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Occupational exposure to noise and hypertension in pilots

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Abstract

The role of occupational exposure to noise as a hypertension risk factor has not been established sufficiently. The aim of the study is to evaluate whether chronic exposure to different levels of noise in two groups of pilots, operating with two types of aircraft, could be a risk for hypertension, what relevance the parameters (intensity, duration and type) of exposure can have and, lastly, whether there are any links between hearing impairment and hypertension. After excluding pilots with confounding factors, a study was made of 77 male pilots of turboprop planes (group A) and 224 male pilots of jet aircraft (group B), matched by age and working life. Blood pressure (supine and standing positions) and heart rate were measured. Electrocardiogram, stress tests on a cycle ergometer, sound-level measurement and audiometric tests were also done. Pilots of group A were exposed to Leq of 93 dBA while pilots of group B were exposed to the Leq of 79 dBA. Significant results in group A compared to group B were found between heart rate, blood pressure, drop in blood pressure, parameters (intensity, duration and type) of exposure to noise and between hearing damage and hypertension. The findings suggest that chronic exposure to noise is a risk factor for blood hypertension in pilots exposed to high noise levels, and that the drop in blood pressure may be a sign of more sensitive effect of noise on blood pressure, according to other studies in literature.

Keywords: *Noise, hypertension, pilots, blood pressure, heart rate*

Introduction

It is well known that noise can seriously damage the hearing system but it is only recently that research has been done on its possible contribution to hypertension (Van Kempen et al. 2002) and cardiovascular disease (Tomei et al. 1991; Kawada & Suzuki 1999). It can be suggested that noise influences vascular tone, through direct and/or mediated mechanisms. The non-auditory effects of noise can manifest themselves through a series of nerve circuits represented, in addition to the reticular substance, by the limbic system, amygdala,

hippocampus, hypothalamus and cortical nerve centres, also by autonomous nervous system (Bao et al. 1999).

Data on how noise affects different cardiovascular parameters are still conflicting, the different results may be linked to measurements taken at different stages of noise exposure (Tomei et al. 1992; Sokas et al. 1995; Marciniak et al. 1999). Sokas et al. (1995) investigated a possible relationship between noise-induced hearing loss and high blood pressure (BP). Other investigations are still being made to see whether there are any links between hearing impairment, hypertension and cardiovascular disease (Tomei et al. 2000).

The aim of the study is to evaluate whether chronic exposure to different levels of noise in two groups of pilots operating two types of aircraft, could be a risk for hypertension, what relevance the parameters (intensity, duration and type) of exposure can have and, lastly, whether there are any links between hearing impairment and hypertension.

Methods

Four hundred-and-sixteen male pilots comprised of 105 pilots of turboprop planes G222 and C130 (group A) and 311 pilots of jet planes F104 and G91 (group B) were randomly extracted from two populations ($n = 1471$, in total) of aircraft military pilots. Both samples of 105 and 311 pilots correspond to 28.1% of each category of pilots. Pilots in both groups were identified and classified in relation to the type of aircraft almost exclusively used (same type of aircraft $> 90\%$).

Excluded from the study were pilots of both types of aircraft, who were aged less than 24 years, who had a working life less than two years, a family history of cardiovascular disease and exposure to audiological risk factors outside work (noise trauma, bomb, mine or other explosions, use of firearms, previous otitis, severe head trauma, use of ototoxic drugs). Also excluded were all pilots with serum total cholesterol > 200 mg/dl, blood glucose > 110 mg/dl, body mass index (BMI Kg/m^2) more than 30 and smoking habits (past and/or present smoking habit). As far as alcohol consumption was concerned, all the subjects included in the study were teetotallers or had negligible alcohol consumption (no spirits and less than 1 glass of wine or beer once a day or less).

