

EFFECT OF PROLONGED STRESS OF REPEATED ELECTRIC SHOCK ON RAT MYOCARDIUM

P. LAURIA, V. N. SHARMA AND S. VANJANI

*Department of pharmacology and Experimental Therapeutics,
S. M. S. Medical College, Jaipur.*

Summary : Electric shocks were given to albino rats for thirty days and myocardial glycogen, cholesterol, acetylcholine, cholinesterase, 5-HT and hisatamine contents were compared with those of the control group. There was a singificant decrease in myocardial glycogen, 5-HT and his-tamine contents while the myocardial cholesterol, acetylcholine and cholinesterase contents were significantly increased. Histological abnormalities were found in one third of the animals sub-jected to stress.

Key words : chronic stress Rat myocardium

INTRODUCTION

In previous studies reported from our laboratory, it has been shown that sudden exposure of animals to stresses like cold, heat and restraint, produced effects on myocardial contents of glycogen, acetylcholine, and 5-HT (16,17, 18). However, there is paucity of information on the effects of prolonged stress on these parameters. The present communication concerns itself with the investigation of the effects of chronic stress made along these lines.

MATERIALS AND METHODS

Albino rats (Haffkine strain) of either sex weighing 90-120 g were used. One group was subjected to electric shock induced by the method described by Hall *et al.* (8) and the other group served as a control. The shocks (10-15 volts) were given for 2 min every half an hour for 6 hr daily for 30 days.

Cardiac glycogen was estimated colorimetrically according to the method of Kemp and Heijnigen (10) in terms of glucose equivalents. Myocardial cholesterol was estimated by the method of Cavanaugh and Glick adaptation (5) of Schoenheimer and Sperry (15) for cholest-erol and cholesterol esters.

Acetylcholine was extracted in 10 ml of Ringer solution treated with physostigmine (12 mg of physostigmine in 800 ml of Ringer solution) from frozen heart at pH 4, at 90°C - 100°C. The assay was done on frog rectus abdominis muscle as described by Anand (1). Cholinesterase activity in the myocardium was estimated colorimetrically in all essential

details by the method of Angustinsson (2) as described by Quastel (13). Myocardial tissue extracts were prepared according to the method of Bartlet (3) with minor modifications. 5-HT was estimated employing the rat fundus preparation of Vane (19). Histamine was estimated in the myocardium by the method of Parratt and West (12).

For the histological studies the heart was fixed in 10% formolsaline for 24 hr. Paraffin sections were prepared and stained by haematoxylin and eosin and studied microscopically.

RESULTS AND DISCUSSION

The results of histological examination following chronic stress are given in Table I. It can be seen that 10 of the 31 hearts exhibited abnormalities. The remaining 21 hearts appeared normal on histological examination.

TABLE I : Histological effects in the myocardium of animals given stress of electric shocks daily for 30 days.

Total No. of animals	Normal	Abnormalities				
		Fragmentation of fibres	Oedema of myocardium	Focal necrosis	Round cell infiltration	Myxomatous degeneration
31	21	5	3	1	1	1

TABLE II : Analysis of the results in rats subjected to 30 days electric shock as compared with the control group.

Parameter	Control group		30 days electric shock		
	Range	Mean \pm S.E.	Range	Mean \pm S.E.	P values
Myocardial glycogen (mg/100 g.)	446.0-600.0 (16)	520.6 \pm 3.94	318.0-442.0 (14)	392.51 \pm 1.23	<0.001
Myocardial cholesterol (mg/100 g.)	76.0-112.0 (15)	96.0 \pm 2.69	96.0-114.0 (14)	103.9 \pm 0.60	<0.02
Myocardial acetylcholine (μ g/g.)	2.0-3.8 (15)	3.24 \pm 0.045	3.00-4.60 (9)	3.66 \pm 0.16	<0.05
Myocardial cholinesterase activity equivalent to micromoles of ACh hydrolysed by 1 g of tissue/hr.	83.0-166.0 (10)	119.0 \pm 7.21	108.0-172.0 (8)	147.6 \pm 7.81	<0.05
Myocardial 5-hydroxytryptamine (μ g/g.)	250-400 (15)	340 \pm 28	8.89-23.34 (9)	14.56 \pm 2.17	<0.001
Myocardial histamine content (μ g/g.)	1.60-2.8 (12)	2.23 \pm 0.18	0.77-1.50 (6)	1.03 \pm 0.12	<0.01

Figures in parantheses indicate the number of animals.

The remainder of the observations are presented in Table II. The myocardial glycogen was reduced significantly after 30 days of electric shock. Similar results are reported by Blount and Meyer (4) in acute stress. The fall in glycogen has been attributed to myocardial hypoxia (7). The involvement of sympathetic nervous system in glycogenolysis has been observed by Hess *et al.* (9) and is most probably due to stimulation of *beta*-adrenergic receptors (11). It is likely that in our experiments catecholamines may have been liberated to cause glycogenolysis.

An increase in myocardial cholesterol level was observed after 30 days of electric shock. Serum cholesterol is elevated in physiological stress, particularly in emotional stress (20). Atherosclerosis is closely related to lipid metabolic disturbances. Recurrent emotional stress with rise in cholesterol each time, may give rise to atherosclerosis. The potentially injurious catecholamines have necrotising properties and may predispose the arterial muscular wall to the acceptance and deposition of lipids during stress (14).

An increase in the myocardial acetylcholine as observed in the present study may suggest an increased activity of parasympathetic division as a compensatory phenomenon (16). As acetylcholine content of an organ increases, it appears logical to assume that cholinacetylase and cholinesterase should also increase. The activity of the latter enzyme which was estimated did indeed increase.

It has been reported that 5-HT and histamine are released in stress (6). Since 5-HT and histamine are released during acute stress, myocardial lowering of these amines in chronic stress as observed in our experiments is understandable.

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