

Effect of noise on blood pressure and 'stress' hormones

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Summary

1. Noise stimulation (95 dBA) for 20 min caused a significant increase in diastolic (12%, $P < 0.001$) and mean arterial pressure (7%, $P < 0.001$) in 15 healthy normotensive male subjects.

2. There was no significant change in systolic blood pressure or heart rate during exposure to noise.

3. Adrenaline, noradrenaline, prolactin, cortisol and growth hormone concentrations in venous plasma were not affected during noise stimulation.

Key words: adrenaline, cortisol, growth hormone, haemodynamics, hypertension, noise, noradrenaline, prolactin, stress.

Introduction

Noise is an important environmental stress factor in all industrialized societies. Adverse reactions to noise are hearing impairment [1], probably due to vasoconstriction in the arterioles leading to the cochlea and the organ of Corti [2], and increased vascular resistance [3], leading to hypertension [4, 5] and cardiac hypertrophy [6]. Metabolic disturbances have been reported, such as elevated levels of blood cholesterol, increased depositions of cholesterol in the tissues and a greater degree of aortic atherosclerosis [7]. Noise also influences the renal handling of salt with increased urinary output of sodium and potassium [8]. This effect is believed to be due to an isolated secretion of

oxytocin without concomitant secretion of vasopressin [8].

Noise has a teratogenic effect in rats [9, 10]. Reduced fertility has been described as well as an enlargement of the ovaries, which has been attributed to increased secretion of gonadotropins [11]. Deaf rats do not respond, indicating that the stimulus is truly auditory [12].

The secretion of a number of hormones may be increased by different stressful stimuli. Such hormones are adrenaline, noradrenaline, adrenal steroids, prolactin and, possibly, growth hormone [4, 13–20]. We have now studied the effect of a well-defined stress factor (industrial noise 95 dBA) on the secretion of adrenaline, noradrenaline, prolactin, growth hormone and cortisol in man.

Subjects and methods

Subjects. Fifteen healthy male medical students volunteered for the study. They had normal hearing as tested by audiometry. Their mean age was 26 years (range 23–31). We instructed the subjects carefully before the investigation in order to avoid the influence of anxiety and 'expectancy stress'. The subjects were also told to avoid smoking tobacco and drinking coffee on the day of the investigation.

Noise stimulation procedure. All experiments were performed in the afternoon in a specially equipped noise laboratory. This consisted of an exposure room with eight loudspeakers built into the walls. The subjects rested comfortably on a bed in the recumbent position in the centre of the room. The sound level was 40 dBA. The room was air-conditioned and the temperature was kept

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at 20°C. The rest period before application of the noise stimulus was 20 min. After 20 min of industrial noise (95 dBA) the subjects were studied for another 15 min.

Tape recorders, amplifiers and equipment for acoustical measurements were placed in an adjacent control room. Recorded industrial noise was replayed through the loudspeakers. Throughout the studies the noise intensity in the exposure room was measured continuously at ear level about 10 cm from the subject's head. The A-weighted noise level and the frequency spectrum of the noise were analysed.

The acoustical measuring equipment was made by Brüel and Kjaer (Copenhagen, Denmark; microphone type 4145, preamplifier type 2619, real time analyser type 3347, sound level recorder type 2305 and sound level analyser type 4426).

Blood pressure measurement. Blood pressure and heart rate were recorded repeatedly before, during and after exposure to noise. Blood pressure was measured non-invasively in the brachial artery by an automatic blood pressure recorder (Bosch Bosomat). The accuracy of the blood pressure measurements had previously been assessed by simultaneous comparisons with blood pressure measurements made with a mercury sphygmomanometer (phase V). Here, the correlation coefficient between simultaneous measurements was $r = 0.92$, $P < 0.001$ for systolic blood pressure values and $r = 0.93$, $P < 0.001$ for diastolic blood pressure values.

Blood sampling procedures. Venous blood was collected for catecholamines, prolactin, growth hormone and cortisol at the end of the resting period. Blood samples for measurement of adrenaline and noradrenaline were then drawn after 5 min of exposure to noise. Prolactin, growth hormone and cortisol were measured after 20 min of such exposure. All blood samples for catecholamines were collected in heparinized tubes containing solid reduced glutathione and all tubes were placed in ice immediately and centrifuged in a cold room (4°C). The plasma was then removed and stored at -30°C until analysed.