Therefore, 115 workers were excluded from the study (mean age 41.9, SD 7.0 years, range 25 – 59 years; mean working life 21.9, SD 6.8 years, range 2 – 40 years). Thus, 301 pilots were studied: 77 in group A (turboprop pilots) and 224 in group B (jet pilots) matched by age and working life (by mean, SD and distribution; see Table I). Both populations of pilots perform analogous shift work. The flight hours from the start of the pilot's working life were calculated by means of a questionnaire and a check on the personal flight booklet. The pilots were further broken down into two sub-groups of pilots with flight hours of < 3000 and $\geqslant 3000$, because 3000 hours was approximately the mean flight hours in both groups (see Table I). An International Electrotechnical Commission (IEC) 804 Class 1 noise meter-integrator (Amplifon Amplaid 309, Italy) was used to measure the equivalent continuous noise level (Leq), i.e., (the mean intensity of a variable noise, integrated over time, for the various working stages, expressed in decibels A (dBA), and computed the noise-frequency distribution). The measurements were taken in the cockpit with a phonometer situated at the level of the pilot's ear, inside the protecting flight helmet. All the subjects, after at least 16 hours not exposed to noise, underwent a liminal tonal audiometric examination for air and bone conduction, in a range of frequencies from 125 to 8000 Hz. The audiometric curves for each ear were classified according to Klockoff, as modified by Merluzzi et al. 1987. Audiometry was done in accordance with International Standards Organization (ISO) rules 6189 – 1983, against normal room background noise, so as to measure an audible threshold of

Table I. Characteristics of the population studied.

Parameters	Group A (turboprop planes pilots) $n = 77$		Group B (jet planes pilots) $n = 224$	
Age (yr)				
mean (SD)	40.5 (8.0)*		39.0 (8.0)	
range	25 – 57		23 – 56	
Working life (yr)				
mean (SD)	20.6 (7.8)*		19.6 (8.5)	
range	4 – 36		4 – 38	
Flight hours				
mean (SD)	3285.7 (1665.6)*		2851.9 (1769.6)	
range	800 – 10040		750 – 10600	
	< 3000 $n = 29$	⩾ 3000 $n = 48$	< 3000 $n = 141$	⩾ 3000 $n = 83$
Flight hours				
mean	1950.0	4395.5	1849.6	4554.8
(SD)	(607.1)	(1554.8)	(865.3)	(1762.7)
range	800 – 2900	3000 – 10040	800 – 2900	3000 – 10600

* not significant with respect to group B.

0 dB, in accordance with ISO 389 – 1979. Audiometric findings for workers exposed to noise were compared with those for the general population, not exposed to noise, and not suffering from otological disease as specified in ISO 1999 – 1990. The correlation between flight hours and degree of hearing loss was also assessed. The workers fasted for at least 10 hours, and rested for at least 15 min in a room at a comfortable temperature, before the recording of heart rate (HR), BP, and electrocardiogram (ECG). All the workers were asked not to take any anti-hypertension medication for 12 hours before the test.

BP was measured in the morning after 5 min lying down, again 3 min later, and a third time after another 3 min, with the subjects still lying down, and to study any postural change 30 sec after they stood up according to World Health Organization guidelines (WHO 1999). The mean of the first three measurements was considered the mean BP and used to calculate the percentage of basal hypertensives (BH), using the WHO classification (WHO 1999). Lastly, the cases with a drop in systo-diastolic pressure while standing up (drop in BP) were taken into consideration. The cut-off point used for detecting the drop in systolic-diastolic blood pressure was 5 mmHg.

At resting HR ⩾ 84 beats/min is predictive of an increase in cardiovascular mortality (Petiot et al. 1992). The percentage of subjects with at rest HR ⩾ 84 beats/min was calculated. The stress test on the cycle ergometer was done immediately afterwards, in a seated position, increasing intensity of the stress by 30 W every 3 min with 50 revolutions a minute until the load that resulted in a HR of (200-age) was reached. The functional parameters, HR, BP and ECG were measured at each workload up to the maximum load and after 6 min of recovery.

All of the subjects consented to their personal details being available, declaring that they had been made aware that this data is ranked as ''sensitive information'', and consented that the data arising from the research protocol should be treated in an anonymous and collective way, with scientific methods and for scientific purposes in accordance with the principles of the Declaration of Helsinki.

Data were statistically analysed using frequencies, mean and standard deviation (SD) according to the nature of variables. Differences between means were assessed by Student's *t*-

test. Analyses of the variance were done to compare the means between the groups. Differences between frequency distributions were assessed by the chi-square test; Yate's correction for 2×2 contingency tables or the Fischer's exact test was used. The Pearson correlation coefficient (r) was calculated. A p value less than 0.05 was considered significant. Solo BMDP® statistical software was used for the statistical analyses.

Results

In group A, the equivalent continuous noise level (Leq) was 93 dBA, with peaks at takeoff and landing of 104 dBA. The noise-frequency distribution is unbalanced for low frequencies. In group B, Leq was 79 dBA with similar characteristics related to frequencies. There was during taxiing an increase of 33 dB at 4000 Hz and up to 110 dB at 250 Hz. Mean dyastolic blood pressure (DBP) and basal HR were significantly higher in group A than in group B (respectively $p = 0.001$ and $p = 0.001$) (see Table II).

