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Blood Pressure Changes in Man during Infrasonic Exposure

An Experimental Study

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ABSTRACT. Danielsson Å. Landström U. (Department of Internal Medicine, University Hospital, and National Board of Occupational Safety and Health, Umeå, Sweden.) Blood pressure changes in man during infrasonic exposure. Acta Med Scand 1985; 217: 531–5.

Twenty healthy male volunteers were exposed to infrasound in a pressure chamber especially designed for the experiments. The effects on blood pressure, pulse rate and serum cortisol levels of acute infrasonic stimulation were studied in a series of different experiments. Varying frequencies (6, 12, 16 Hz) and pressure levels (95, 110, 125 dB(lin)) were tested. Significantly increased diastolic and decreased systolic blood pressures were recorded without any rise in pulse rate. The increase in diastolic blood pressure reached a maximal mean of about 8 mmHg after 30 min exposure. The results suggest that acute infrasonic stimulation induces a peripheral vasoconstriction with increased blood pressure, previously shown to occur in conjunction with industrial noise. Chronic long-term exposure to environmental infrasound may be of importance for the development of essential hypertension in predisposed individuals. *Key words: blood pressure, infrasound, frequency, intensity.*

The etiology of essential hypertension is complex and probably multifactorial. It seems well established that hereditary factors are important, and it has been suggested that environmental factors contribute substantially (1).

Noise is considered an environmental stress factor (2). Elevation of blood pressure during short-term exposure to noise has been demonstrated in controlled experiments in man (3). Moreover, an increased prevalence of hypertension has been reported in populations chronically exposed to traffic (4) or aircraft noise (5). Epidemiological studies on exposure to noise and hypertension have yielded somewhat conflicting results. Workers with noise-induced hearing dysfunction have been shown to have significantly higher blood pressure and an increased prevalence of hypertension than matched controls with normal hearing function (6). No relationship between exposure to noise and hypertension has been recorded in other studies (7, 8, 9). Previous investigations concerning the effects on blood pressure have in general dealt with noise of broad band frequencies, not distinguishing between low-frequency sound and sound within the normal hearing range.

The existence of high levels of environmental low frequency noise has been documented in recent years. However, little is known about whether the low frequency noise is harmful or not (10). In general, exposure to infrasound as high as 140 dB is recognized as being safe (11, 12). To our knowledge, no direct studies on blood pressure changes during infrasonic exposure have been carried out. The aim of the present investigation was to study the effects of different frequencies and pressure levels of infrasound on blood pressure and heart rate in healthy subjects under controlled conditions.

MATERIAL AND METHODS

The experiments were carried out in a pressure chamber especially constructed for the generation of low frequency sound. The dimensions of the chamber were 2.0×1.6×1.2 m, and the sound was

produced by eight 50 W loudspeakers, which were driven by two 100 W power amplifiers. By adjusting the length and diameter of a tube in the wall it was possible to tune the chamber to different frequencies according to the principle of a Helmholtz resonator.

The chamber was thoroughly tuned in order to reduce the harmonics as far as possible. The sound was considered acceptable when the pressure levels of the harmonics were below the auditory threshold level. The first harmonics were always more than 40 dB below the level of the main frequency and the following harmonics even lower. The background noise was about 50–55 dB(lin), (dB(A)<50). The dB(lin) is the physical sound level per se. The dB(A) is a weighted sound level correlated to the subjective response to different frequencies.

During exposure to noise the subject was seated inside the chamber. The chamber was air-conditioned to 20°C and provided with an intercommunication system and a window. The subjects were always allowed to adapt to the chamber for about 15 min prior to the experiment.

The project was approved by the local Committee of Ethics and carried out in 20 healthy male subjects, 20–30 years old.

Two different types of experimental designs were applied. In the first series of experiments, either different sound frequencies (6, 12 and 16 Hz) or different pressure levels (95, 110 and 125 dB(lin)) were tested. When the frequency was varied, the pressure level was adjusted to 125 dB(lin), and the different pressure levels were tested at a frequency of 16 Hz. All exposures were carried out for 20 min and were preceded and followed by silent control periods of the same duration. The order of the test periods was chosen at random. Blood pressure and heart rate were measured every second minute. A mean value for each period and individual was then calculated. In the second set of experiments, a one-hour test period was preceded and followed by silent control periods of the same duration. On alternate days the same subjects were randomly exposed to either infrasound (125 dB(lin), 16 Hz) or low grade high frequency noise (50 dB(lin), 50 Hz). The latter exposure copied that of the high frequency noise produced during infrasonic stimulation and served also as control periods. Blood pressure and heart rate were measured every fifth minute. During the experiment an intravenous canula was inserted and blood samples were taken at regular intervals for hormone analysis.

