EFFECT OF FLICKERING LIGHT STRESS ON CERTAIN BIOCHEMICAL PARAMETERS IN RATS

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Summary : The acute effects of flickering light of 80 Lux intensity for thirty minutes duration, on plasma corticosterone, total serum cholesterol, serum triglycerides, serum glutamic pyruvic transaminase (SGPT) and serum glutamic oxaloacetic transaminase (SGOT) levels were studied in albino rats.

Statistically significant increase was observed in the corticosterone, cholesterol, SGOT and SGPT, while a marked reduction was seen in the serum triglyceride level, indicating that the flickering light is a potent stressor to these animals causing alterations in the biochemical parameters studied.

Key words : flickering light stress

INTRODUCTION

In literature it has been shown that in experimental animals prolonged exposure to ordinary indoor lightning has been linked with reproductive abnormalities and enhanced susceptibility to cancer and so on. Studies in human beings also indicate increased fatigue, decreased performance, diminished immunological defenses, reduced physical fitness and possibly impaired fertility associated with living and working under incandescent or cool white fluorescent light.

Experiments with animals have shown that chronic exposure of the eyes to ultraviolet light of 290-320 nanometers results in progressive opacity of the crystalline lens leading to cataract in about 35% of the population. It has also been shown that visible part of the spectrum influences the body rhythms and hormonal levels (12). Like other forms of stress visible flickering light stress is also expected to cause internal biochemical and physiological effects. Since our survey of literature has shown that there is lacuna in the field of visual

stress especially with flickering light this study was undertaken to elucidate the effects of flickering light of known intensity on certain biochemical parameters in albino rats.

MATERIAL AND METHODS

Adult Wistar strain albino rats of both sexes fed *ad libitum* with water and commercial rat feed, weighing 120-150 g were used in this study. The animals were divided into control and experimental groups consisting of ten animals in each group. The control group animals were placed in the Light Stress Chamber which was fabricated in this department for thirty minutes without flickering light. Blood samples were taken immediately after this by cardiac puncture under mild ether anesthesia. The experimental animals were individually exposed to flickering light of 80 Lux which was switched on and off 60 times per minute for a period of thirty minutes. After thirty minutes blood samples were obtained from these animals for biochemical analysis.

The following biochemical investigations were carried out in these blood samples. The blood samples were divided into two equal aliquots, one with heparin to obtain plasma for corticosterone estimation (8) and the other was allowed to stand at room temperature to obtain serum for cholesterol, (19). Serum triglycerides was estimated by the procedure of Schettler and Nussel (16). Serum Aspartate Aminotransferase (SGOT-EC 2.6.1.1) and (Alanine Aminotransferase (SGPT-EC 2.6.1.2) were estimated using the procedure of Reitman and Frankel (13).

RESULTS

Corticosterone : The corticosterone level in the control animals was 111.71 ± 13.11 *micro gram/dl* and in the experimental group the level increases to 221 ± 51.28 *micro gram/dl.* This increase was statistically significant ($P < 0.001$).

Serum cholesterol : The serum cholesterol showed a significant rise in the experimental animals and the level rose from 52.01 ± 15.41 *mg/dl* to 86.33 ± 26.66 *mg/dl* ($P < 0.001$).

Serum triglycerides : Stressed animals in this study showed a decrease in the triglyceride level which from the control value of 55.45 ± 9.96 *mg/dl* decreased to 37.49 ± 4.56 *mg/dl* ($P < 0\,001$).

Transaminases : The level of both the transaminases (SGPT and SGOT) were elevated

from the control value of 43.5±12.65 *IU/ml* to 68.2±17.26 *IU/ml* and 67.7±7.56 *IU/ml* to 119.9±32.85 *IU/ml* respectively ($P<0.001$) (Table I).

TABLE I : Effect of acute visual stress (30 mins) on plasma corticosterone, serum cholesterol, serum triglyceride, SGOT and SGPT levels in rat.

