

The Degeneration of the Brain in Chronic Alcoholism. (*Proc. Roy. Soc. Med.*, vol. xxvi, p. 1151, July, 1933.) *Marchiafava, E.*

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 commissure, 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.

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Glycolytic Mechanisms of Brain. (*Biochem. Journ.*, vol. xxvii, p. 903, July, 1933.) *Ashford, C. A.*

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.

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The Effects of Amines on Oxidations of the Brain. (*Biochem. Journ.*, vol. xxvii, p. 1609, Nov., 1933.) *Quastel, J. H., and Wheatley, A. H. M.*

The authors investigated the effect of a number of amines on the oxidations of brain-tissue. β -phenylethylamine, β -phenyl- β -hydroxyethylamine, tyramine,