

*The Acetylcholine Metabolism of a Sympathetic Ganglion.* (*Journ. Physiol.*, vol. lxxxviii, pp. 265-83, 1936.) Brown, G. L., and Feldberg, W.

Acetylcholine liberated in a ganglion by preganglionic stimulation can, if protected by eserine, reach such a concentration that it produces a partial paralysis of the ganglion-cells. Synthesis of acetylcholine appears to occur during a stimulation of the preganglionic fibres. This synthesis is not affected by eserine. Prolonged stimulation does not significantly alter the amount either of acetylcholine or of choline extractable from a ganglion. E. D. WALTER (Chem. Abstr.).

*A Contribution to the Study of Polypeptidæmia (Contribution à l'étude de la polypeptidémie en clinique neuro-psychiatrique).* (*Ann. Méd. Psych.*, vol. xv (ii), p. 572, Nov., 1936.) Artur, Boucher et Coulonjou.

In 14 out of 27 investigations of the polypeptide level in the blood, there was a hyperpolypeptidæmia between 0.043 and 0.090 grm. Of these 14 cases 11 could be explained by an associated peptic insufficiency. In 2 cases mental improvement was paralleled by a lowering of the polypeptide level. No direct association was found between hyperpolypeptidæmia and any specific neurosis or mental disorder. The majority of the cases studied had alcoholic psychoses.

Sixteen investigations of the polypeptides in the cerebro-spinal fluid showed no abnormally high level; this group included a number of cases in the acute stages of delirium tremens. STANLEY M. COLEMAN.

*Contribution on the Origin of Increased Protein in the Cerebro-spinal Fluid of Tumours of the Central Nervous System.* (*Journ. Nerv. and Ment. Dis.*, vol. lxxxv, p. 373, April, 1937.) Deane, J. S.

The writer investigated the data provided by 81 consecutive cases of tumour within the intracranial or intraspinal cavities. He found that the presence of spontaneous hæmorrhage or necrotic products or both had little if any relation with an increased cerebro-spinal fluid protein. Generalized increased intracranial pressure *per se* appears to have no relation with increased protein, neither does the histological type of the tumour.

Three types of internal hydrocephalus are distinguished according to their modes of origin and the amount of cerebro-spinal fluid protein in each type:

(a) Internal hydrocephalus due to partial or complete obstruction of the venous effluence of the choroid plexus, in which the rate of fluid formation exceeds the rate of absorption. In this type the fluid protein is usually increased.

(b) Internal hydrocephalus due to interference with the absorption of fluid, in which its formation continues although the rate is decreased. This type is usually associated with a normal or often decreased fluid protein.

(c) In this type a combination of both *a* and *b* exists. The amount of fluid protein may be variable, but is usually increased.

The increase in fluid protein appears to be due almost exclusively to increased transudation of blood-plasma proteins into the fluid, which occurs because of a partial or complete obstruction to venous return flow.

Increased fluid protein in intracranial tumours is apparently caused by local compression of relatively non-anastomotic venous channels draining the choroid plexuses, especially those of the lateral and third ventricles.

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