There were significant differences between group A in respect to group B for the HR $\geqslant 84$ beats/min ($p = 0.001$), for the frequency of BH ($p = 0.001$), for the drop in BP ($p = 0.005$) and for the ECG anomalies ($p = 0.001$) (see Table II).

The prevalence of audiometric deficits was significantly higher in group A compared to group B ($p = 0.002$) (see Table II). The audiometric deficits due to noise exposure consisted of: Class II, 62% in group A and 63.2% in group B; class III, 6.9% and 10.3% respectively; class IV, 13.9% and 7.4% respectively; and class VI, 17.2% and 19% respectively.

The mean rise in the audiometric threshold in pilots of both groups was clearly higher than that expected in the general population of the same age not exposed to noise in ISO 1999 –

Table II. Heart rate (HR), systolic blood pressure (SBP), dyastolic blood pressure (DBP), HR $\geqslant 84$ beats/min, basal hypertension (BH), drop in BP, ECG anomalies and audiometric deficits in two groups.

Parameters	Group A (turboprop planes pilots) $n = 77$	Group B (jet planes pilots) $n = 224$
HR b/min		
mean (SD)	87.9 (10.1) ⋆	71.2 (11.2)
range	56 – 102	50 – 105
SBP		
mean (SD)	127.0 (13.3)	125.6 (11.2)
range	100 – 165	95 – 160
DBP		
mean (SD)	84.9 (10.1) ⋆	78.8 (9.4)
range	60 – 110	60 – 115
HR $\geqslant 84$ b/min		
No. cases (%)	30 (38.9%) ⋆	40 (17.8%)
BH		
No. cases (%)	30 (38.9%) ⋆	20 (8.9%)
Drop in BP		
No. cases (%)	47 (61.0%) ⋆⋆	68 (30.3%)
ECG anomalies		
No. cases (%)	30 (38.9%) ⋆	35 (15.6%)
Audiometric deficits		
No. cases (%)	30 (38.9%) #	46 (20.5%)⋆

⋆$p = 0.001$ with respect to group B; ⋆⋆ $p = 0.005$ with respect to group B; # $p = 0.002$ with respect to group B.

1990. Prevalence of BH, drop in BP and ECG anomalies in relation to audiometric deficits are shown in Table III.

Strong correlations between flight hours and systolic blood pressure (SBP) and DBP were found in both group A (SBP, $r = 0.97$, $p = 0.003$; DBP, $r = 0.98$, $p = 0.003$) and group B (SBP, $r = 0.86$, $p = 0.025$; DBP, $r = 0.76$, $p = 0.005$). No correlation was found between flight hours and ECG anomalies. A correlation was also found between flight hours and degree of hearing loss in both group A ($r = 0.88$, $p = 0.02$) and group B ($r = 0.87$, $p = 0.02$). The BH, the drop in BP, the ECG anomalies and the auditory deficits in relation to flight hours (< 3000 flight hours and > 3000 flight hours) are shown in Table IV.

The stress test on the cycle ergometer showed a greater systolic hypertensive response ($p = 0.001$) in the BH compared to the normotensives in both groups. The different SBP during stress between subjects with BH and normotensives was also maintained ($p = 0.001$) during the recovery phase. The maximal workloads reached were found to be in relation to age and working life. The subjects with a maximal load of up to 120 W mainly belonged ($p = 0.001$) to group A, while those with a maximal load of up to 210 W belonged to group B (no.6 = 1.9%).

In group A, a greater prevalence of ECG anomalies during ergometric test to the maximum load were found, compared to group B (group A 28.6%, group B 8.9%; $p = 0.001$), and to recovery (group A 25.9%, group B 8.9%; $p = 0.001$).

In group B, the interruption at the various loads showed the same in pilots with and without drop in basal BP. In group A the subjects with interruption at 150W were mainly those with drop in basal BP, the ones at 180W were mainly those without drop in basal BP ($p = 0.045$).

The HR increased in parallel to the BP, although the former tended to increase more. There were no significant differences in the HR during stress and on recovery, between the two groups of pilots. Some cases showed a low increase in HR at maximum stress (group A 2.8% and group B 2.2%). In all cases, the reason for interruption of the test was muscular exhaustion (7.6% in group A, 5.4% in group B); there was no statistically significant

Table III. Prevalence of basal hypertension (BH), drop in BP and ECG anomalies in relation to audiometric deficits.

