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CUMULATIVE EFFECTS OF COMMUNAL AND INDUSTRIAL NOISE ON CARDIOVASCULAR SYSTEM

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Summary: The aim of the present study was to assess the cumulative effects of communal and industrial noise on blood pressure, blood lipid fractions and coronary artery disease in workers exposed to noise in working and living environment.
The studied group included 180 workers employed in metal industry exposed to industrial noise levels exceeding 80dB and to communal noise levels exceeding 70dB. The control group consisted od 90 workers that have never worked or lived in a noisy environment. The studied and control group are similar with regard to factors other than noise exposure that can affect the cardiovascular system (sex, age, obesity, smoking habits, positive family history) These results indicate that communal and industrial noise could be possible contributing factors in the development of arterial hypertension, coronary heart disease, coronary risk factors (hypercholesterolaemia, hypertriglyceridaemia), vasospastic and atherosclerotic changes on arterial blood vessels of low extremitas.

Key words: Noise, arterial hypertension, coronary heart disease, cholesterol, tryglicerides

Introduction

Noise presents a significant nox which takes a leading position in the pollution of human working and living environment. The level of noise is getting higher and higher day by day, thus, in the last few years, it has become one of the main causes of damaging human health. Noise forces its way through every pore of human life, damages men at work, in the street, in their homes, by day and by night. The knowledge of harmful effects of noise is expanding day by day. Beside the well_known effects of noise on hearing organs, there are also less known and not enough defined extraauditive effects which could be registered in almost all the systems and organs of the exposed people. A disease caused by noise is a disease of the whole organism. For the last few years "sickness of noise" has become a modern disease, which may become the 21st century disease unless appropriate measures are taken. The predictions of Robert Koch are becoming more and more realistic. At the end of the last century Koch said: "A day will come when noise is going to become one of the biggest enemies of man, thus we' ll fight it the same way we fough colera and plague".

Many studies have shown that noise influences the cardiovascular, endocrine, metabolic, gastroin–testinal and neurological systems (1).

The cardiovascular system is considered to be the most involved and therefore has received the most attention, whereas few specific studies have been performed concerning the relationship between noise and myocardial disease. There is evidence that noise presents a significant factor in the genesis of arterial hypertension (2), coronary disease (3), and disorders of peripheral arterial circulation (4).

There s evidence that the use of appropriate preventive measures, the harmful effects of noise could be moderated and even eliminated (2). It is completely certain that the aplication of the personal protective means reduce these harmful auditive and extraauditive effects of the industrial noise (5).

The aims of this study were to evaluate the influence of noise exposure on blood pressure, to investigate the effects of noise on blood lipid fractions and coronary artery disease in workers,

exposed to noise in working and living environment.

Subjects and methods

The studied group included 180 workers employed in metal industry, exposed to industrial noise levels exceeding 80dB and to communal noise levels exceeding 70dB. The control group consisted of 90 workers that have never worked or lived in a noisy environment.

The examinations included and standardized questionnaire, measurement of weight, height, blood pressure, doppler echo – sonogram, electrocardiogram, polycardiogram, plethysmogram and determination of serum uric acid and lipid concentrations, Blood pressure data were taken with a mercury sphygmomanometer by a physician according to the method utilized by Jonsson and Hansson (6).

In this study, hypertension was defiend according to the recommendation of the WHO Expert Committee: Systolic blood pressure 21.3kPa or more, or diastolic blood pressure 12.7kPa or more (7). For borderline hypertension blood pressure values from 18.8/12kPa to 21.2/12.5kPa were taken, and for hypotension values of blood presure under 13.3/8kPa.

Diagnosis of coronary heart disease was established after clinical observation, ECG and echocardiography examination. A submaximal or symptom limited exercise test was performed. For the coronary heart disease the following criteria had to be fulfilled:

1.Previous miocardial infarction.

2.Chronic stable angina pectoris for more than three months.

3.Ischemic response on exercise electrocardio–gram. The exercise test was considered to be abnormal in the presence of more than 0,1mV horisontal or downsliping ST segment depression in at least two leads of the 12 lead standard electrocardiogram with or without anginal pain.

4.Regional myocardial contractility disturbances on echocardiogram.

