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EFFECT OF NOISE STRESS ON SOME CARDIOVASCULAR PARAMETERS AND AUDIOVISUAL REACTION TIME

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Abstract : A study of the effect of noise stress on some of the physiological parameters was carried out on healthy male workers of thermal power station (exposed to sound level 90-113 dBA) and compared with age and sex matched healthy controls (exposed to sound level 48-66 dBA). The parameters recorded were heart rate (HR), systolic blood pressure (SBP), diastolic blood pressure (DBP), galvanic skin resistance (GSR), auditory and visual reaction time (ART and VRT) and audiogram.

Significant impairment in audiogram at 3000 Hz and 4000 Hz, increase in HR, SBP, DBP and decrease in GSR, ART and VRT were recorded in workers who were exposed to noise stress. Also a higher prevalence of hypertension was observed in them and that they were at a higher risk of developing hypertension than the control group. It was also observed that these modifications are related to duration of exposure to noise stress. It is presumed that all the above extra auditory effects are due to activation of autonomic nervous system and hypothalamo-hypophyseal adrenal axis, and the resultant release of catecholamines from adrenal medulla due to noise stress.

Key words : noise stress, heart rate, audiogram, audiovisual reaction time, blood pressure, galvanic skin resistance

INTRODUCTION

The 20th Century has been described as the century of noise. The term 'Noise Pollution' signifies the vast cacophony of sounds that are being produced in modern life, leading to health hazards (1).

Noise not only produces hearing impairment, but may also be responsible for many extra auditory adverse effects viz annoyance, interference with speech, efficiency, cardiovascular effects, disturbances in food and water intake, changes in the serum level of pituitary hormones and pituitary adrenal rhythm (1-8).

Contrasting reports have been published on the effects of noise on blood pressure (3, 9, 10) and there is little information about the relationship between duration of exposure to noise and cardiovascular changes. This prompted us to study the influence of varying duration of noise exposure on HR, BP, prevalence of hypertension and galvanic skin resistance (GSR). A study was also attempted on the auditory and visual reaction time which is an indicator of the processing capability of the CNS and sensory motor performance (11), since there is a dearth of information in the literature on this aspect. Audiometry was also done to find out if hearing

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impairment was present and to demonstrate that the basic problem is exposure to noise stress.

METHODS

The study was conducted on 156 apparently healthy male subjects between the age groups 22-58 years. They were divided into control and experimental groups.

The experimental group consisted of subjects who were working in a thermal power station in Delhi for 8 hrs each day and 6 days a week, and were exposed to noise emitted by turbines and boilers (Sound level 90-113 dBA, mean 95±5 dBA). The sound level was measured in decibels (dBA), by a Bruel and Kjoer Impulse Precision Sound level meter fitted with octave filter set type 2209 (Denmark). The sound level of 90-113 dBA is certainly regarded as sound stress, since daily exposure upto 85 dBA is the limit that people can tolerate without causing any substantial damage to hearing (1). The experimental group was further subdivided into 3 groups depending on the duration of exposure to noise stress: Experimental Group I (n=14) with <10 yrs exposure; Experimental Group II (n=40) with 10-20 yrs exposure; Experimental Group III (n=40) with > 20 yrs exposure.

The control group consisted of either office workers or laboratory staff who were exposed to sound level of 48-66 dBA mean 55±4 dBA considered as safe sound pressure level. The control group was also subdivided, so as to match in their age and anthropometric data with experimental group, as follows: Control group I (n=22) who acted as controls for experimental group I: Control group II (n=40) who acted as controls for both experimental group II and III.

A questionnaire was made to record their age, height, weight (without shoes) and any history of present or past illness including hypertension, heart diseases, diabetes, symptoms of deafness, cardiovascular risk factors like smoking and family history of cardiovascular diseases.

Each subject was made familiar with the apparatus and the procedure to alleviate any fear or apprehension. Their audiogram, heart rate (HR), systolic blood pressure (SBP), diastolic blood pressure (DBP), galvanic skin resistance (GSR), auditory reaction time (ART), visual reaction time (VRT) were measured in a quiet room of the Department of Physiology, at LHMC, New Delhi after 30 min of relaxation.