Adrenaline and noradrenaline were determined

by a sensitive isotope derivative method and by ion-pair liquid chromatography [21].

Growth hormone, prolactin and cortisol were determined by double-antibody radio-immunoassays. For the growth hormone assay we used purified human growth hormone and rabbit anti-growth hormone from AB Kabi (Stockholm, Sweden), swine anti-(rabbit IgG) from DAKO immunoglobulins A/S (Copenhagen, Denmark) and the WHO standard 66/217 (Medical Research Council, London). Labelling was carried out by the lactoperoxidase procedure and purification by gel filtration (Sephadex G 75, 2.5 cm × 60 cm column). The standards were dissolved in phosphate-buffered sodium chloride solution containing bovine albumin (20 g/l) and polyethylene glycol (20 g/l). Prolactin and cortisol were assayed with reagents from Diagnostic Products Inc. (Los Angeles, CA, U.S.A.). The samples were assayed within a single assay series, and the intra-assay imprecision (CV) was less than 5% for the three methods.

Statistical methods. Student's *t*-test for paired observations was used for statistical evaluation (two-tailed). $P < 0.05$ was accepted as the minimal value for statistical significance.

Results

The results from the plasma hormone assays are given in Table 1. In general, there was a wide spread in the values. The mean change in plasma noradrenaline concentration was 6% ($P > 0.05$) and the mean change in plasma prolactin concentration was 3% ($P > 0.05$). For growth hormone the values were low and unchanged in 12 individuals. In two individuals there was a rise, whereas, in one individual, there was a sustained elevation. For plasma adrenaline concentration there was a rise in only one of nine individuals and for cortisol there was a definite rise in only two out of 15 individuals. The group mean values decreased for both hormones ($P > 0.05$). In summary, there was no significant concentration changes in any of the plasma hormones studied.

The diastolic and mean arterial blood pressures

TABLE 1. Hormone concentrations in plasma from subjects at rest (40 dBA) and during exposure to noise (95 dBA)

Mean results ± SEM are shown. Results are not significant.

Noise (dBA)	Noradrenaline (nmol/l)	Adrenaline (nmol/l)	Prolactin (µg/l)	Cortisol (nmol/l)	Growth hormone (munits/l)
40	1.25 ± 0.17	0.27 ± 0.11	5.83 ± 0.53	286 ± 30	2.29 ± 1.35
95	1.33 ± 0.16	0.24 ± 0.10	5.98 ± 0.46	266 ± 18	3.19 ± 1.57
	$t = 0.525$	$t = -0.638$	$t = 0.773$	$t = -0.881$	$t = 1.234$

TABLE 2. Blood pressure and heart rate in subjects at rest (40 dBA) and during exposure to noise (95 dBA)

Mean results \pm SEM are shown.

Noise (dBA)	Arterial blood pressure (mmHg)			Heart rate (beats/min)
	Systolic	Diastolic	Mean pressure	
40	118.9 \pm 3.4	69.8 \pm 2.4	86.1 \pm 2.4	59.0 \pm 2.4
95	120.6 \pm 3.3	78.2 \pm 2.2	92.3 \pm 2.2	60.4 \pm 2.3
	$t = 0.748$ $P = \text{N.S.}$	$t = 4.979$ $P < 0.001$	$t = 4.372$ $P < 0.001$	$t = 1.305$ $P = \text{N.S.}$

TABLE 3. Correlation coefficients between changes in haemodynamic and hormonal factors

	Δ Arterial blood pressure			Δ Heart rate
	Systolic	Diastolic	Mean	
Δ Adrenaline	0.42	0.01	0.22	-0.38
Δ Noradrenaline	0.04	-0.30	-0.22	-0.18
Δ Prolactin	-0.04	-0.24	-0.20	0.28
Δ Growth hormone	-0.09	-0.04	0.07	0.02
Δ Cortisol	0.01	-0.53*	-0.43	0.10

* $P < 0.05$.

increased by 12% ($P < 0.001$) and 7% ($P > 0.001$; Table 2). Systolic blood pressure and heart rate increased by 1% ($P < 0.05$) and 2% ($P > 0.05$; Table 2). There was a significant negative correlation between the changes in cortisol concentration and diastolic blood pressure ($r = -0.53$; Table 3). There were no significant correlations between the other hormonal and haemodynamic changes during exposure to noise (Table 3). The diastolic and mean arterial blood pressure elevations persisted for 5 min after cessation of noise stimulation and had disappeared after 10 min of rest at 40 dBA.

