

SHORT COMMUNICATION

EFFECT OF COLD STRESS ON GLYCOGEN AND CHOLINESTERASE CONTENTS OF RAT MYOCARDIUM AND ITS MODIFICATION BY PHYSOSTIGMINE*

By

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A marked depletion of myocardial glycogen occurs under different stressful situations (4, 5, 11, 13, 14). There is a rise in acetylcholine content of rat myocardium after restraint stress (13) and rabbit myocardium after cold stress (14). In the present communication an attempt has been made to evaluate the effect of cold stress on myocardial glycogen and cholinesterase contents. Further, physostigmine was employed to elucidate the role of cholinergic system, if any.

MATERIALS AND METHODS

83 albino rats of either sex, weighing between 90 and 120 g and kept under identical conditions (diet and surroundings), were studied in four groups: Group I (controls) rats were not subjected to any procedure. Group II rats were exposed to cold stress by immersion in ice cold water (3 ± 0.5 °C) thrice during 24 hr for three min at every exposure by the modified method of Raab *et al.* (10). Group III rats received physostigmine salicylate in doses of 0.1 mg/kg subcutaneously in the morning and evening for one day. Group IV rats were exposed to cold stress in the morning and evening one hr after the administration of physostigmine salicylate (0.1 mg/kg s.c.). The third exposure to cold stress was given at the end of 24 hr. For removal of hearts the animals were sacrificed after ether anaesthesia given immediately after the third exposure to cold stress in groups II and IV and 24 hr after the administration of the first dose of physostigmine in group III.

Cardiac glycogen estimation was done colorimetrically according to the method of Kemp and Kits (7) in terms of glucose equivalents. Cholinesterase in the cardiac tissue was determined colorimetrically by the method of Augustinsson (1) as described by Quastel (15). The method is based upon the chemical determination of unreacted acetylcholine. The ester reacts with hydroxylamine to form acetydroxamic acid, which further reacts with ferric ions in acid solution to form soluble red purple complex.

RESULTS AND DISCUSSION

The cholinesterase activity and the glycogen content were statistically significantly reduced after cold stress. The former was insignificantly increased while the latter was more

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markedly reduced in animals treated with physostigmine. The present study revealed that physostigmine did not prevent the depletion of glycogen content and reduction of cholinesterase activity caused by the cold exposure of the animals (Table I). Adequate availability of oxygen to the myocardial cells is essential for the maintenance of the cellular glycogen level within physiological limits (4, 5, 11, 13, 14). In anaerobic condition, glycogen is preferentially utilized with a resultant fall in its level. Raab *et al.* (10) have suggested that the stress induced myocardial changes are due to sympathetic predominance which occurs under various stress situations (9, 12). Sympathetic predominance substantially increases the oxygen need of the myocardium and causes oxygen deficit. This mechanism appears to be responsible for the depletion of myocardial glycogen on cold exposure too. The glycogen depletion induced by physostigmine appears also to be due to sympathetic predominance because physostigmine has been reported to cause increased discharge of adrenaline from the adrenal medulla (8) and release of locally acting catecholamines from the heart (6).

TABLE I
Effect of various procedures on glycogen and cholinesterase contents of rat myocardium

Procedure	Mean glycogen mg/100 g \pm S.E.	P	Cholinesterase activity, Mean μ mol of Ach hydrolysed/g tissue/1 hr \pm S.E.	P
Control	520.6 \pm 3.9 (16)	—	119.0 \pm 7.2 (10)	—
Cold stress	227.0 \pm 10.7 (15)	< 0.001	88.7 \pm 3.1 (8)	< 0.001
Physostigmine treatment	182.0 \pm 4.9 (10)	< 0.001	124.0 \pm 4.4 (8)	> 0.05
Physostigmine treatment and cold stress.	214.5 \pm 4.3 (8)	< 0.01	85.0 \pm 8.96 (8)	< 0.001

Figures in parantheses indicate the number of animals.

The decrease in myocardial cholinesterase activity in the animals subjected to cold stress can be attributed to endogenous secretion of adrenaline during stress as adrenaline has been shown to inhibit the cholinesterase activity (2, 3). It is surprising that cholinesterase activity was not inhibited in rats treated with physostigmine. It is not possible to give a suitable explanation for this unusual finding.

SUMMARY

1. The cholinesterase activity and glycogen content of rat myocardium were reduced after cold stress.
2. Physostigmine further reduced the glycogen content but did not affect the cholinesterase activity.

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