	Group A (turboprop planes pilots) $n = 77$		Group B (jet planes pilots) $n = 224$	
	With audiometric deficit $n = 30$	Without audiometric deficit $n = 47$	With audiometric deficit $n = 46$	Without audiometric deficit $n = 178$
HR $\geqslant$ 84 b/min				
No. cases (%)	21 (70 %)$\star$	9 (19.1 %)	10 (21.7 %)$\star\star$	30 (16.8 %)
BH				
No. cases (%)	24 (80 %)$\star$	6 (12.7 %)	12 (26.1 %)	8 (4.5 %)
Drop in BP				
No. cases (%)	24 (80 %)#	23 (48.9 %)	23 (50 %)	45 (25.3 %)
ECG anomalies				
No. cases (%)	21 (70 %)$\star$	9 (19.1 %)	8 (17.4 %)	27 (15.1 %)

$\star p = 0.001$ with respect to pilots without audiometric deficit in the same group and pilots with audiometric deficit in group B; $\star\star p = 0.001$ with respect to pilots without audiometric deficit in the same group; # $p = 0.013$ with respect to pilots without audiometric deficit in the same group and $p = 0.017$ with audiometric deficit in group B.

Table IV. The BH, the drop in BP, the ECG anomalies and auditory deficits in relation to the time of exposure in the two groups of pilots studied.

	Group A (turboprop planes pilots) $n = 77$		Group B (jet planes pilots) $n = 224$	
	< 3000 $n = 29$	$\geqslant 3000$ $n = 48$	< 3000 $n = 141$	$\geqslant 3000$ $n = 83$
Flight hours				
mean (SD)	1950.0 (607.1)	4395.5 (1554.8)	1849.6 (865.3)	4554.8 (1762.7)
range	800–2900	3000–10040	800–2900	3000–10600
Age				
mean (SD)	35.6 (9.1)	43.4 (5.6)★	37.2 (8.2)	43.3 (6.8)★
range	25–56	34–55	25–56	35–56
Working life				
mean (SD)	15.9 (8.9)	23.4 (5.6)★	16.3 (8.2)	23.4 (6.3)★
range	4–36	16–34	4–36	15–32
HR $\geqslant 84$				
No. cases (%)	5 (17.2 %)	25 (52.0 %)★★	21 (14.8 %)	19 (22.9 %)
BH				
No. cases (%)	2 (7.0 %)	28 (58.3 %)★★★	8 (5.6 %)	12 (14.4 %)#
Drop in BP				
No. cases (%)	10 (34.4 %)	38 (79.2 %)★★★	40 (28.4 %)	28 (33.7 %)
ECG anomalies				
No. cases (%)	7 (24.1 %)	23 (47.9 %)##	21 (14.8 %)	14 (16.9 %)
Hearing loss				
No. cases (%)	6 (20.1 %)	24 (50.0 %)§	26 (18.4 %)	20 (24.1 %)

★$p = 0.001$ with respect to pilots < 3000h in the same group; ★★$p = 0.005$ with respect to pilots < 3000h in the same group and $p = 0.001$ with respect to pilots $\geqslant 3000$h in group B; ★★★$p = 0.001$ with respect to pilots < 3000h in the same group and to pilots $\geqslant 3000$h in group B; #$p = 0.049$ with respect to pilots < 3000h in the same group; ## $p = 0.001$ with respect to pilots $\geqslant 3000$h in group B; §$p = 0.021$ with respect to pilots < 3000h in the same group and $p = 0.005$ with respect to pilots $\geqslant 3000$h in group B.

difference between the two groups. No significant differences were found in prevalence of BH and basic ECG anomalies, age and working life between the two groups of pilots that interrupted the test.

Discussion

Considering that the principal confounding factors were excluded from the study and that pilots investigated were matched by age and working life, the above mentioned results suggest that exposure to noise in pilots, related to intensity and type of exposure may have an influence on BH and on HR (Kawada & Suzuki 1999; Tomei et al. 1991; Tomei et al. 1992; Byers & Smyth 1997; Talbott et al. 1999; Van Kempen et al. 2002).

The pilots of group A, who are at higher levels of exposure to noise, are at higher risk of BH, in particular DBP, compared to group B.

