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Hearing Impairment and Hypertension Associated with Long Term Occupational Exposure to Noise

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Abstract

Background: Noise induced hearing loss, a permanent bilateral hearing impairment due to long term exposure to high levels of noise, represents one of the most common occupational hazards. This historical cohort study was undertaken to evaluate the auditory effects of noise and to further examine the hypothesis that a link between noise exposure and hypertension exists.

Methods: The study population consisted of 140 healthy male employees from a local petrochemical industry with a history of past and present exposure to noise and 140 matched healthy unexposed individuals from the same industry (reference group). A questionnaire with 40 items concerning age, sex, weight, height, length of employment, workplace noise level, and history of hypertension was administered. Furthermore, the subjects were physically examined, their blood pressure was taken under normal resting position and all underwent audiometry testing. Similarly, sound pressure level and octave band analyzing in different stations of the workplace were carried out for every employee, and then Leq was calculated.

Results: The prevalence of hearing impairment in this study was 38.5% among the exposed and 7.8% among the unexposed group and the difference was statistically significant. Similarly, the prevalence of hypertension in the exposed group was significantly higher than that in the unexposed group.

Conclusions: These findings provide corroborative evidence to further substantiate the notion that exposure to noise is associated with hearing impairment. They also support the proposition that long term occupational exposure to noise appears to be a risk factor for arterial hypertension.

Keywords: Noise induced hearing loss; Occupational exposure; Hypertension

Introduction

Noise, probably, is the most common occupational and environmental hazard and the most common cause of hearing loss. Noise induced hearing loss is an irreversible hearing impairment resulting from long term exposure to levels of noise beyond 85 dB. About 10% of the American population suffered from a hearing loss that affected their ability to understand normal speech. Similarly, about 25% of the work force in the US (as many as 30 millions) is regularly exposed to potentially damaging noise.[1-3] Additionally, it has been estimated that more than half of industrial machines emit noise levels between 90 and 100 dB and approximately 50% of the industrial work environments in the US have noise levels between 85 and 95 dB.[4] While exposure of workers to noise represents one of the most prevalent risks of the industrialized societies, its importance varies depending on the type of production as well as duties involved.[5]

Some non-job related hobbies can also produce harmful noise. For instance, about 20% of the US population own firearms, and many use them without proper hearing protection. Other non-occupational sources of noise include chain saws and other power tools, loud music and motorcycles.[6] The economic costs of occupational hearing loss have been estimated to be billions of

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dollars.[7] In the United Kingdom (UK), it has been estimated that between 1 and 4% of the population are exposed to harmful or potentially harmful noise levels. Furthermore, 12% of adults suffer from sensorineural hearing impairment of which approximately 33% is age-related (presbycusis) and 5% is accounted for by noise. This would imply that around 0.6% of the adult population has noise-related sensorineural hearing impairment.[6] Although certain work duties in practically any industry can present a risk to hearing, in some industries such as petroleum, lumber and food processing, a greater proportion (up to 25%) of workers are exposed to noise levels beyond the Occupational Safety and Health Administration (OSHA) permissible exposure level of 90 dB and at risk of noise induced hearing loss.[8]

While noise, in high enough doses, produces permanent damage to the auditory system that can lead to significant hearing loss, it also produces stress and interferes with the ability to communicate.[9] The major risk factor for noise-induced hearing loss is long term, unprotected exposure to levels of noise beyond 85 dB.[8] Furthermore, in some studies noise has been implicated as a risk factor of arterial hypertension[8, 10-12] Although this has been difficult to quantify, it is a controversial issue and epidemiologic evidence is still limited.[13] For instance, while a positive association between long term exposure to noise and blood pressure levels has been reported by some investigators,[14,15] the scientific rigor of these studies has been questioned by others.[11] Since major risk factors of hypertension (potential confounders) such as obesity, alcohol intake, etc. were not considered and accounted for in the associations observed, the role of long term exposure to noise as a risk factor of hypertension remains to be convincingly demonstrated**.**

Recently, concern raised about the prevalence of hearing impairment among employees of a local petrochemical industry as a consequence of their exposure to noise prompted this investigation with two fold aims. First, there was an attempt to assess the extent to which these subjects were exposed to noise and to evaluate the degree to which, if any, noise exposure had resulted in hearing impairment. Secondly, this study aimed at further examining, in the absence of confounding variables, the hypothesis that there exists a link between noise exposure and hypertension.

Materials and Methods

Subjects

This historical cohort study was carried out in a local petrochemical plant consisting of 31 units of which 12 were identified as being noisy (SPL>85 dBA). From these noisy units, 5 were selected by multistage random sampling. Likewise, 140 out of 500 employees of these units were selected by the same method. Similarly, one hundred and forty unexposed employees at the same age level, serving as the referent group, were also selected from the units with very low levels of ambient noise (≤55dBA).

The sample size was calculated based on the prevalence of occupational noise induced hearing loss of at least 16% among noise exposed populations, as detailed in the discussion section, and a 95% confidence interval.

