BIOCHEMICAL CHANGES IN ACUTE NOISE STRESS IN RATS

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(Received on December 7, 1987)

Summary : The effects of acute auditory stress on certain biochemical parameters like blood corticosterone, total cholesterol, triglyceride, Serum glutamic pyruvic transaminase (SGPT) and Serum glutamic oxaloacetic transaminase (SGOT) were studied in albino rats.

A significant increase was observed in the blood level of corticosterone. total cholesterol, SGOT and SGPT while a marked reduction was noticed in the Sr. triglyceride level. These data indicate that noise could be a potent stressor and cause disturbances in the biochemical parameters of the body. It is presumed that most of the effects are indirect, being manifested through the activation of autonomic nervous system which liberates catecholamines and hypothalamo pituitary adrenal axis responsible for the liberation of corticosteroids.

Key words :

noise stress SGOT

SGPT

Γ serum triglycerides

corticosterone

INTRODUCTION

Noise is defined as unpleasant, unwanted or intolerable sound and has come to be considered as a special form of environmental pollution. Noise from cars, trucks, jet aircrafts, television, disco music, huge crowd, vacuum cleaners, and other industrial machineries is proved to be harmful. Although the effect of noise is known to influence many physiological parameters like food and water intake, serum levels of pituitary hormones (1, 2), pituitary adrenal rhythm, the degree of changes in other biochemical parameters like triglycerides, SGOT and SGPT have not been clearly elucidated. Further Ramsey (17) has suggested that noise not only produces hearing impairment but may also be responsible for widespread disturbances in various physiological and biochemical activities of the body. Since the perusal of literature has not shown the specific effects of noise stress on the above mentioned parameters, we have planned to study the effect of noise stress on these parameters in rats.

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MATERIAL AND METHODS

Wistar strain albino rats of either Sex with body weight of 150-170G were used. All the animals were provided with standard laboratory diet and water *ad libitum*. The animals were divided into two groups.

Control group: These animals were brought to the laboratory and allowed in the chamber to get accustomed to the existing conditions.

Experimental group: The experimental rats are exposed to sound stress of >97 dB (1000Hz) for thirty minutes continuously in an audiogenic stress chamber specially fabricated for this particular type of study. Immediately after the exposure to sound stress, the animals (both the control and experimental) were anaesthetised with ether and sufficient quantity of blood was withdrawn from the jugular vein for biochemical studies.

Biochemical estimations : Plasma Corticosterone was estimated using the procedure described by Mattingly (12). Serum Cholesterol was estimated by Zlatkis, Zak and Boyle's procedure (26). Serum Triglyceride was estimated using the procedure of Schettler and Nussel (22). Aspartate Aminotransferase (SGOT-EC 2.6.1.1) and Alanin Aminotransferase (SGPT-EC 2.6.1.2) were estimated using the procedure of Reitman and Frankel (18).

RESULTS

The acute effects of noise on the biochemical parameters studied are depicted in Table I.

Group	Plasma corticosterone (µg/100 ml)	Serum cholesterol (mg/100 ml)	Serum triglyceride (mg/100 ml)	Serum SGPT (IU/ml)	Serum SGOT (IU/ml)
Control	97.77±2.86	65.28±4.66	81.07±4.36	38.4±2.96	81.7±3.04
Experimental	178.77±3.33*	136.08±6.41**	36.7±2.10**	97.8±3.94**	130.3±3.04**

TABLE : I Effect of acute noise stress on blood levels of corticosterone, cholesterol, triglyceride, SGPT and SGOT in albino rats.

Each value is Mean±S.E.M. of 10 amimals, *P<0.001 control Vs experimental, **P<0.01 The plasma corticosterone level was increased in the stressed animal compared to that of the control group. The mean corticosterone level in the experimental group was $178.77\pm$ $3.33 \ \mu g/100 \ m l$ which is statistically significant compared to the control value of $97.77\pm2.86 \ \mu g/100 \ m l$ (P<0.001). The total serum cholesterol increased after 30 min of noise stress from the control value of 65.28 ± 4.66 to $136.08\pm6.41 \ m g$ % which was also statistically significant (P<.01). Serum triglyceride showed a significant decrease from the control value of $81.07\pm$ 4.36 to $36.7\pm2.10 \ m g$ % (P<.01). Both SGPT and SGOT in the serum showed a significant elevation from the control values. SGPT rose from the control value of 38.4 ± 2.96 to $97.8\pm$ $3.94 \ IU/dl$ (P<.01). The SGOT level increased from the control value of 81.7 ± 3.04 to $130.3 \pm 3.04 \ IU/dl$ (P<.01).

DISCUSSION

In the present study certain biochemical parameters were studied after exposing the rats to thirty minutes of acoustic (noise) stress. The control values in our experiments are in agreement with the results reported in the literature (13, 19). Though the various types of stresses have been studied extensively in the past, studies on the systemic effects of noise is very limited. Further, most of the effects are indirect, manifested through the activation of autonomic nervous system (ANS) and the hypothalamo hypophyseal adrenal axis.

Serum Cholesterol : As expected there is a significant rise in total serum cholesterol level after exposing the rats to 30 minutes of noise stress. Similar findings were reported by Ramsey (17) in rabbits after exposure to noise at 102 dB for ten weeks; after high altitude stress in human by Chackraborti *et al.* (8); after surgical stress by Sane *et al.* (21); after examination stress by Bijlani *et al.* (5) after psychological stress in rats by Berger *et al.* (4). Though convincing evidences are still lacking it is probable, that the rise in cholesterol level after acute noise stress might be due to the activation of hypothalamo sympathetic adrenal axis and the resultant release of catecholamines from the adrenal medulla (17) may play a part in the mobilization of lipids from the fat stores which in turn might enhance the synthesis of cholesterol.

A significant rise in Plasma corticosterone after noise stress in this series of experiments was similar to the observation reported by Kobegenova *et al.* (16) in goats exposed to noise stress at 80 dB. Similar reports were also available in the literature in other forms of stress like anesthetic stress (6, 11), cold stress (16), heat stress (3, 9) and immobilization stress (23). The elevated plasma corticosterone level in this acute study could be due to the hyperactivity of the adrenal cortex mediated through neuroendocrine mechanisms. Volume 32 Number 2

Decrease in serum triglyceride level similar to that seen in our study was also reported by Robertson and Smith (20) after electric shocks in rats and Carruthers *et al.* (7) in people seeing violent movies. Contradictory to tnese reports an acute elevation of triglyceride was observed by Taggart and Carruthers (24) after motor racing and after public speaking (25). The contradictory results seen in triglyceride levels might be due to the type, intensity, and the duration of the stress and species variation. Further, stress is known to increase the metabolic activities of the tissues (17). If it is true, then decreased level of triglyceride might be due to the increased utilization after noise stress. Though it is difficult to explain the cause for the decrease in triglyceride level in this series of experiments with the available experimental data it is worthwhile studying this phenomenon further as there is a possibility of hepatic secretion of the triglyceride being influenced by the sympathetic innervation of the liver.

Significant changes in serum transaminases observed in this series of experiments is probably due to a nonspecific response to stress althought the exact mechanism is not clearly known. Similar increase in SGOT was observed by Moss and Murray (14) in pigs after pre slaughter stress. Nyandicka (15) demonstrated an increase in both SGOT and SGPT in rats after nutritional stress. The effects observed could be due to a direct action of stress on the tissues of the animal resulting in a generalized tissue damage which can result in a "leakage" of these intracellular enzymes in to the blood stream.

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