In a group of workers, Talbott et al. (1999) assumed that chronic exposure to noise at levels > 89 dB(A) was a significant predictor of DBP.

The pilots in both groups could be exposed to other cardiovascular risk factors such as: vibration, acceleration, shifts, hypoxia and the variations of the microclimate (Van Kempen 2002).

Some authors compared HR with BH (Burger et al. 1998) and atherosclerosis (Rehman et al. 1997). Studies showed that a resting HR $\geqslant 84$ beats/min, is predictive of an increase in cardiovascular mortality (Petiot et al. 1992). Tachycardia in hypertensives may reflect a sympathetic hyperactivity and a reduction in the vagal tone (Holand et al. 1999) and can be an additional indicator of cardiovascular risk. Many authors found that chronic exposure to noise provokes an increase in HR, probably due to a catecholamines response (Byers and Smyth 1997).

In the pilots of group A, ergometric tests confirm that exposure to higher levels of noise influence BH, reducing response to workloads, probably due to lower sympathetic adaptability (Marciniak et al. 1999). In aeronautical workers exposed to noise at levels of > 90 dB(A) Marciniak et al. (1999) have found structural change in the heart.

The reaching of the workloads may also depend on whether the subjects show a drop in basal BP or not. The low increase in the HR at maximum load might in some cases depend on muscular training or could be a sign of poor dysregulation adaptability of the sympatho-vagal balance.

Even though the level of noise while cruising, markedly lower in jet planes than in turboprop planes, is obviously important, account could also be taken of peaks during takeoff and landing, which are usually more numerous in the former (jet pilots land and take off more often than turboprop pilots). In jet planes the peaks are also related to the opening of the canopy during taxiing.

Studies on the relationship between hearing impairment and hypertension indicate that the response of the cardiovascular system is different from that of the hearing system (Merluzzi et al. 1987; Van Kempen et al. 2002).

The results on the relationships between BH and audiometric deficits seem to indicate that the vascular damage is accompanied by hearing impairment. The behavior observed is not similar for the ECG anomalies, indicating a less close relationship between these parameters and hearing impairment. In previous studies, it has been suggested that noise, depending on the intensity, duration and type of exposure and individual sensitivity, causes effects with different thresholds both for the system affected (cardiovascular and/or auditory) and the occurrence of the damage (type and extent): audiometric deficits and/or BH (Tomei et al. 1992; Van Kempen et al. 2002). Lastly, considering that the noise is mainly distributed over low frequencies, it could be suggested that studies should be done to confirm whether noise at low frequencies can have greater and more specific hypertensive effects (Tomei et al. 1992).

Noise reduction by means of flight helmets and headphones, already moderate at low frequencies, is further reduced by intercommunication systems. The effectiveness of such personal means of protection in satisfactorily attenuating noise, especially at low frequencies, is therefore not certain.

The results described agree with previous studies (Tomei et al. 1992; Van Kempen et al. 2002); drop in BP is a sign of a more sensitive cardiovascular effect than BH and ECG anomalies.

In this study, age is significantly different for pilots with > 3000 flying hours for both types of pilots. Therefore, age and flying co-vary in the data. However, in pilots a greater number of flight hours, with consequent longer time of exposure to noise, cause greater prevalence of postural alterations in the BP. It may be suggested that the drop in BP might be related to an alteration of the baroreceptor sensitivity (Chester & Barnett 1995) by chronic sympathetic activation with consequent dysregulation of the sympatho-vagal balance.

Chronic exposure to noise on the cardiovascular system may cause a high catecholamine secretion, with consequent catecholamine depletion and/or reduced catecholamine response, also due to β-receptor desensitization (Chester & Barnett 1995). Then, a reduction in myocardial adrenergic β-receptors is associated with reduced cardiac response to the

catecholamines (Fowler et al. 1986). Initially, the mechanism serves to protect against arrhythmias and toxic effects on the myocardium. In this study, the response of the sympathetic hypertonus to postural alterations, could be assumed to be precociously altered and the response of sympathetic hypertonus to workloads, is lower in the pilots of group A.

Conclusions

The different effects on the cardiovascular system might therefore depend on whether the clinical or experimental investigation is done (soon after the noise has started, after some length of exposure, or after a long exposure) and on related dysregulation of the sympathetic nervous system. In this research, pilots who had been exposed to noise for a long time were studied, so the effect might reflect catecholamine depletion and altered baroreceptor reflexes, probably no longer reversible.

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