Fibrous Tumour of the Bulb [*Tumeur fibreuse du Bulbe*]. (*Rev. de Psychiat., July*, 1908.) *Vigouroux, M. A.*

The rare condition of a fibroma in the bulb is recorded. It occurred in a man, æt. 51, a drunkard who had received several head injuries, of which the most important and most recent had taken place six years previously, *i.e.*, a fracture of the right temporal bone. Mentally, for the eight months previous to his death, he was violently excited with hallucinations and ideas of persecution and grandeur. He died in a coma which followed a short convulsive attack of the right side. Several weeks before his death he had shown signs of defective equilibrium; he tended to fall to the left, stood on a broad base and was not able to stand erect alone.

At the autopsy no sign of the fracture could be seen, but the tumour could be seen in the angle of the bulbar protuberance, and there was some atrophy of the over-lying cerebellum, the left hemisphere weighing 30 grms. less than the right. On section it was found to be conical in shape, the base, measuring ·01 cm. in diameter, being superior. It lay in the left lateral half of the bulb, but did not reach the tubercle. It extended to just above the decussion of the pyramids. The overlying pia mater was thickened and contained a number of vessels.

The tumour was of firm consistency but without being hard, and its centre showed no liquefaction or calcification. Although to the eye it was easily discernible, yet it could not be shelled out. It had destroyed the nerve-fibres of the left restiform body, and those of the eighth and ninth nerves, but the olive and acoustic fibres were undisturbed. No other cranial nerve nuclei were affected.

Histologically it consisted of elongated fibres joined in loose bundles, crossing each other in different directions, and in its midst were a number of congested vessels, certain of which seemed to be undergoing a hyaline degeneration. The overlying pia was thickened, congested and fibrous, and connected with the tumour, which appeared to arise from it.

The author considers it possible that the growth was of traumatic origin. SIDNEY CLARKE.

Contribution to the Pathological Anatomy of Multiple Sclerosis, with Particular Regard to the Cerebral Cortex [*Zur pathologischen Anatomie der multiplen Sklerose mit besonderer Berucksichtigung der Hirnrindenherde*]. (*Neur. Cbl., Nr.* 19, 1908.) *Oppenheim, G.*

The examination of the cortex of the brain in multiple sclerosis shows that there exists a peculiar condition of the affected areas, in that these contain no compact fibrous glia proliferation, but an increase of the net-like protoplasmic glia structures. Ætiologically it is of particular importance, that in three of the four cases cited there was found a diffuse perivascular infiltration of plasma cells analogous to those seen in progressive paralysis (Nissl, Alzheimer), and in trypanosomic diseases (Speilmeyer). A principal difference, however, lies in the fact that in these diseases there are very marked degenerative changes of the ganglion cells, while in multiple sclerosis these changes are inconsiderable, but in spite of this difference, the diffuse infiltration

of the vessel sheaths with plasma-cells may be accounted for in the way explained by Nissl and Alzheimer, *viz.*, as the expression of a more or less chronic inflammatory process, which points to an exogenous origin of the disease. HAMILTON C. MARR.

The Pathogenesis of Epilepsy: An Experimental Research [*Sulla Patogenesi Dalla Epilessia*]. (*Riv. Speriment. di Freniat., vol. xxxiv, fasc. i–ii.*) *Guidi, G.*

In an article extending over forty pages and accompanied by a number of diagrams showing the quantities of urine, total nitrogenous excretion, amount of urea, ammoniacal constituents, etc., in several cases of epilepsy and hysteria under examination for successive days, Dr. Guidi discusses the pathogenesis of epilepsy.

In spite of numerous researches the fundamental nature of epilepsy is still obscure. There is a variety of opinion regarding the pathological findings in epilepsy. The majority of writers, however, agree in attributing the disease to irritation of the cortical centres. The source of this irritation has naturally been the object of much research. Féré attributes epilepsy to a convulsive poison in the urine of the epileptic. Agostini is of opinion that leucomaines are the source of irritation. Other authors point out that the urine before the fits is hypo-poisonous and after them hyper-poisonous; others that epilepsy is due to intestinal auto-intoxication; others, again, that the epileptic organism retains a large quantity of urea, and it is this agent that acts as a poison in the disease.

Weber is of opinion that in epilepsy the bodily tissue is modified in a pathological direction and poisons are thus produced from the secretions. The poisons act on every organ, on the vessels, the kidneys, and above all on the central nervous system.

Kransky states that the poison inducing epilepsy is carbamic acid derived from uric acid. Carbamic acid is a poison chemically intermediate between carbonate of ammonia and urea. It is produced in the transformation of the albuminoid molecule.

According to modern views, urea and uric acid have different modes of origin. Urea is formed in the assimilation of the albuminoid products of the organism, and is mainly due to ammoniacal bodies produced by the cellular elements, or formed during the digestive processes, while uric acid is derived from the dissolution of the cellular nuclei, particularly the nuclei of leucocytes.

The author made daily and minute examinations of the nitrogenous products of the urine of epileptics. The subjects were kept, some of them on mixed diet, others on vegetarian. In all cases the excretion of ammoniacal products was above normal. From a minimum the values of these products rose to a high maximum. This coincided with the epileptic attack; after fits the values continued higher for a day or so, and then they declined.

The author is of opinion that the organ which is the cause of epilepsy is the liver; that it is the organ which presides over the transformation of the albuminoid molecule. There is a deficiency in the capacity of the hepatic cells of the liver to complete the synthesis of