Blood pressure and heart rate were recorded automatically (Dinamap 845, 950). The equipment was thoroughly tested for possible interference from the test sound. Serum cortisol levels were measured by commercial radioimmunoassay.

Statistical evaluations were carried out by applying Student's *t*-test for group means and paired observations.

Table I. *Blood pressure (mmHg) and heart rate (strokes/min) during exposure to 125 dB(lin) infrasound at different frequencies and adjacent silent control periods (mean ± SEM, n=20)*

	Exposure (Hz)		
	6	12	16
Diastolic			
Test	66.2±2.2	65.8±2.2	67.3±1.9
Control	65.9±1.9	66.4±2.1	66.3±2.3
Difference	+0.3, $p<0.05$	−0.6, NS	+1.0, $p=0.05$
Systolic			
Test	118.3±1.6	118.9±1.8	117.4±1.6
Control	119.2±1.7	119.0±1.6	119.5±1.7
Difference	−0.9, NS	−0.1, $p=0.05$	+2.1, $p<0.01$
Pulse rate			
Test	59.1±1.6	59.3±1.9	59.2±1.8
Control	61.1±1.8	60.9±2.0	61.0±1.9
Difference	−2.0, $p<0.01$	−1.6, $p<0.01$	−1.8, $p<0.01$

The difference between test and control period was assessed by paired *t*-test. NS = not significant.

RESULTS

In the first series of experiments the effects of 6, 12 and 16 Hz on blood pressure and pulse rate were studied at a pressure level of 125 dB(lin) (Table I). In addition, sound pressure levels of 95, 110 and 125 dB(lin) at a static frequency of 16 Hz were investigated (Table II). The results were compared with the mean value of the surrounding control periods of 20 min. The results in Table I can be summarized as follows. Close to or statistically significant increases in diastolic blood pressure were recorded at 125 dB(lin), 6 and 16 Hz, as well as reductions in systolic blood pressure at 125 dB(lin), 12 and 16 Hz. A statistically significant increase in diastolic blood pressure was observed at 95 dB(lin), 16 Hz (Table II). There were no increases in heart rate during any period of exposure. On the other hand, significant reductions in heart rate were recorded at 125 dB(lin), 6, 12 and 16 Hz as well as at 110 dB(lin), 16 Hz.

The changes in blood pressure were small during the short-term stimulation with infrasound. In the second set of experiments, the subjects were exposed to 125 dB(lin), 16 Hz or to a 50 Hz sound adjusted to 50 dB(A). The experiment was divided into three periods of 60 min each. The test period was surrounded by silent control periods of the same duration, and the experiment was repeated twice with different exposures, infrasound or high frequency noise. The order of the exposures was selected at random. The results are shown in Fig. 1. There was a gradual increase in diastolic blood pressure and a concomitant decrease in systolic pressure during the first 30 min. The mean increase in diastolic pressure during infrasonic exposure amounted to 5.17 ± 0.90 mmHg and the reduction in systolic blood pressure to -3.20 ± 1.03 mmHg. During the control stimulation with 50 dB(A), 50 Hz, there was no effect on systolic or diastolic blood pressures. The difference between infrasonic and control exposure was significant for both diastolic ($p<0.001$) and systolic blood pressure ($p<0.05$).

