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Influence of oxotremorine on glycogen content in various brain structures of rat brain

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THERE is much evidence to support the importance of corpus striatum in the genesis of static tremor.[1,2] It was shown that tremor-producing drug, oxotremorine (2′-oxo-1:4-dipyrrolidinobutyne) decreased the level of iron[3] and total flavines[4] in whole rat brain[5] and rat corpora striata.[6] Physostigmine and

TABLE 1. THE INFLUENCE OF ATROPINE AND PROPANOLOL ON GLYCOGENOLYTIC EFFECT OF OXO-TREMORINE

Treatment of the animals	Brain structures				
	Cortex cerebri	Caudate	Thalamus	Cortex cerebelli	Caudal brain stem
1. Control	52·2 ± 1·6	70·0 ± 1·7	42·3 ± 1·9	87·6 ± 3·7	102 ± 4·4
2. Oxotremorine (0·25 mg/kg)	48·0 ± 2·6	20·2 ± 2·2*	19·8 ± 1·1*	83·6 ± 1·9	23·5 ± 3·0
3. Atropine (0·5 mg/kg)	53·0 ± 1·4	72·4 ± 1·8	45·1 ± 1·3	81·0 ± 1·1	93·3 ± 3·7
4. Propranolol (10 mg/kg)	56·8 ± 1·4	68·5 ± 1·6	38·9 ± 1·3	88·4 ± 2·2	108·6 ± 2·0
5. Atropine (0·5 mg/kg) +Oxotremorein (0·25 mg/kg)	53·0 ± 1·8	74·0 ± 1·6	42·1 ± 1·6	93·4 ± 2·2	98·5 ± 2·4
6. Propranolol (10 mg/kg) +Oxotremorine (0·25 mg/kg)	49·8 ± 1·5	71·4 ± 1·7	45·6 ± 1·4	95·4 ± 2·5	101 ± 2·6

* $P < 0.01$ in comparison with the controls.

The content of glycogen is expressed in mg% of freshly frozen tissue. The numbers indicate the mean value (M) of five experiments ± S.E.M.

amiton, which evoke the static tremor and increase the amount of acetylcholine in central nervous system, also decreased the concentration of total flavines in rats brain as oxotremorine did.[6] These decreases could be prevented by the application of atropine. On the other hand, physostigmine produced the decrease of glycogen content in cortical and subcortical brain structures in the rat.[7] This glycogenolytic effect of physostigmine could be blocked with atropine, as well as with propranolol. Beta-adrenergic blocking drug propranolol also prevented the static tremor produced with oxotremorein.[8]

The aim of this work was to find whether oxotremorine has any influence on glycogen concentration in various brain structures of the rat.

The experiments were carried out on adult Mill-Hill rats. Oxotremorine (kindly donated by Professor P. Stern, Institute of Pharmacology, Medical Faculty, Sarajevo, Yugoslavia) was administrated intravenously in a dose of 0·25 mg/kg. Atropine sulphate (0·5 mg/kg) was administrated intraperitoneally 20 min and propranolol (10 mg/kg) 30 min before oxotremorine.

From the frozen brain tissue, glycogen was extracted by the method of Le Baron[9] and after purification from the water solution was determined by spectrophotometric method of Montgomery.[10]

The results obtained are presented in Table 1.

Our data show that atropine, as well as propranolol prevents the glycogenolytic effect of oxotremorine in rat brain. The results are similar with those obtained with physostigmine.[7] On the other hand, Hutchins and Rogers[11] did not find any effect of physostigmine and oxotremorine on glycogen concentration in whole mouse brain. Such results are not unexpected because, there are differences in metabolism of oxotremorine in rat and mouse which produce the difference in pharmacological activities.[12]

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Lipid peroxide formation in experimental inflammation*

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ANTI-INFLAMMATORY drugs have been reported to inhibit cellular oxidation and thus interfere with energetics.[1] Concurrent effects, if any, of these drugs on lipid peroxidation arising out of the resultant disturbances in the redox values and the consequent autocatalytic disruption of membrane systems

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