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SHORT COMMUNICATION

RETICULOENDOTHELIAL FUNCTION IN ACUTE NOISE STRESS

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Abstract: Wistar strain albino rats of either sex were subjected to acute noise stress (3000 Hz at >97 dB) for 30 minutes. Carbon clearance test was conducted in noise stressed animals immediately after the stress period. Significant (P<0.001) increase of the clearance constant K was observed in stressed animals compared to the controls, indicating increased phagocytic activity of the reticuloendothelial system

Key words: noise stress carbon clearance RES function

INTRODUCTION

Stress has been defined as a non-specific response of the body to any demand with emphasis on non-specific (1). Interest in noise stress is comparatively new and its effects of are classified into two main categories – Auditory effects and Extra auditory effects.

It has been reported that exposure of mice to high intensity sound (500 Hz at 135 dB) for three hours/day for three days caused a leucopenia during the stress period followed by a transient leucocytosis after the termination of stress (2). Depressed granuloma formation following subcutaneous implantation of cotton pellets in noise exposed animals has also been reported (3,4). Animals exposed to various stressors like noise, surgery and other trauma showed a decreased phagocytic activity initially followed by a gradual rise (5). Investigations on serum clearance and tissue uptake of immune complexes and saturation of reticuloendothelial system (RES) by these complexes in normal animals and animals with murine lupus have shown that RES capacity to handle immune complexes seemed to be unimpaired (6). Okimura et al (1986) reported depressed T helper and suppressor cell activities and enhanced activities of B cell in mice exposed to restraint stress (7).

Since the perusal of literature has failed to show the specific effect of noise stress on RES function, this study was undertaken to elucidate the effect of acute noise stress of known intensity and duration on the phagocytic function of RES.

METHODS

Healthy Wistar strain albino rats of either sex (120 –130 gm) maintained under standard laboratory conditions were divided into control and test groups (n = ten animals each group). All experimental rats were placed individually in a specially fabricated stress chamber and exposed to noise stress (3000 Hz at > 97 dB) for 30 minutes. The frequency, intensity and duration of noise used in this study was selected based on our previous work and acts as a definite stressor in rats. The control animals were also placed in the same chamber without exposure to noise for the same period of time. Both groups of rats were anesthetized with ether at the end of 30 minutes period and carbon clearance was measured.

Carbon Clearance

A single dose of carbon, 8 mg/100 gm of body weight was injected through jugular vein. After 45 minutes, aliquots of blood (0.02 ml) were collected at

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regular intervals of 5 minutes, for approximately 25 minutes from the other jugular vein with the aid of micropippets pre washed with heparin. The aliquots were then added to 4 ml of 0.1% Sodium carbonate solution. The carbon concentration of the hemolyzed blood sample was then measured spectrophotometrically at 600 nm. The clearance constant K was determined by using the following expression :

$$k = \frac{\log C1 - \log C2}{t2 - t1}$$

(where C1 & C2 are the concentrations of the test colloid in blood at times t1 and t2 expressed in minutes (8).

The results were expressed as mean ± SEM. The data was analyzed by student t test and $P<0.05$ and above was considered statistically significant.

RESULTS

Carbon clearance was found to be increased in stressed animals compared to that of control group. The clearance constant K in control animals was 0.0062 ± 0.004 which has significantly increased ($P<0.001$) to 0.014 ± 0.005 in noise stressed animals (Table I).

TABLE I : Carbon clearance (K) after exposure to Acute Noise Stress.

	Control Group (n=10)	*Experimental Group (n=10)*
Mean	0.062	0.141
S.E.	0.004	0.005
t :		11.8205
Significant		P < 0.001

DISCUSSION

In this present study, effect of acute noise (>97 dB for 30 minutes) on the phagocytic functions of RES was investigated. The RES is a multiorgan system whose primary function is phagocytosis. Our data suggests a significant increase of carbon clearance by RES after noise stress indicating the stimulation of RES phagocytic activity by noise. This phagocytic activity which is measured in the slow phase or the second phase of the clearance used in this study could be a measure of the phagocytic activity of the macrophages of RES (8). Though the mechanism through which stress activates the phagocytic activity of RES is not yet clearly known, recent studies have implicated certain factors which by themselves or in combination may be responsible for this increase.

Acute noise activates Pituitary adrenal axis resulting in increased secretion of corticosteroids (9). Our previous work employing the same type of noise stress as used in this study for 30 minutes revealed an increased level of blood corticosterone in albino rats (10). However, it has been shown that cortisol is a potent depressant of RES (11). Hence it becomes clear that the phagocytic activity of the RES in noise stressed animals increases in spite of an increase in steroid level. Further, the dose response curve for the phagocytic coefficient K of RES, measured 24 hours after injection of cholesterol shows a mild stimulation with a moderate dose. Similar results were also reported from our laboratory in rats after acute auditory stress (10). In addition, as early as 1948, it has been suggested that sympathin (Adrenalin & Nor-adrenalin) liberation is responsible for the activation of phagocytic system (12).

Ramsey (13) reported that exposure of rats to noise at 120 dB for duration as short as 5 seconds has resulted in a tenfold increase in urinary catecholamine metabolites (13). It has also been shown by Selye (1) and subsequently by many others that in alarm reaction the circulating catecholamines increases (14). During the alarm reaction the rate of disappearance of the injected carbon particles also increases. From these evidences, it is safe to conclude that during noise stress there is an activation of RES as indicated by an increase in the clearance constant of carbon. However, it must also be borne in mind that changes in phagocytosis could occur due a change in the composition of the serum as shown by Okimura et al (7) who have demonstrated *in vivo* suppression of phagocytic activity following restraint stress, recovering after infusion of serum from normal animals (7).

Thus the actual mechanism for altered RES function may involve a delicate balance between the circulating catecholamines and corticosteroids, in addition to other less known serum factors.

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