5. Non–stable angina pectoris.

Body mass index (Quetelet s index) was used to show the ratio of body weight (W) and heigh (H) by following formula:

$Qi = W/H2$

Values of Qi of less than 2.13 were considered the sign of the underweight.

Values of Qi of 2.14 to 2.56 were considered the sign of the normal weight.

Values of Qi above 2.57 were the sign of the overweight.

Blood samples were drawn after an overnight fast, betwen 7.00–8.00 am, by cubital venipunc–ture. Serum cholesterol and uric acid concentra–tions was determined by a colorimetric method, serum trigliceride concentration by the extraction method.

Statistic analysis of the data obtained in the studied and control group was done.

Results

The mean age od the examined workers belonging to the control group was 48.9±10.3 years, and those of the noise exposed group was 49.1±11.9 years which does not present any statistic significant difference ($P>0.05$). The mean employment duration of the workers belonging to the control group was 19.8±8.4 years, and those of the noise exposed group was 19.24±7.6 years which does not present any statistic significant difference ($P>0.05$). Both groups have similar structure according to sex, obesity, smoking habits and hereditary predisposition to cardiovascular diseases ($P>0.05$) (Table 1).

Prevalence rates of hypertension were compared between the noise exposed and control groups. The prevalence of hypertension and borderline hypertension of thenoise exposed group were higher than that of the control group ($P<0.05$). The prevalence of hypotension of the control group was higher than that of the noise exposed group ($P<0.05$) (Table 2).

Prevalence rates of coronary artery disease of the noise exposed group were higher than that of the control group ($P<0.05$) (Table 3).

The analysis of morphological look of plethys–mographic curve and echosonogram of low extremitas showed statistic significant higher

Table 1. Comparations of study variables between the noise exposed and control group

Variables	Noise exposed group		Control group	
	N	%	N	%
Overweight	52	28.9	25	27.8
Underweight	37	20.5	19	21.1
Normal weight	91	50.6	46	51.1
Smokers	85	47.2	43	47.7
Non smokers	95	52.8	47	52.2
Males	157	87.2	79	87.8
Females	23	12.8	11	12.2
Positive family history	68	37.8	35	38.9

percentage of workers with vasospastic and atherosclerotic changes in the noise exposed group regarding those of the control group (Table 4).

Mean values of serum low density lipoprotein cholesterol, triglycerides and uric acid concentra–tions in the noise exposed group were significantly higher than in the control group .The prevalence of hypertriglyceridaemia and hypercholesterolaemia were significantly higher in the noise exposed than in the control group (Table 5).

Table 2. Distribution of blood pressure at workers of the noise exposed and control group

	Noise exposed group		Control group	
	N	%	N	%
Hypotension	14	7.7	20	22.2*
Normotension	83	46.1	47	52.2
Borderline hypertension	22	12.2*	4	4.4
Hypertension	61	33.8*	19	21.1

Statistical comparisons betwen the noise exposed and control group : *p < 0.05

Table 3. Coronary artery disease at workers of the noise exposed and control group

Variables	Noise exposed group		Control group	
	N	%	N	%
Angina pectoris stabilis	5	2.7	1	1.1
Angina pectoris non stab.	2	1.1	–	–
Previous myocardial nfarction	3	1.6	–	–
Totally	10	5.5*	1	1.1

Statistical comparisons betwen the noise exposed and control group : *p < 0.05

Table 4. Changes on arterial blood vessels of low extremitas of the examined workers

Variables	Noise exposed group n=180		Control group n=90	
	N	%	N	%
Normal blood vessels	115	63.9	88	97.7***
Vasospasm	47	26.1***	3	3.3
Atheroscleros. obliter.	16	8.9*	1	1.1

Statistical comparisons betwen the noise exposed and control group: * p < 0.05, *** p < 0.001

Table 5. Mean values of serum uric acid and lipid concentrations and prevalence of hypercholesterolemia and hypertrigliceridemia at workers of the noise exposed and control group

Variables	Noise exposed group		Control group	
	Mean (mmol/l)	SD	Mean (mmol/l)	SD
Triglycerides	1.8***	0.7	1.1	0.6
Uric acid	289.1*	84.1	261.5	79.2
LDL cholesterol	6.4***	1.4	5.2	0.9
HDL cholesterol	1.3	0.8	1.4	0.9
	N	%	N	%
Hypertriglyceridemia	47	26.1*	12	13.3
Hypercholesterolemia	53	29.4*	14	15.5

Statistical comparisons betwen the noise exposed and control group : * p < 0.05 , *** p < 0.001

Discussion

It is very important that the noise exposed and control group are similar with regard to factors other than noise exposure that can affect cardiovascular system.

