may also connect the substantia nigra with other nuclei on the opposite side of the mid-brain. Foix and Nicolesco think that some fibres of the peduncle do not cross in the posterior commissure, but descend and come into relationship with grey masses lower down in the brain-stem. In regard to afferent fibres, descending fibres come from the corpus striatum, particularly the globus pallidus. There may be fibres from the cortex, but these are unimportant. In his series of eight cases of encephalitis lethargica the author found that the cortex, thalamus and corpus striatum were practically normal. The substantia innominata of Reichert, the corpus Luysii and upper part of the red nucleus were normal, or only showed a slight increase in glial content. In the mid-brain, the area occupied by the substantia nigra was poorly delimited and appeared paler. In some cases the cells had almost disappeared; those that remained showed chromatolysis. In other cases groups of cells were to be found, some of which were normal. In some cases quantities of pigment were lying free in the parenchyma. There was, except in one case, an increase in the number of glial cells. The gliosis was practically confined to the substantia nigra and the peri-aqueductal grey matter in the pons and mid-brain. Neuroglial fibres were especially plentiful in the region of vessels. Marked cuffing of the vessels only occurred in two out of the eight cases. The capillaries were present in excessive numbers. The nerve-fibres in Weigert-Pal sections showed alterations in their myelin sheaths. In some cases there was a thinning out of the fibre network; in others the fibres were interrupted in their course, the myelin sheath appearing as a series of beads. The pons showed a gliosis in the grey matter underlying the aqueduct of Sylvius; this process spread out laterally and often involved the *locus cæruleus*. In two cases the cells of the *locus cæruleus* were almost entirely destroyed on one side. In some cases there was depigmentation with chromatolysis of some of the cells of the *locus cæruleus*. The author thinks the changes in the *locus cæruleus* may be responsible for some of the symptoms attributed to the vegetative system. G. W. T. H. FLEMING.

6. Mental Deficiency.

The Nature of Hereditary Mental Defect. (*Proc. Amer. Assoc. for the Study of the Feeble-minded*, 1926.) *Davenport, Charles B.*

Concerning the exact nature of inheritance in the feeble-minded we are, says Dr. Davenport, still very ignorant. "If," he writes, "we adopt the hypothesis that prehistoric man had not gained all of the mental capacities that have appeared in some of his descendants, it is possible that he has left other descendants that remain more nearly on his intellectual plane."

As to the specific question whether mental defect is inherited in Mendelian fashion, Dr. Davenport is of the opinion that the subject deserves further study. The need, he maintains, is for more methods of measuring mentality. When these have been evolved

and applied in a large number of families, "then we may hope to learn just how far special limitations in intellectual capacity appear as family traits, and to learn of the particular laws of their distribution in the family and of their recurrence in successive generations." H. FREIZE STEPHENS.

Researches in Feeble-mindedness. (*Proc. Amer. Assoc. for the Study of the Feeble-minded*, 1926.) *Reported by Myerson, A.*

This is a summary of researches undertaken under the general direction of Dr. Myerson, of Boston, details of which are to appear in subsequent papers. The belief is held that the problem of feeble-mindedness is a problem *sui generis*, and that the subject-matter of feeble-mindedness must be broken up into groups, and each clinical group studied intensively as a problem in itself.

Two general conclusions emerge from the work already done:

(1) That feeble-mindedness, when inherited, appears to have no relationship to anything but feeble-mindedness; that, therefore, as a biological problem, feeble-mindedness is distinct from the mental diseases and epilepsy.

(2) That unsuspected birth trauma and infections of the encephalon occurring early in life account for a good deal of the feeble-mindedness in non-hereditary cases.

H. FREIZE STEPHENS.

Cerebral Accidents of Childhood. Relationship to Mental Deficiency. (*Proc. Amer. Assoc. for the Study of the Feeble-minded*, 1926.) *Smith, Groves B.*

Fifty cases were studied. They were not selected, being the first fifty histories with this diagnosis from the files of the Henry Ford Hospital at Detroit for the past three years. Of these, 88% were found to be mentally defective—morons, 26%; imbeciles, 40%; and idiots, 22%. H. FREIZE STEPHENS.

Glycuresis in Mental Defectives. (*Proc. Amer. Assoc. for the Study of the Feeble-minded*, 1926.) *Bronfenbrenner, A. N.*

The metabolic mechanism of aments has its peculiarities. Routine urine examinations, a number of basal metabolism tests, etc., made on the population of a large institution for mental defectives showed failures in metabolism. This paper, as the first of a series of articles reporting the results of this work, deals with "the metabolic phenomenon suggested by the ability of urine to reduce a copper salt"—"glycuresis," as mentioned by Benedict, being a better term than "glycosuria" for this phenomenon. It was found that, as a general condition, the quantity of the circulating glucose is a factor in assimilation itself, and, as a fact, the feeble-minded individual requires more carbohydrate food than the mentally normal person, "in order to make up for the extravagant and wasteful way in which his metabolism works."

H. FREIZE STEPHENS.