

*The Chemical Mechanism of Glycolysis in the Brain.* (Boll. Soc. Ital. Biol. Sper., vol. x, pp. 725-7, 1935.) Mazza, F. P., and Valeri, C. M.

The purpose of the investigation was to see whether the principal chemical reactions which characterize glycolysis of muscle and alcoholic fermentation (Embden, Meyerhof) also occur by the action of brain enzyme extracts. Rabbit brain extracts were inactivated by allowing them to stand at room temperature for three hours and then dialysed. When added to fructosediphosphoric ester, triosephosphoric esters were produced, thus showing the presence of zymase in the brain. By the action of brain pulp on starch solutions in the presence of lactates, and on hexosediphosphates in the presence of fluorides,  $\beta$ -phosphoglyceric acid was formed (isolated as the Ba salt). Therefore the second reaction in the Embden-Meyerhof mechanism takes place in the brain. Brain extracts prepared in a manner similar to that used by Meyerhof for muscle extracts act on phosphoglyceric acid by splitting off  $H_3PO_4$  and producing pyruvic acid; if  $\alpha$ -glycerophosphoric acid is also present there is a marked production of lactic acid. In the presence of fluorides the scission of phosphoglyceric acid is inhibited, but if the extracts act on a mixture of  $\alpha$ -glycerophosphoric acid and pyruvic acid, the latter is transformed completely into lactic acid and the reaction is not inhibited by fluorides. Particularly noticeable is the fact that  $H_3PO_4$  is not liberated. Like the glycolytic enzymes of muscle, the enzymes of brain need a co-enzyme (the same as that of muscle); brain extracts when dialysed lose their activity.  $ICH_2CO_2H$  partially inhibits the formation of lactic acid when added to a mixture of brain extracts, phosphoglyceric acid and glycerophosphoric acid or to brain extracts, pyruvic acid and phosphoglyceric acid. In the first case, it does not completely inhibit the scission of phosphoglyceric acid with liberation of  $H_3PO_4$  but does inhibit much more so the production of lactic acid.

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*Pathological Anatomy and Physiology of Epilepsy* [*Anatomopathologie et physiopathologie de l'épilepsie*]. (Ann. Méd. Psych., vol. xv (i), p. 145, Feb., 1936.) Steck, H.

Reviewing the various anatomical changes that have been found in epilepsy, the writer insists that it is first necessary to distinguish carefully between symptomatic and essential epilepsy. Essential epilepsy should show anatomical changes common to all the epilepsies and also the absence of gross irritative lesions. Inflammatory changes, trauma, syphilitic foci, congenital malformations, etc., are irritative lesions provoking the crises in symptomatic epilepsy. It is concluded, in agreement with Spielmeyer, that the principal lesions found in the brains of epileptics are neither the cause of the disease nor of the epileptic crises. They are the results of the epileptic crises, more particularly of the preceding vascular disorder.

Turning to the neuropathology, the writer is inclined to favour the theory of irritation followed by paralysis, rather than that of inhibition and liberation.

Regarding physiopathology, the view is taken that the various biochemical, endocrine and other physiological changes have no direct causal association with epilepsy, but are, for one part, concomitant factors, preparoxysmal alterations of the organism, and, for the other part, the expression of the continual oscillation of and the re-establishment of the physiological equilibrium upset by the crisis.

It is stressed that each crisis is to some degree the consequence of the preceding and the cause of the subsequent crisis. This depends upon not only reversible biochemical changes, but also upon a fundamental property of the central nervous system—the dynamic canalization or automatization of discharge phenomena. The therapeutic value of combating the formation of the epileptic habit is an implicit recognition of these phenomena.

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*On the Differentiation of Coloured Cerebro-spinal Fluids.* (Amer. Journ. Med. Sci., vol. cxciv, p. 538, April, 1936.) Robinson, F. H., and Miller, B. N.

The writers carried out compression experiments on the spinal cords of dogs. They found that the yellow spinal fluid of cord compression gives a negative