The experiment also included determination of plasma cortisol during the different

Table II. *Blood pressure (mmHg) and heart rate (strokes/min) during exposure to 16 Hz infrasound at different pressure levels and adjacent silent control periods (mean ± SEM, n=20)*

	Exposure (dB)		
	95	110	125
Diastolic			
Test	71.8±1.5	70.8±1.5	71.3±1.5
Control	70.4±1.5	70.8±1.5	71.8±1.5
Difference	+1.4, $p<0.05$	−0.0, NS	−0.5, NS
Systolic			
Test	123.3±1.8	121.4±1.5	122.8±1.6
Control	122.8±1.8	121.6±1.6	122.4±1.7
Difference	−0.5, NS	−0.2, NS	+0.4, NS
Pulse rate			
Test	60.5±2.5	60.0±2.3	60.9±2.4
Control	61.1±2.4	61.2±2.4	60.8±2.3
Difference	−0.6, NS	−1.2, $p<0.01$	+0.1, NS

The difference between test and control period was assessed by paired *t*-test. NS = not significant.

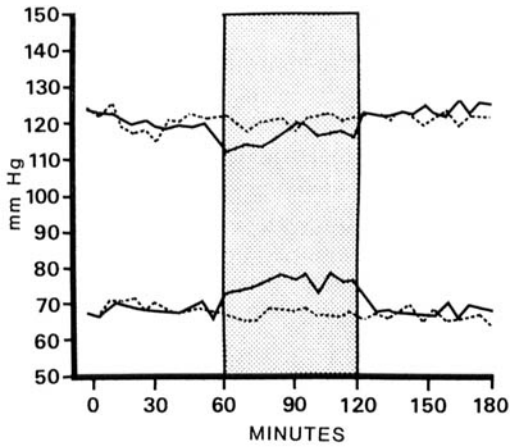

Fig. 1. Mean systolic and diastolic blood pressure during exposure to infrasound 125 dB(lin), 16 Hz or high frequency noise of 50 dB(A), 50 Hz. ▩ = test periods, —— = infrasonic exposure, --- = 50 Hz experiment.

periods. No significant changes were observed between the test and control periods (not shown).

DISCUSSION

The present investigation demonstrated that exposure of healthy individuals to infrasound 6–16 Hz increased the diastolic blood pressure and tended to decrease the systolic. The changes in blood pressure were small during 20 min of exposure, but became more marked after 30 min. The pulse rate was unchanged or slightly decreased. Exposure to 16 Hz seemed to have the most marked effect, elevating the diastolic blood pressure by about 8 mmHg. The effect was recorded for 95 dB as well as 125 dB, and was found to be maximal after 30 min of stimulation for 125 dB. The results obtained during exposure to infrasound were similar to those recorded for industrial and aircraft noise of broad band frequencies (3, 13, 14). The pulse rate decreased significantly during the test periods at different frequencies. An increased diastolic blood pressure without any increase in pulse rate could be ascribed to a rise in total peripheral resistance. A noise-induced increase in blood pressure and a concomitant peripheral vasoconstriction have been reported earlier (15, 16).

In patients with essential hypertension, broad band noise stimulation has been shown to increase mean arterial blood pressure. The effect was not counteracted by β-adrenoceptor blockade thus excluding increased sympathetic activity as causative (16). No changes in plasma cortisol levels were encountered during infrasonic exposure, which is in agreement with observations in studies on industrial noise (17).

The peripheral vascular resistance is increased in essential hypertension (18, 19). On the basis of the present study on infrasonic exposure, as well as previous studies on industrial noise (3, 17), it seems justifiable to assume that different kinds of noise stimulation elevate the blood pressure temporarily by increasing the peripheral resistance. Exposure to noise for decades could be a factor underlying smooth muscle changes of the precapillary resistance vessels and the subsequent development of manifest hypertension in predisposed individuals. In fact, the prevalence of cardiovascular disease is increased among people chronically exposed to aircraft noise (5), which consists to a great deal of infrasound. However, audible noise stimulation induces vasoconstriction in rats (20), but does not seem to cause hypertension in normotensive or spontaneously hypertensive rats after lifelong exposure (21).

Studies on noise-induced effects on blood pressure have yielded diverging results. The

characteristics of the noise frequencies involved in the physiological reactions are mostly unknown. In the present study, low frequency noise in the infrasonic region was shown to affect the blood pressure mainly by increasing the diastolic pressure without increasing the pulse rate. A frequency of 6 Hz has previously been shown to induce a drowsy state with EEG changes which may be hazardous in certain working situations (22). Otherwise infrasound has been regarded as safe. Further studies are needed to clarify the role of long-term infrasonic exposure for the development of essential hypertension.

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