The correlation between noise and high blood pressure has been interpreted as an evidence that noise is a cardiovascular risk factor (8,9). A separate problem is the mechanism of hypertension. Some data taken from the literature point to the following mecnanisms: The influence of catecholamines released from adrenall medulla as a result of activation of adrenergic system,the effect of suprarenal gland steroids, angiotensin and also the direct effect of noise on arterial wall tension (10,11).

Stimulation by noise, through sympathetic nervous mechanism, causes an elevation of blood pressure by an increase in total peripheral resistance and myocardial contractility (12). The repeated stimulation with noise could then accelerate the development of structural vascular changes in the peripheral resistance vessels and by this mechanism create a permanent blood pressure elevation to hypertensive levels (13). The blood pressure elevations was caused by vasoconstriction in patients with the essential

hypertension and in normotensive subjects with a positive family history of hypertension, while the blood pressure response in normotensive subjects without a family history of hypertension was due mainly to an increase in cardiac contractility (10). This might indicate that there are genetically determined differences in the cardiovascular response to noise.

The significant more values of plasma low density lipoprotein cholesterol and triglicerides in the noise exposed group than in the control group are considered important observations of this study because of their pathogenetic implications. These effects are compatible with the lipolytic action of adrenergic overactivity that may increase the mobilization of plasma free fatty acids from adipose tissue and the formation of triglycerides and cholesterol, integrating the lipoproteins. These modifications in the blood lipids and the elevation of blood pressure found in this study may exert a pathogenic action on cardiovascular system, where they may accelerate atherosclerosis. Also, chronically increased blood lipids plus the direct cardiotoxic effects of epinephrine and norepineph–rine can lead to coronary heart disease, degenerative changes in the myocardium and to atherosclerotic changes on arterial blood vessels of low extremitas.

The results of this study are in accordance with those of other authors who have studied the problem of cardiovascular effects of noise (3,14).

These results show that communal and industiral noise cause disturbance of cardiovascular system of the exposed workers, so that one should take appropriate preventive measures.

Conclusions

These results indicate that communal and industrial noise are a possible contributing factors in the development of arterial hypertension, myocardial infarction, angina pectoris non stabilis and vasospastica, coronary risk factors (hypercho–lesterolaemia, hypertriglyceridaemia), vasospastic and atherosclerotic changes on arterial blood vessels of low extremitas.

These results should not be neglected because our country belongs to these ones where heart–diseases and diseases of blood vessels are becoming freqent causes of death and early invalidity.

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KOMULATIVNI EFEKTI KOMUNALNE I INDUSTIJSKE BUKE NA KARDIOVASKULARNI SISTEM

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Kratak sadržaj: Cilj rada je bio da proceni kumulativne efekte komunalne i industrijske buke na krvni pritisak, serumske lipidne frakcije i koronarnu bolest kod radnika izloženih buci i životnoj i radnoj sredini. Ispitivanu grupu je činilo 180 radnika zaposlenih u metalopreradjivačkoj industriji, izloženih industrijskoj buci intenziteta preko 80dB i komunalnoj buci intenziteta preko 70dB. Kontrolnu grupu je činilo 90 radnika koji nikada nisu živeli i radili u bučnoj sredini. Kontrolna i ispitivana grupa su sem ekspozicije buci bile slične strukture u odnosu na faktore koji mogu uticati na stanje kardiovaskularnog sistema (pol, starost, gojaznost, navika pušenja, pozitivna porodična anamneza) ($P>0,05$). Rezultati pokazuju da su komunalna i industrijska buka mogući potencirajući faktori u nastanku arterijske hipertenzije, koronarne bolesti srca, faktora rizika koronarne bolesti (hiperholesterolemija, hipertrigliceridemija), vazospastičkih i aterosklerotičkih promena na krvnim sudovima donjih ekstremiteta.

Ključne reči: Buka, arterijska hipertenzija, koronarna bolest, holesterol, trigliceridi

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