Discussion

Noise alone or together with other stress stimuli can produce blood pressure elevation and accelerate the development of permanent hypertension in rats [4, 5, 14, 22]. There is a marked difference in susceptibility in different strains of rats [23]; only individuals with a normal peripheral sympathetic function develop noise-induced hypertension [4]. This indicates that there are genetic factors that control the sympathetic activity to different stress forms, including noise. In humans, several studies have shown that acute stimulation by noise causes mainly a diastolic blood pressure elevation [3, 24-27]. The peripheral resistance may increase as a result of stimulation by noise [3, 27] and the pulse pressure decreases [3, 24-27].

The hormonal changes in animals such as increments of urinary catecholamines [13, 14] and of serum corticosterone [4, 14, 20, 28] induced by noise are transient and disappear in a few weeks in spite of persisting noise stimulation [4, 14]. Hormonal responses to noise in humans, reported by Arguelles *et al.* [29, 30], are increased excretion of urinary catecholamines and increased concentration of plasma 17-hydroxycorticoids [29]. The urinary 17-ketogenic steroid excretion was also increased [29]. Brandenberger *et al.* [31], however, found no increase in plasma cortisol during stimulation by noise.

Whereas, in the present study, industrial noise (95 dBA) produced a temporary elevation of diastolic and mean arterial blood pressures in man, there were no changes in heart rate or systolic blood pressure, nor was there any evidence that the noise produced any significant effect on the hypothalamus-pituitary system responsible for prolactin and growth hormone release. Nor was there any significant activation of the adrenal medullary system.

The negative correlation between the change in cortisol concentration and diastolic blood pressure was the only correlation out of 20 which attained statistical significance. This is exactly what would be expected by chance and for this reason we consider it a random occurrence (Table 2).

The haemodynamic results in this study are thus well in concordance with results from

previous animal and human studies. There may be several explanations for the divergent results as regards hormone response to noise in humans. Firstly, there are important methodological differences between this study and the studies by Arguelles *et al.* [29, 30]. In measuring acute effects of noise we considered it more reliable to measure the plasma levels of catecholamines as opposed to the urinary levels used by Arguelles *et al.* [30]. Secondly, it is difficult to separate the effects of anxiety and 'expectancy stress' from the effects of pure noise stimulation. We tried to diminish such unspecific effects of 'expectancy stress' by choosing medical students whom we knew well and who were carefully instructed in the study design before the investigation.

Noise is probably a stronger 'stress factor' for animals than for humans. Hearing is often essential for the survival of animals; thus non-familiar noise could produce a fully developed defence-alarm reaction with haemodynamic and hormonal adaptation for fight and flight. The hormonal response in animals (catecholamine and corticosterone elevation) may be due to fear of an unfamiliar noise and not due to the noise itself. This is supported by the fact that the hormonal elevations are only transient and disappear in spite of continued stimulation by noise [20].

Acute noise stimulation in man, although easy to standardize in physical terms, could be perceived differently under other environmental conditions. The model we used induced a slight discomfort in the subjects but there was never any feeling of threat or fear because they knew that the noise was not harmful.

There are several epidemiological studies which suggest that individuals exposed to noise for many years may develop hypertension [32–34]. The haemodynamic changes induced by noise exposure in animals persist in spite of only transient increases of catecholamines and corticosterone [4, 14]. This indicates that the prolonged elevation of blood pressure after repeated exposure to noise does not depend on elevated levels of these hormones.

Obviously, the findings of the present study do not indicate that the elevation of blood pressure during exposure to noise is dependent on elevated levels of catecholamines, cortisol, prolactin and growth hormone.

A more likely explanation is that acute stimulation by noise, through sympathetic nervous mechanisms, causes an elevation of blood pressure by an increase in total peripheral resistance, as shown in our previous studies [3].

Repeated stimulation with noise could then

accelerate the development of structural vascular changes in the peripheral resistance vessels [35] and by this mechanism create a permanent blood pressure elevation to hypertensive levels [4, 35].

Thus noise could be one of several environmental risk factors for the development of permanent hypertension in man.

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