The audiogram was recorded by screening audiometer type 4 IEC 645 (Amplaid 121) on 25 controls and 25 subjects who were exposed to noise stress. Audiometric testing was conducted prior to work in both groups to avoid temporary threshold shifts during work. Standardized audiometric testing procedures were used. The following frequencies were tested: 125, 250, 500, 750, 1000, 1500, 2000, 3000, 4000, 6000 and 8000 Hz.

Heart rate was calculated from ECG tracings, which were recorded by using the ECG machine. Blood pressure was recorded with a Sphygmomanometer and GSR by using a Biofeed-back apparatus supplied by Medicaid System (Chandigarh). GSR was measured by fixing the electrodes on the index finger after cleaning the finger with spirit.

ART and VRT were measured by reaction time instrument supplied by Medicaid Systems (Chandigarh). This instrument is equipped with a quartz clock, which can measure upto 1/10th of a msec. All the subjects were right handers and used their right hand to press the switch to stop the quartz clock of the apparatus. Before measuring VRT, each subject was asked to identify the flashing of yellow light. He was instructed to press the switch as soon as he saw the light. For measuring ART he was asked to concentrate on the sound signal produced and press the switch immediately. The sound signal was a continuous beep of 1 KHz on speaker. The intensity of stimulus was same for both groups and reaction time was recorded only on persons who could hear the sound clearly. From the auto-display reaction time was noted. Three readings were recorded and the lowest was taken as the reaction time.

The percentage of hypertensive subjects at rest was determined according to World Health Organization definition of hypertension i.e systolic blood pressure ≥160 and/or diastolic blood pressure ≥ 95 mmHg (12).

Results were analysed statistically (mean, SD, Student's t-test, Chi-square test and odds ratio for estimation of the risk of hypertension associated with exposure to noise).

RESULTS

There was no significant difference in age, height and weight between control and experimental groups. Their alcohol intake, family history of hypertension and smoking patterns were comparable.

The mean hearing levels across various frequencies are shown in Table I for both groups. There is a significant hearing loss at 3000 Hz (P<.05) and 4000 Hz (P<.01) in the experimental group as compared to that in the control group.

Table II shows the data in control group I and experimental group I (<10 yrs exposure to noise stress). A significant increase in the heart rate (P<.05), SBP (P<.05) and DBP (P<.05) and a decrease in GSR (P<.05) were recorded in the experimental group. Decreases in the values of ART and VRT were also observed in this group but were not significant statistically.

TABLE II : Data with statistical analysis of control subjects (Control group I) and subjects exposed to noise stress of <10 yrs duration (Experimental group I).

Parameters	*Control Group I (n=22) (Mean ± S.D)*	*Experimental Group I (n=14) (Mean ± S.D)*	*P value*
Age (yrs)	25.60 ± 2.30	26.60 ± 4.30	NS
Duration of exposure (yrs)	—	3.8 ± 2.30	—
HR (per min)	76.10 ± 13.20	86.6 ± 10.00	<.05
SBP (mm Hg)	109.90 ± 12.10	121.40 ± 16.10	<.05
DBP (mm Hg)	71.20 ± 7.30	78.10 ± 10.50	<.05
GSR (Kilo ohms)	523.40 ± 264.90	348.60 ± 171.90	<.05
ART (msecs)	207.50 ± 31.60	198.00 ± 57.60	NS
VRT (msecs)	213.70 ± 21.60	206.20 ± 45.60	NS

TABLE I : Hearing levels across various frequencies (Mean ± SD values) in control and Experimental groups.