Group	*Pl. corticosterone (μg/100 ml)*	*Serum cholesterol (mg/100 ml)*	*Serum triglyceride (mg/100 ml)*	*SGOT (IU/ml)*	*SGPT (IU/ml)*
Control	111.71±4.15	52.01±4.87	55.45±3.15	67.70±2.39	43.50±4.00
Experimental	221.10±16.23***	86.33±8.43***	37.49±1.44***	111.90±10.39***	68.20±5.46***

Each value is mean±S. E. M. of 10 animals.
*** ($P<0.001$) control Vs Experimental.

DISCUSSION

Increased Corticosterone level after flickering light stress is similar to the report of Guazdawska *et al.* (7) after ether stress in lactating rats. Chemical stress also induces similar increase in corticosterone (6). Though no specific occipito hypothalamic pathways have been described in the literature this corticosterone response should be mediated through hypothalamo pituitary adrenal axis via ACTH mechanism. It can be presumed that visual impulses from occipital cortex can reach hypothalamus through non-specific corticohypothalamic routes or via cortico reticulo hypothalamic pathways. Similar pathways are known to play an important role in the mediation of other forms of nociceptive stimuli like pain and temperature.

The rise in serum cholesterol after flickering light stress is similar to that reported by Berger *et al.* (1) after physiological stress in rats. Rise in serum cholesterol was also reported by Bijlani *et al.* (3) in examination stress in medical students, Sane and Kukreti (15) in patients during preoperative period, and in subjects with high anxiety score (2).

The mechanism by which stress raises serum cholesterol is uncertain. It is likely to be related to the enhanced activity of hypothalamo - hypophyseal axis resulting in increased liberation of catecholamines and corticosteroids. This might lead to a rise in blood cholesterol as epinephrine is known to mobilize lipids from adipose tissue. On the other hand cholesterol may also increase as an adaptive mechanism to maintain the adrenal cholesterol as adrenal

cholesterol gets depleted during stress as a result of enhanced secretion of adrenal steroids, though the adrenal cortex itself can synthesize cholesterol. Further cholesterol output from the liver may also be modulated either directly or indirectly due to stress. The exact mechanism underlying the cholesterol response in stress needs further exploration.

The effect of stress on serum triglyceride has been subjected to some controversy. The stress associated with motor racing (12) and public speaking (18) resulted in an acute elevation of serum triglycerides. Lowering of triglycerides in man was also reported after watching violent movies (5). The results of our present study with flickering light stress is in close conformity with the work of Robertson and Smith (14) who showed a decreased serum triglyceride level in sand rats after electric shock stress. These conflicting results in triglyceride values presumably due to the failure to distinguish acute from chronic stress and due to the varied types of stressful situations tested. Since a close relationship was established between urinary catecholamines and serum triglycerides (4), it could be suggested that both these events might be mediated via adrenal medullary secretions and perhaps through the activation of sympathetic nervous system.

Meenakshi *et al.* (9) reported an acute elevation of transaminases levels in different types of stresses like formalin injection, multiple fractures, spinal cord transection, exposure to cold, and starvation. They suggested that this response is probably of a non specific nature. The significant rise of SGOT seen in this series of experiments is in agreement with that reported by Moss and McMurray (10) who have shown a similar rise in SGOT level in preslaughter handling of pigs. Nyandeika (11) also reported similar changes in SGOT level in rats after chemical stress. Though the exact mechanism responsible for the elevation of of SGOT is not known, and needs further in-depth study, it is probably due to the alterations in the membrane permeability which might occur in the cells during stress. These changes probably represent a functional alteration of the cell membrane due the steroidal storm which occurs during stress. Moss and McMurray (10) have shown similar increase in Creatinine kinase, pyruvate kinase and lactate dehydrogenase which lend support to this concept of changes in cell membrane permeability. A study of membrane transport kinetics in stressed and control animals will throw more light on the mechanism of transaminase elevation in stressed animals.

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