
The author describes a change in the brain which he regards as characteristic of chronic alcoholism, and which has been described in over 40 cases.

The principal change is a grey degeneration localized in the two commissures, the corpus callosum and the anterior comissure, and more precisely in the middle layers, limited by two normal white layers, superior and inferior. In the corpus callosum the degeneration extends from the genu to the splenium, and on the sides to the corona radiata. In Weigert-Pal sections the median layer of the corpus callosum is pale in vivid contrast with the dark blue of the dorsal and ventral layers.

The tissue of the degenerated areas is less compact and more vascular, there is frequently hyaline degeneration of the walls of the minute blood-vessels, which often have a tortuous course. Granule corpuscles with products of degeneration of the medullary sheaths of the nerve-fibres are abundant in recent cases. In the neuroglia there is swelling of the fibres. The nerve-fibres are degenerated and deprived of the medullary sheath. Many axis-cylinders persist, often with irregular swellings. In some cases areas of degeneration were observed in the white matter in other parts of the brain. The degeneration is most frequently found in the area fronto-parieto-occipitalis.

The author considers that part at least of the mental symptoms of chronic drinkers is due to degeneration of the great commissures. If to this is added alterations in the median peduncles of the cerebellum, through which the frontal lobe of one hemisphere is connected with the opposite cerebellar hemisphere, and the degeneration of more or less extensive areas of the white substance in both hemispheres which the author found in some cases, the severe action of alcohol on the functions of the nervous system in chronic drinkers may be better understood.

G. W. T. H. FLEMING.


In the brain there are two glycolytic mechanisms, one concerned with the breakdown of glycogen and requiring the presence of inorganic phosphate, the other with the breakdown of glucose and taking place without the participation of inorganic phosphate. The limited ability of brain-tissue to produce lactic acid from glycogen may be regarded as due to its relative inability to synthesize active phosphoric esters, since it has been shown that hexose mono- and hexose diphosphates are converted into lactic acid to a much greater extent than is glycogen. Glucose breakdown to lactic acid by brain is not inhibited by the presence of glycogen. Probably the two mechanisms are quite independent. When glucose and hexose monophosphate or hexose diphosphate are present together, the glycolysis observed is the sum of the glycolyses produced from each substrate separately, showing that the enzyme systems responsible for the two effects are independent. When glucose and mannose are present together, the results show that the same enzyme is responsible for their breakdown. When hexose diphosphate and hexose monophosphate are present together, the results suggest in the main a common enzyme system.

The author considers that it is possible that the glucose mechanism in the brain may give rise to methyl-glyoxal as an intermediate product, while the glycogen mechanism may give rise to pyruvic acid, the final result of both, of course, being lactic acid.

G. W. T. H. FLEMING.


The authors investigated the effect of a number of amines on the oxidations of brain-tissue. β-phenylethylamine, β-phenyl-β-hydroxyethylamine, tyramine,
indole, isoamylamine and mescaline inhibited the oxidation of glucose, sodium lactate and sodium pyruvate by the brain. They also inhibited the oxidation of sodium glutamate, but, with the exception of tyramine, they had little or no effect on the oxidation of sodium succinate. The effects of the amine were similar to, and of the same order of magnitude as those of typical narcotics. Neuline, cadaverine, putrescine, ethylamine and histamine had relatively little effect on the oxidation by the brain of glucose, sodium lactate or sodium succinate. Skatole also diminishes the oxidation of glucose by brain.

G. W. T. H. FLEMING.

Mental Derangement as a Result of Oxygen Lack in Circulatory Failure. (Wien. klin. Woch., vol. xlvi, p. 865, 1933.) Hitzenberger, K.

Two cases are reviewed in which arterial blood had an oxygen saturation ranging from 62.6–83.5%. The carbon dioxide contents were 58 and 73 vols. %. From these data it was deduced that the oxygen supply to the brain was inadequate.

D. B. DILL (Chem. Abstr.).

The Blood Velocity in Hyperthyroidism. (La velocidad sanguínea en el hipertiroidismo). (La Semana Méd., vol. xl, p. 38, July 6, 1933.) Del Castillo, E. B., Berconsky, I., and Cossio, P.

Estimations of the blood velocity were made in 24 cases of hyperthyroidism (6 cases of exophthalmic goitre, 13 cases of toxic adenoma and 5 cases of hyperthyroidism without goitre). These estimations were compared with those made on 19 control cases. The average velocity of the blood in hyperthyroidism is increased as compared with that found in normal subjects. No relation was observed to exist between the blood velocity and the various clinical forms of hyperthyroidism.

M. HAMBLIN SMITH.


After cessation of function of the generative glands (post-menopause, castration, atrophy), the pituitary sex hormone is regularly increased in the urine and the blood, and is demonstrable in the pituitary gland itself provided only that the latter is intact. Such an increased excretion of pituitary hormone is necessary, but not sufficient criterion of absent gonadal function. The absence of both the pituitary and sex hormones suggests pituitary damage.

HARRY EAGLE (Chem. Abstr.).

Investigations of Liver Function in Mental Disease. (Norsk-Mag. Laeg., vol. xciv, p. 170, 1933.) Lingjaerde, O.

Symptoms of liver affection were found in various forms of mental disease, except in constitutional and psychopathic cases and oligophrenia, where conditions were always normal. Symptoms of liver affection were present in 80% of cases of schizophrenia in the active phase. Long-time experiments with 25 patients, with careful control of the daily supply of protein, fat and carbohydrate, and observation of ketonuria and urobilinuria, showed that the latter is frequently due to deficiency of carbohydrate: this leads to the assumption that urobilinuria is an indication of the glycogen content of the liver. Deficiency in this glycogen content may affect the metabolism of the ganglia cells, and may also contribute to intestinal intoxication. Hence, special attention should be paid to the supply of carbohydrates and the control of ketonuria and urobilinuria in cases of schizophrenia and other mental diseases.

H. C. M. INGEBERG (Chem. Abstr.).