	Right Ear		*Left Ear*	
	Control (n=25)	*Experimental (n=25)*	*Control (n=25)*	*Experimental (n=25)*
Age	*43.3 ± 6.6*	*44.7 ± 8.1*	*43.3 ± 6.6*	*44.7 ± 8.1*
frequencies	*Hearing loss in dB (Mean ± SD)*		*Hearing loss in dB (Mean ± SD)*	
125 Hz	29.4 ± 9.1	34.4 ± 8.8	31.2 ± 8.6	31.0 ± 13.3
250 Hz	32.8 ± 9.1	38.2 ± 10.8	35.8 ± 8.7	37.0 ± 9.4
500 Hz	31.6 ± 10.7	37.6 ± 11.6	31.8 ± 11.4	33.2 ± 14.6
750 Hz	25.6 ± 10.2	33.2 ± 14.9	24.0 ± 10.6	26.6 ± 11.9
1000 Hz	21.0 ± 10.9	27.0 ± 18.0	21.0 ± 10.5	21.0 ± 9.6
1500 Hz	19.0 ± 10.0	27.6 ± 20.3	19.2 ± 9.4	21.8 ± 12.9
2000 Hz	19.0 ± 10.5	24.8 ± 21.2	16.2 ± 9.5	17.0 ± 10.9
3000 Hz	19.4 ± 10.5	29.4 ± 20.1*	17.4 ± 11.3	28.2 ± 17.9*
4000 Hz	21.2 ± 13.6	37.0 ± 24.0**	18.6 ± 10.9	34.6 ± 20.7**
6000 Hz	30.6 ± 14.3	38.8 ± 29.9	24.0 ± 16.3	31.8 ± 17.1
8000 Hz	29.0 ± 18.0	27.8 ± 20.1	22.4 ± 15.9	24.6 ± 19.4

*P<.05; *P<0.1

TABLE III : Data with statistical analysis of control subjects (Control group II) and subjects exposed to noise stress of 10-20 yrs duration (Experimental group II).

Parameters	*Control Group II (n=40) (Mean ± S.D)*	*Experimental Group II (n=40) (Mean ± S.D)*	*P value*
Age (yrs)	43.70 ± 8.30	40.90 ± 3.80	NS
Duration of exposure (yrs)	—	16.70 ± 2.50	—
HR (per min)	81.70 ± 12.90	91.80 ± 13.90	<.01
SBP (mm Hg)	123.60 + 16.90	130.60 ± 8.70	<.05
DBP (mm Hg)	82.60 ± 8.70	86.90 ± 7.10	<.05
GSR (Kilo ohms)	678.20 ± 430.30	362.20 ± 180.40	<.001
ART (msecs)	223.00 ± 40.90	197.50 ± 47.30	<.05
VRT (msecs)	235.30 ± 66.50	202.90 ± 43.50	<.05

TABLE IV : Data with statistical analysis of control subjects (Control group II) and subjects exposed to noise stress of >20 yrs duration (Experimental group III).

Parameters	*Control Group II (n=40) (Mean ± S.D)*	*Experimental Group III (n=40) (Mean ± S.D)*	*P value*
Age (yrs)	43.70 ± 8.30	46.50 ± 4.30	NS
Exposure (yrs)	—	25.70 ± 2.90	—
HR (per min)	81.70 ± 12.90	89.50 ± 13.90	<.05
SBP (mm Hg)	123.60 ± 16.90	136.40 ± 14.10	<.001
DBP (mm Hg)	82.60 ± 8.70	87.90 ± 10.70	<.05
GSR (Kilo ohms)	678.20 ±430.30	401.40 ±190.50	<.001
ART (msecs)	223.00 ± 40.90	200.80 ± 52.40	<0.5
VRT (msecs)	235.30 ± 66.50	204.00 ± 33.60	<0.5

Experimental group II (10-20 yrs exposure to noise stress) showed a significant increase in HR (P<.01), SBP (P<.05), DBP (P<.05) as compared to control group II (Table III). There was also a significant decrease in GSR (P<.001), ART (P<.05) and VRT (P<.05).

Experimental group III (>20 yrs exposure to noise stress) when compared with control group II (Table IV depicts significant increase in HR (P<.05), SBP (P<.001), DBP (P<.05) and significant decrease in GSR (P<.001), ART (P<.05) and VRT (P<.05).

