumsiness of the fingers. Two years later, the patient entered the hospital on account of cardiac insufficiency. At this time the symptoms of the previous hemiplegia referable to the right extremity, had about disappeared. The face was absolutely asymmetrical, the mouth turned to the left, the left commissure of the mouth open, the right commissure firm, lowered, and allowed a continual dribbling of saliva. The left nostril less prominent, the ala of the nose on this side completely immobile, and the lines of expression are effaced. The right eyelid drooping; meanwhile, it is possible to close it voluntarily, but not so well as the left lid. The movements of the eyeball are well preserved, but the pupil of the right eye is greatly dilated, but it reacts to light and distance. The lines on the forehead on the right side are quite effaced, but are very pronounced on the left side of the median line.

On autopsy of the brain, a cortical lesion, an area of yellow softening situated in left Rolandic operculum, just behind the frontal operculum, was found. The softening extended down to the Island of Reil. There was no other superficial lesion of the hemispheres. The two peduncles were equal without tracts of degeneration, but on examination with the microscope it was possible to recognize a great number of granular bodies in the neighborhood of the inner part of the left peduncle. The pons, the medulla, and the pyramids were symmetrical.

The author concludes that the centre for the movement of the face in man is in that portion of the ascending parietal operculum situated just posterior to the inferior extremity of the fissure of Rolando. J. C.

Oxaluria and its Relation to Nervous Disease.—I. Adler, M.D. (*Med. Record*, June 3, 1893). Oxalic acid is a normal, though possibly not a constant constituent of the urine. The amount present in a given quantity of urine can be determined with any degree of reliability, only by quantitative analysis. All approximations by means of microscopic examination are untrustworthy. Its chief source is the oxalic acid in the food, though minute quantities probably are produced in the course of normal metabolism. Further investigation will be necessary to demonstrate, if and under what conditions morbid metabolism affects the production of oxalic acid. Impeded respiration, diseases of the heart and lungs do not of themselves tend to produce an excess of oxalic acid in the urine. The establishment of pathological oxaluria as a type of disease *sui generis* is not warranted by facts. The nerve symptoms assumed as characteristic of pathological oxaluria, are not caused by excess of oxalic acid in the blood and urine. Analysis will show that such excess is by no means as frequent as has often been assumed. When such excess does occur, not to be accounted for by ingesta, it is probably one of several symptoms of metabolic alterations primarily caused by disturbances of the nervous or digestive organs, or both, but no factor in the causation of disease. In considering the excretion of oxalic acid in the urine it is of the utmost importance to take into account at the same time the excretion of the other principal constituents, particularly urea and uric acid. A. F.