is greatly reduced by post-papillitic atrophy. These ocular symptoms are due to the oxycephaly; it is known that oxycephaly leads to increase of intracranial pressure, necropsies having shown that it is accompanied by thinning of the cranial bones, with flattening of the cerebral convolutions, and often also internal hydrocephalus, which appears to be provoked by a bending of the Sylvian aqueduct. Demole diagnosed a lesion, probably due to an endarteritis, in the region of the left Sylvian fossa, involving in front the sensori-motor area. [Leonard J. Kidd.]

Anton, A. ROENTGEN PICTURES OF THE HEAD IN DISTURBANCES OF DEVELOPMENT. [Neurol. Centralbl., Vol. XXXVII, No. 24, p. 817.]

A Roentgen picture of a case was obtained which showed the cerebellum to consist of only a minimal remainder and the posterior fossa to be narrowed and overgrown with bone. In another case a picture was obtained which showed a hypertrophy of the cerebellum with corresponding broadening of the posterior fossa. In this case the suprarenals were stunted and the thymus was enlarged. About thirty other Roentgen pictures of the skull were obtained on which various abnormalities were visible: enlargements of the cerebellar fossa, diminished size of the same, oxycephalia with distortion of the cerebellar fossa, etc. The following summing up of these cases was given: (1) The defective development or the early wasting of the cerebellum is manifested in reduced size of the posterior fossa and a compensatory growth of the bone of the same an, sometimes, by the steep angle of the clivus. (2) The overdevelopment of the cerebellum is also discernible in the Roentgen picture, the upper boundaries of the same being usually indicated by the sinus transversus and the ridge of bone at that place. Hypertrophy of the cerebellum may be discovered by the Roentgen photograph more easily than by any other method; it is only rarely accompanied by choked discs and tumorous phenomena. Where there is oxycephalia other anomalies of the skull are usually recognizable not dependent on the premature closing of the coronal suture, for oxycephalia is a partial phenomena of a general disturbance of development in which the inner secretory glands play a rôle. The discrepancies between the capacity of the skull and the space taken by the brain may be visibly perceived in life by means of the Roentgen photograph. The veins, especially on the convex upper surface of the brain, also become visible, and the Roentgen photograph is therefore a valuable guide in operations, permitting the veins to be avoided. [J.]

Schaffer, Karl. MICRO-MORPHOLOGY AND THE ANATOMICAL CHARACTERISTICS OF INFANTILE AMAUROTIC IDIOCY. [Zeitschr. f. d. ges. Neurol. u. Psychiat., 1919, Vol. XLVI, p. 1.]

The following abnormalities were discovered in a case of infantile amaurotic idiocy examined by the author: the regio precentralis showed a distortion of its boundaries so that the line of demarkation between the precentralis and the postcentralis was not situated in the fundus of the

central sulcus but in the occipital slope of the precentral convolution. The extension of the area striata could be traced on the convexity beyond the occipital pole, on the one hand, and, on the other, included nearly the entire lingualis convolution, so that the cortical visual field was much more extended than under ordinary circumstances. The area striata showed an overdifferentiation of the cytotoxic components, resulting in an extraordinarily richly stratified visual cortex. These are structural peculiarities of the monkey's brain and indicate the pithecoïd form of the brain in infantile amaurotic idiocy; that is to say, its primitive conformation. From these facts it may be conclusively inferred that this disease is due to an inferiority of organization with an endogenous tendency to early destruction. The tectonic anomalies in heredo-degeneration of the cerebellum cortex may assume one of two forms, namely, that of deviations of structure in the sense of a lower (pithecoïd) organization, or of a deviation in the sense of ontogenetic alterations. The first leads to amaurotic idiocy, the second to Huntington's chorea. One manifestation of the infantile amaurotic idiocy is the disturbance of development of medullary sheaths in the phylogenetically younger structures, where the medullary element fail to reach maturity, in marked contrast to that which takes place in the phylogenetically later formations (Rhombencephalon, mesencephalon, etc.), for here this element attains perfect development. Infantile amaurotic idiocy may, therefore, be called a disease of a phylogenetic system. The inroad of the disease in the inferiorly developed central organ leads to a rapidly progressing degeneration, resulting in a general destruction of the ectodermal hyaloplasm and, finally, of all the neural and glial elements. As the only form of hereditary degeneration which the author studied was infantile amaurotic idiocy, he cannot affirm with certainty that the other forms of hereditary nervous disease present the same characteristics, but morphological stigmata may be anticipated in other forms—and, among them, in the neuropathic diathesis. [J.]

Kraepelin, Emil. THE STUDY OF PSYCHIATRIC DISEASE FORMS. [Zeitschr. f. d. ges. Neurol. u. Psychiat., 1919, Vol. LI, p. 224.]

The time has long passed for regarding clinical psychiatric study as a fruitless task. The problem before psychiatrists of to-day is to define and group the various psychiatric disease forms. All effort must, in the future as in the past, be directed toward careful observation and accurate description of the clinical forms under which mental diseases make their appearance. The clinical signs are often deceptive, however, because quite different diseases have very similar disease pictures, or the symptoms may change even during the course of the same case. Besides, the personal equation is of very great importance in the determination of the symptomatology of mental diseases. The course and outcome of the same disease may not always be the same, and it is often difficult to distinguish between the real cause and the activating factor. In only a part of the cases are there autopsy findings, and these are not