Thus Tables II-IV reveal that changes were statistically more significant in SBP, GSR, ART and VRT with increased duration of exposure to noise stress whereas changes in HR were statistically less significant with more than 20 yrs exposure to noise stress.

TABLE V : Prevalence of hypertension and estimation of hypertension risk associated with exposure to noise stress (Odds Ratio).

	Groups		
	I	*II*	*III*
Control			
Total subjects	22	40	40
No. of hypertensives*	-	3	3
Prevalence of hypertension	-	7.5%	7.5%
Experimental			
Total subjects	14	40	40
Exposure to noise stress	<10 yrs	10-20 yrs	>20 yrs
No. of hypertensives*	1	10	12
Prevalence of hypertension	7.1%	25%	30%
P value*	-	<.05	<.02
Odds Ratio**	-	4.1	5.3

*Chi-square test was used to compare the number of hypertensive individuals in the control and experimental groups.

**For risk of hypertension associated with duration of exposure to noise stress.

There was a higher prevalence of hypertension in subjects exposed to noise stress than those in the control groups (Table V). The prevalence of hypertension in the experimental group I was 7.1% which was increased to 25% (P<.05) in experimental group II, and 30% (P<.02) in experimental group III. The subjects exposed to noise stress with 10-20 yrs duration showed a risk of developing hypertension 4.1 times that of subjects not exposed to noise stress, whereas with increased duration (>20 yrs exposure) the risk of developing hypertension increased to 5.3 times.

DISCUSSION

In the Present study a significant hearing loss at 3000 Hz (P<.05) and 4000 Hz (P<.01) but not at other frequencies indicates that this impairment was due to noise (1, 6, 13).

A significant increase in SBP and DBP were recorded in all the three groups of workers exposed to noise stress. A similar increase in BP was also reported by Jonsson and Hansson (6) with noise stress. The rise in SBP became more significant (P<.001) with increased duration of exposure (>20yrs). The increased SBP with increased duration of exposure to noise (>20 yrs) could be attributed to increased release of plasma epinephrine (14). The less significant rise in DBP (P<.05) even with increasing duration of exposure to noise stress (>20 yrs) is probably due to the effect to epinephrine causing vasodilation in the skeletal muscle and the liver via B_2 receptors (15) balancing the vasoconstriction produced elsewhere by norepinephrine and therefore not further increasing the DBP as that of SBP.

The increased heart rate in the experimental group is probably due to increase in plasma epinephrine and norepinephrine (16) by their action on the β_1 receptors of heart. The increase in heart rate in experimental group II is more (P<.01) than in group III. It is probably because the increase in SBP is more in group III (P<.001) than group II, which increases the activity of baroreceptors leading to a reflex slowing of HR in group III. This increased sympathetic activity also may be the cause of decrease in GSR of workers exposed to noise stress in the present study.

A decrease in both auditory and visual reaction times was observed in workers exposed to noise stress in the present study. Broadbent (17) noted no change in simple tasks such as reaction time or clerical tasks whereas Finkelman et al (18) reported degradation of information processing performance with noise stress. The decreased reaction time in our study would be attributed to catecholamines which are associated with increase in alertness by decreasing the threshold of RAS and thereby shortening the reaction time (19).

From the present study, it may thus be concluded that noise stress causes increased sympathetic activity thereby leading to significant increase in BP, HR, and decrease in GSR, ART and VRT. It can also be concluded that prevalence of hypertension and its risk is higher in subjects exposed to noise stress. It was observed that all these modifications are related to duration of exposure to noise stress. Presumably, all the above effects are due to activation of hypothalamo sympathetic adrenal axis and resultant release of catecholamines from adrenal medulla due to noise stress (20). However, to say that all the extra auditory changes are permanent, needs further observation on a group of subjects who have been removed from noise stress for a short period. We intend to follow up these preliminary findings with detailed examinations of large numbers of individuals exposed to noise stress.

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