tations of the disease, and is accompanied by lesions identical in nature, affecting many groups of neurones situated entirely within the central nervous system; (5) The central changes are not attributable to the peripheral neuritis, and though in some cases the peripheral neurones are mainly affected, in others the morbid process chiefly implicates central neurones; but these two groups of cases do not appear to be sharply divided.

8. *Brain Defects*.—A short note on a defective development of dura mater, old meningitis and internal hydrocephalus occurring in a male idiot of 40 years.

9. *Pain*.—The author defines pain as a disagreeable sensation due to the stimulation of some portion of the cerebrospinal nervous system, and referred to the peripheral distribution in the external body wall of cerebrospinal sensory nerves. Pain in the digestive tract is felt, he believes, across the middle line of the abdomen and in regularly descending areas according as the region affected passes from the stomach to the great intestine. The pain of gastric ulcer is in the epigastrium, the higher the nearer the ulcer to the cardiac end of the stomach. Hyperesthesia and visceral disease he shows to be related, and he further speaks of a visceromuscle relation whereby visceral disease causes painful muscular sensations, hardness and tenderness of the abdominal muscles in peritonitis being an example. The sensibility of the viscera is an interesting subhead discussed by the author. A thorough reading by the author of Head's researches would have clarified many of his conjectures and hypotheses.

JELLIFFE.

MISCELLANY.

THE PATHOLOGICAL ANATOMY OF CHOREA MINOR. M. Reichart (Deutsches Archiv für klinische Medizin, LXXI, Nos. 5 and 6, p. 504, 1902).

Two cases of chorea minor are described, which after a brief period resulted fatally, and which upon examination showed anatomical changes in the central nervous system, consisting of inflammation, hemorrhages and degeneration of nerve fibers. The inflammation was characterized by perivenous, diffuse, small cell infiltration of varying degrees; but only in one area was this change visible microscopically. While in several places inflammatory and hemorrhagic changes were found, it is emphasized that the ganglion cells were but slightly affected, if at all. The many hemorrhages, some of which were macroscopical in size, were irregularly distributed, although present throughout the brain substance. These hemorrhagic areas were especially confined to those portions of the brain where the inflammatory changes were most marked, from which the author presumes that the hemorrhages were to be attributed to inflammatory changes in the blood vessels. The location of the fatty degeneration of nerve fibers showed that this change occurred independent of the inflammation, being absent at the central convolutions, as well as in the greater portion of the internal capsule, being present however in a marked degree in a portion of the optic thalamus, in the posterior portion of the internal capsule, in the transverse fibers of the knee of the internal capsule, as well as in the lateral portion of the pulvinar. In the spinal cord, the principal seats of degeneration were the root fibers, fibers in the anterior and lateral cornua, and here and there a fiber in the posterior columns. The anterior and lateral columns remained exempt from degenerative processes. The bacteriological examination of the brain was entirely negative in one case, and at the least doubtful in the other. Worthy of note, however, was the existence of endocarditis and pericarditis, as well as the presence of staphylococcus aureus in the blood within the heart, in one case, and the presence of streptococci in the deposits on the cardiac valves and the heart muscle, in the other case. The author seems inclined to classify these infectious cases of chorea minor with those cases of encephalitis occasionally occurring in the course of other infectious diseases. He attributes the described changes in the brain as a consequence of the influence of toxins. The diffuse dissemination of the

anatomical changes in both cases does not permit of a conclusion regarding the origin of the choreiform movements. The cortex, as well as that of the central convolutions, was certainly the seat of only slight changes. The question then arises whether the motor disturbances are to be attributed to the degeneration of nerve fibers which was found to some extent in the basal ganglia and their immediate vicinity. But the author calls attention to the fact, that in from seventy to eighty per cent of cases of post hemiplegic chorea it was in the posterior portion of the internal capsule, in the posterior region of the optic thalamus, or the adjoining portion of the lenticular nucleus, that the lesion existed. DINKELSPIEL (Philadelphia.).

CLINICAL STUDIES AND DEDUCTIONS CONCERNING FAMILY MYOCLONIA AND ALLIED DISEASES. Herman Lundborg.

The author whose name is closely associated with our present knowledge of family myoclonus presents us with a second and more exhaustive monograph of the above title. The author's preface states his study of the conditions dates from 1897. A short historical sketch and a brief résumé of cases by Friedreich, Homén, Unverricht, Sepilli and Bresler are given with the conclusions and the nosological relationship of family myoclonies with the other motor neuroses by different authors. Lundborg gives observations and investigations upon some seventeen cases of family myoclonies from his own clinics, and considers at some length the possible relationship between myoclonia, paralysis agitans, myxedema, Basedow's disease and dementia præcox. He holds that they may develop upon a common defect of the thyroid either structurally or functionally. The monograph is illustrated by photographs and accompanied by genealogic and metabolic tables in original case studies. The author concludes that family myoclonia is a distinct class of the myoclonies, autotoxic in nature, the pathology of which concerns the spinal cord. L. PIERCE CLARK.

UEBER NEUERE KLINISCHE GESICHTSPUNKTE IN DER LEHRE VON DER ARTERIOSCLEROSE. Grassmann (Münchener med. Wochenschr., 1902, No. 109).

The pathological anatomy of arteriosclerosis consists essentially in a diffuse thickening of the intima with a loss of the elastic structure of the media. Hypertrophy of the left ventricle takes place after somewhat advanced degeneration of the visceral arteries or thoracic aorta. The process however, may be localized in the smallest vessels. Persistent arterial tension and deficient nutrition to organs affected characteristic. Arterial strain may be very early recognized and is highly diagnostic as a premonitory symptom.

Syphilis, acute infections, alcohol, toxic substances, such as tobacco in excess, etc., are of etiological importance. Palpation of vessels, at times, of much service in examination. Persistent frequency of pulse on lying down, after erect position, quite significant if accompanied with accentuated aortic second sound. Arteriosclerosis of coronary arteries leads to angina pectoris. When the vessels of lower extremities affected, frequently have intermittent claudication. Small doses of iodide of sodium recommended, with milk diet. Baths of aqua carbonica stated, oftentimes, to have remarkable efficacy.

J. E. CLARK (New York).

ON THE PERMANENT CARE OF THE FEEBLE-MINDED. E. F. Pinsent (The Lancet, Feb. 21. 1903).

In dealing with various plans for the care of feeble-minded persons who naturally fall within the numerous well-defined classes of this condition, the author mentions the following as the last kind of work recently undertaken by the Birmingham After-care Committee. It had been known for some time that there were a large number of idiots, imbeciles, epileptics and feeble-minded persons who had not passed through the regular classes of the institutions, and were not on the ordinary after-care lists of the Committee. With the help of the School Board officials and others, a list of these cases was collected. After a sufficient number had been found, it