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Some effects of butoxamine on glycolysis in the mouse brain

N-t-Butylmethoxamine (butoxamine) prevents the rise in blood free fatty acids, glucose and lactate after the administration of adrenaline or isoprenaline (Burns & Lemberger, 1965; Salvador & April, 1965). The drug also produces a selective blockade of some, but not all, β-adrenergic receptor sites in the anaesthetized dog (Levy, 1966) and can therefore be clearly distinguished from such β-adrenoceptor blocking drugs as dichloroisoprenaline and pronethalol (Pilkington, Lowe & others, 1962) and propranolol (Salvador, April & Lemberger, 1967).

The effects of butoxamine on brain glycolysis are now reported.

Groups of 5 specific pathogen free mice (18–22 g) of either sex and of the Alderley Park strain were given the drug intraperitoneally and the animals transferred to a room maintained at $38 \pm 1°$. At different times after injection, groups were killed by total immersion into liquid nitrogen. The brains were removed while still frozen and triturated with cold 10% trichloracetic acid. "Bound" glycogen was estimated in the acid insoluble material by the method of Russell & Bloom (1958). Lactate, pyruvate, glucose and "free" glycogen were estimated in aliquots of the acid soluble fraction by enzymatic methods (Leonard, 1971).

The relation between the effect on some parameters of brain glycolysis are shown in Fig. 1. At 20 mg/kg, butoxamine produced a significant decrease in glucose and "free" glycogen and a significant rise in "bound" glycogen and pyruvate. Brain lactate did not change appreciably. Doses of butoxamine lower than 20 mg/kg did not produce any noticeable change in the concentrations of these substances. The changes produced were not correlated with any change in behaviour; the mice were not unduly affected by this dose of drug compared with those given physiological saline alone.

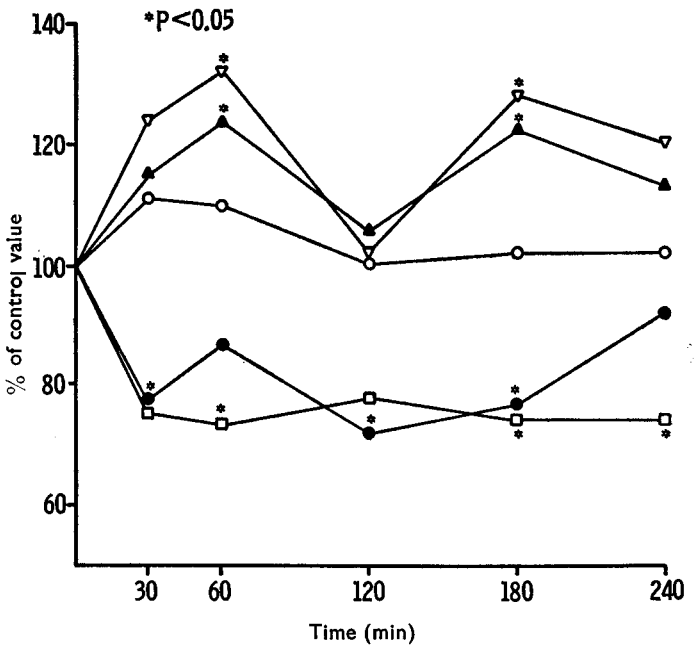

FIG. 1. Effect of butoxamine on mouse brain glycolysis. Butoxamine was administered in a dose of 20 mg/kg i.p. Each point represents the mean of at least 5 animals. All values are compared with controls (100%). The absolute values (μmol/g wet wt; mean $\pm$ s.e.) for the individual parameters were: "Bound" glycogen: 1·68 $\pm$ 0·109 (as glucose). "Free" glycogen: 0·72 $\pm$ 0·063 (as glucose). Pyruvate: 0·113 $\pm$ 0·02. Lactate: 2·17 $\pm$ 0·23. Glucose: 0·430 $\pm$ 0·002. Significance of difference from controls shown by $*P < 0{\cdot}05$.

It is apparent that butoxamine, unlike β-adrenoceptive blocking drugs such as propranolol, has a stimulant type of neurochemical profile in that it increases brain glycolysis. In this respect it more closely resembles the action of sotalol (MJ 1999) than propranolol (Leonard, 1971). It seems that the nature of the β-adrenergic receptor(s) in the brain is unlike those found peripherally.

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