probably combining with ketones and aldehydes involved in glycolysis and the tricarboxylic acid cycle (unpublished). Such observations do not lend support to the concept of a 'sodium pump', but rather corroborate the thesis that phosphorylative mechanisms are not activated during conduction[3,4]. Furthermore, it would appear that phosphocreatine is not at all essential for the maintenance of either the membrane[5] or action potential.

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Increase in the γ-Aminobutyric Acid Content of Rat Brain After Ingestion of Ethanol

γ-Aminobutyric acid is present in relatively large amounts in the mammalian central nervous system, where it exists chiefly in the free form [1,2]. The formation of this amino-acid in brain tissue takes place by the decarboxylation of L-glutamic acid [1, 3, 4]. There are in the literature several reports of a number of substances, mainly hydrazides, which lower the central nervous system level of γ-aminobutyric acid. In addition, a few substances, like diphenylhydantion and acetazoleamide, and some conditions have been reported to elevate the brain γ-aminobutyric acid content [5].

We have observed that ethanol adminstration to the rats caused an increase in the brain γ-aminobutyric acid during the period of intoxication. To four groups of rats of the Wistar strain (7 and 8 animals), previously fasted for 24 hr. was given by means of a stomach tube 430 mgm. of ethanol per 100 gm. of body-weight as a 33 per cent solution. Six rats received the same quantity of ethanol and 167 mgm. of L-glutamine per 100 gm., and to 4 rats was given only the same amount of glutamine as water solution. Three control rats received an equal quantity of water. The control animals were killed immediately and the others at various times up to 24 hr. The brains were excised quickly and homogenized in 75 per cent ethanol. The concentrated brain extracts were then studied chromatographically according to a modified method of Roberts and Frankel [1].

The γ-aminobutyric acid content of whole brain of the control rats was 44 mgm. (range 42·5–45 mgm.) per 100 gm. of fresh tissue. The results of the experiments with the 2 series of rats that received ethanol only are combined in Fig. 1. The maximum elevation of γ-aminobutyric acid, a 34 per cent increase of the initial value, occurred at 60 min. after administration of alcohol. The γ-aminobutyric acid-level of the rats that received ethanol and glutamine or only glutamine (Fig. 2) also rose; but the maximum increase at 40 min. was only 18 and 20 per cent of the initial value, respectively.

The observation that ethanol ingestion increases

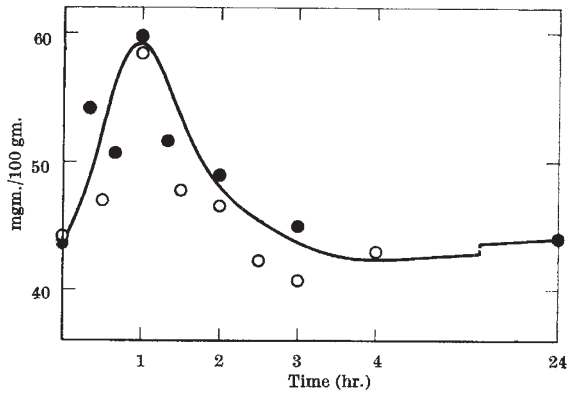

Fig. 1. Change in the γ-aminobutyric acid content of rat brain after ethanol administration. Black and white circles represent two different series of experiments.

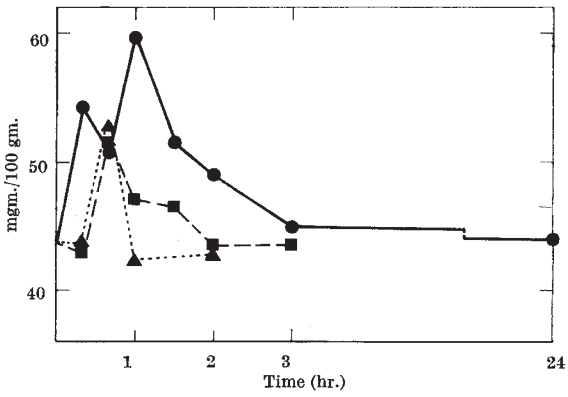

Fig. 2. Change in the γ-aminobutyric acid content of rat brain after administration of the following substances: ●, ethanol only; ■, ethanol and L-glutamine; ▲, L-glutamine.

the γ-aminobutyric acid content of brain is in agreement with the conception presented elsewhere[5] that brain γ-aminobutyric acid content varies inversely with brain excitability.

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Sex Difference in Ethanol Tolerance of Rats

In experiments on factors which influence the behaviour of rats in a simple functional test during alcohol intoxication, it was observed that after identical treatment the performance of young, sexually mature females was less impaired than was the performance of males. In testing, a record is made of the sliding angle on a tilted plane with a rough surface. The sliding angle is a linear function of alcohol dose in the approximate range of 0–8 mgm. ethanol/gm. body-weight, orally administered. A full description of the test has been given elsewhere.[1]

Rats from the laboratory supply were fasted overnight, 2 mgm. ethanol/gm. body-weight was injected intraperitoneally as a 10 per cent (w/v) solution in saline, and testing was carried out with 15-min. intervals until 90 min. after injection. When blood alcohol was determined, samples were drawn