modified in katatonia, and it would be interesting to note whether similar vagotonic alterations might not be discovered there also.

W. McC. HARROWES.

The Anatomic Substratum of the Convulsive State. (*Arch. of Neur. and Psychiat., May*, 1930.) *Spielmeyer, W.*

The author gives an account of his own work, especially on the sclerosis of Ammon's horn. This area of sclerosis when stained by Nissl's method shows a loss of ganglion cells. In the earliest stages of this condition, there is a loss of cells and an increase of glia cells of the rod form on the border between the area attacked and the normal tissue. These changes are almost as frequent in the cerebellum, where there is a proliferation of the fibroglia in the molecular zone. Spielmeyer considers these changes to be closely related to the epileptic attacks and probably of circulatory origin. He found similar changes in arterio-sclerosis, thrombosis and embolism. The localization in Ammon's horn and in the cerebellum is due to the unsatisfactory blood supply of these areas. One of Spielmeyer's co-workers, Neuberger, has demonstrated recent destruction of muscle elements in the heart without organic changes in the coronary arteries.

G. W. T. H. FLEMING.

The Problem of Localization in Experimentally Induced Convulsions. (*Arch. of Neur. and Psychiat., May*, 1930.) *Pike, F. H., Elsberg, C. A., McCulloch, W. S., and Chappell, M. N.*

The authors believe that both the rubro-spinal and pyramidal systems are concerned with the control of movement. The pyramidal is the main motor system responsible for clonic convulsions. Before the pyramidal fibres have become myelinated and attained their full functional capacity, clonic convulsions arise from lower motor mechanisms. When the central nervous system is otherwise intact, clonic convulsions are of cortical origin. Tonic convulsions arise from the lower motor mechanisms in the period immediately succeeding an injury to the cortical motor mechanisms. They are absent in the early post-operative stages in animals in which the midbrain has been split longitudinally in the mid-line, while clonic convulsions persist if the pyramidal system is intact.

G. W. T. H. FLEMING.

Diagnostic Significance of Sensory Auræ in Epilepsy. (*Brit. Journ. of Med. Psychol., May*, 1930.) *MacCurdy, J. T.*

The author investigated the auræ of a large number of patients at the Manhattan State Hospital and the Craig Colony. The first conclusion he came to was that no such thing as a pure motor aura existed. The idiopathic aura is either sensory or psychic, and is invariably accompanied by an emotional reaction which is always of a painful nature, and is usually fear. Every patient with a

definite aura always has attacks in which the aura alone occurs and does not lead to a fit. These MacCurdy looks on as psychoneurotic symptoms not followed by epileptic manifestations. The general sequence of events in cases with definite auras is somewhat as follows: There is first a prodromal stage lasting from a few hours to days, which is characterized by restlessness, depression, irritability or some general mood alteration. The patient, on the first appearance of the aura, makes some effort to distract his attention from it; he feels that success depends on will-power. In the last stages the patient finds the sensations of the aura so engrossing that he can give no attention to anything else. His fear culminates in the belief that the fit is now inevitable and then consciousness is lost. Distraction in some cases is a successful treatment of the aura. In Jacksonian epilepsy the aura is essentially motor in type. If a sensory aura is removed by any means the patient is apt to substitute another one.

G. W. T. H. FLEMING.

Interpretation of Encephalographic Observations; Comments on Those Found in the Convulsive State. (*Arch. of Neur. and Psychiat., May*, 1930.) *Pendergrass, E. P.*

The author found in the convulsive state atrophy of the brain and arachnoiditis. From a roentgenological point of view it is possible to distinguish two main groups of atrophy—the superficial, presumably due to pressure, and the deep, which is ascribed to a lesion within the cortex. The author classifies atrophy of the brain into three groups: those due to (1) birth trauma, (2) trauma later in life, (3) those in which no factor could be assigned. In the first group in children the changes were very definite and consisted of atrophy or aplasia beginning in the frontal lobes. The area next affected was the parietal lobe in the region of the motor area. The atrophy extends towards the base, and finally one finds compensatory pathways in the occipital region which may be regarded as evidence of atrophy of the brain. These conditions may be unilateral or bilateral. The cisterna venæ magnæ cerebri is usually the largest compensatory space. In the post-traumatic group there exists atrophy of the brain or arachnoiditis, or a combination of both. The frontal region seems to be the first to suffer. In more advanced cases the parietal region over the motor area suffers. In the most advanced cases there is almost complete disappearance of the subarachnoid markings in the frontal and parietal regions. In some cases there may be cerebellar atrophy. In those encephalograms in which there is an absence of subarachnoid pathways arachnoiditis appears to be present. This may be localized on the motor area, and be the only positive observation in Jacksonian epilepsy. This has been confirmed at operation several times. The arachnoiditis is more often associated with Jacksonian epilepsy, and may be unilateral, bilateral, localized or associated with atrophy of the brain. In the idiopathic group the atrophy does not vary from that seen in the post-traumatic group. According to the