

atrocious and unintelligible crimes. Such crimes, when primary schizoid tendencies are present, are symptomatic of a latent schizophrenia. From the medico-legal aspect, such criminals must be treated as if suffering from a manifest psychosis. The matter is of special importance in countries where capital punishment obtains.

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*Crime and Punishment: Contribution to the Study of the Psychology of the Psychopathic Delinquent* [*Crimen y castigo: contribución al estudio de la psicología del psicópata delincuente*]. (*Arch. de Neurobiol.*, vol. xiv, p. 579, 1934.) Garma, A.

The crime of the psychopath is a "symptomatic action" largely independent of the offender's will. If we punish the psychopathic offender we increase his mental conflicts and set up a vicious circle of offences and punishments. Investigation has shown that there are three main factors in the mechanism of these cases: unconsciousness of the motives for the offence, masochism, and a sense of guilt. The primary step in treatment is to discover the psychological motives which impel the psychopath to his anti-social conduct. Only when this has been done can we hope to make the offender comprehend the social reality by which he is surrounded. Punishment may have a place in this secondary process, but such punishment must be of the least possible intensity, and the offender must understand that it is not intended as a retaliatory measure.

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## 8. Mental Deficiency.

*Amaurotic Family Idiocy: Genealogical, Clinical and Histopathological Studies of the Infantile Form.* (*L'Encéphale*, vol. xxix, p. 505, Sept.-Oct., 1934.) Bertrand, I., and van Bogaert, L.

The importance of racial factors is stressed. The incidence of the disease predominatingly amongst Jewish or partly Jewish peoples is in no doubt. Consanguinity is less certain as a causal factor. Tuberculosis, epilepsy or endocrine dyscrasias have been constantly observed in the family history, together with other neuro-psychiatric features, schizophrenia, melancholia, etc. The disease assumes the character of a Mendelian recessive. A gliosis of microglia is constantly observed. Various other histopathological points not of an original nature are mentioned. The degenerative process follows the track of immature myelinization, which explains the shading of progressing degeneration observed with Weigert's method from anterior pole to posterior pole of the brain. The authors use the word "scale" to describe this. The cerebellipetal type of atrophy is restricted to this condition. The pathogeny is in doubt. Schaffer is in disagreement with Spielmeyer and Bielchowsky. The latter two consider it as a manifestation of a general lipid disorder, while the former regards it as an undifferentiated cellular defect of protoplasmic constitution.

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*A Case of Infantile Dementia with Amaurosis.* (*L'Encéphale*, vol. xxix, p. 561, Sept.-Oct., 1934.) von Mayendorf, E. N.

This is a case where a female child of five died after showing symptoms suggestive of Tay-Sachs' disease. At autopsy, however, the brain showed no confirmation. This led to the consideration of the status of the case. One striking feature was the presence of very large cells similar to those described by Schaffer (*Arch. f. Psych.*, 84, xiii, p. 493). These cells were found in all parts of the brain. At a congress Schaffer stated after hearing an account of the case that it occupied a position midway between Tay-Sachs' disease and the juvenile amaurotic idiocy of Spielmeyer. The child had died at the age of five, whereas the age at death in Tay-Sachs is generally one and a half years.

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