The subjects underwent physical examination and audiometry testing at the site (as part of their annual periodic examination) and their blood pressure was taken at normal resting position. Using WHO criteria, we defined hypertension as a systolic blood pressure (SBP) of 140 mmHg or greater and/or a diastolic blood pressure (DBP) of 90 mmHg or greater.[16] Furthermore, the subjects were interviewed and a questionnaire with 40 items was completed by them. This contained items concerning, age, sex, weight, height, eye color, qualitative estimation of dietary salt intake (low, normal, high), years of service, workplace noise levels, history of using ear protective devices as well as past and present complaint of tinnitus, vertigo, speech perception impairment, sleep disturbances and history of some diseases such as renal failure, thyroid, autoimmune and meniere's diseases, meningitis, encephalitis, syphilis, scarlet fever, diphtheria, and rubella. Similarly, the history of diabetes mellitus, hypertension, hyperlipoproteinemia, ischemic heart disease and family history of hypertension as well as present and past history of using ototoxic drugs such as streptomycin, vancomycin, aspirin and quinine and its derivatives were included.

Likewise, the questionnaire included items on the history of exposure to known ototoxic chemical agents such as carbon monoxide and solvents such as toluene, carbon disulfide, methanol, styrene, xylene, trichloroethylene, benzene and n-heptane.

To eliminate or minimize the effects of confounding variables, the employees with a history of exposure to non-occupational high noise levels as well as individuals with a family or personal history of hypertension or current use of ototoxic drugs were excluded.

Using a modular precision sound level meter (B&K / Type 2231/ Integrating SLM module BZ 7110/), an octave band analyzer (B&K / Type 1625 /Octave filter

set) of sound pressure level (dBA), and octave band analyzing in different stations of workplace carried out for every employee, separately related to his work time duty, and then Leq calculated.

The subjects of both groups underwent an audiometry test, using an audiometer device (Inter Acoustic/ Model AD 27). To effectively minimize the effects of noise-induced temporary threshold shift (NITTS), noise exposure was avoided forty eight hours prior to the audiometric testing.[17]

The data were statistically analyzed, using Student's t-test (or Welch's alternate t-test, when the standard deviation of the two comparable variables with the F test were significantly different) and Chi-square or Fisher exact test, Cochran-armitage test of trend as well as Mantel-Haenszel test of association. When the direction of an effect caused by an independent variable was not predictable, a statistical analysis was conducted, using a two-sided p-value. The experimental results are presented as arithmetic mean± standard deviations. Statistical tests were analyzed, using SPSS software (Version 10, Chicago, IL, USA).

Results

The subjects' physical characteristics as well as the noise levels to which they were exposed are presented in Table 1. As shown in the table, no significant difference was noted between the mean values of age, weight, height, and length of employment (Student's t test, p >0.05). Most of the subjects (92.1% and 93.6% for exposed and unexposed groups, respectively) were educated (holding high school or university degrees) and no statistically significant difference was noted between groups as far as the level of education was concerned.

Similarly, the dietary salt intake of a majority of subjects (97.1% and 97.8% for exposed and unexposed employees, respectively) was either low or normal. However, the number of exposed individuals claiming to use low sodium diet was significantly higher (36.4% vs. 12.8%) than that of their unexposed counterparts (Chi-square test, p<0.001).

The prevalence of hearing impairment among exposed and unexposed subjects was found to be 38.5% and 7.8%, respectively and the difference was statistically significant (Chi-square=37.046, p<0.001). Likewise, the prevalence of vertigo, tinnitus, sleep disturbances, disturbances in speech perception and recognition of words and voices was significantly higher in the exposed population.

Table 2 exhibits the distribution of smokers and nonsmokers among both groups.

As displayed, there exists no statistically significant difference between the number of smokers in both groups (Chi square test, p=0.260). The intensity of noise induced hearing loss was studied and the results are presented in Table 3. As shown, hearing loss was significantly more severe in the exposed subjects. Similarly, the distribution of hearing impairment by length of employment was studied and the results are exhibited in Table 4.

These data indicate that as the length of employment increases, the number of individuals with normal hearing decreases and the number of subjects

Table 1: Subject's physical characteristics, their duration of exposure to noise and the mean value of sound pressure level.

Parameter	Exposed(n=140)				Control(n=140)			
	Mean	SD	Min	Max	Mean	SD	Min	Max
Age (year)	41.2	7.4	24	57	41.5	7.6	24	57
Weight (kg)	75.5	10.8	45	115	76.1	11.1	55	112
Height (cm)	173.0	6.6	158	190	172.2	6.0	160	188
Length of employment (yrs).	17.4	8.0	1	32	15.8	8.1	1	34
Leq (dBA)	87.9	10.6	57	92.9	N/A	N/A	N/A	N/A

N/A, Not applicable

Table 2: Distribution of exposed and referent individuals by smoking habit

Parameter	Exposed (n=140)		Unexposed (n=140)	
	No.	%	No.	%
Smoker	26	18.6	22	15.7
Non smoker	114	81.4	118	84.3

Table 3: Mean value of hearing loss in different frequencies in dB

EAR	Exposed group					Unexposed group				
	500 Hz	1000 Hz	2000 Hz	4000 Hz	8000 Hz	500 Hz	1000 Hz	2000 Hz	4000 Hz	8000 Hz
Right ear mean(SD)	14.3* (5.7)	13.0* (8.1)	13.0* (9.2)	22.6* (16.1)	23.0* (18.9)	11.7 (4.8)	7.8 (4.9)	6.0 (6.1)	9.2 (8.5)	12.0 (9.3)
Left ear mean(SD)	14.9* (6.0)	13.2* (8.4)	14.7* (9.6)	23.0* (15.8)	23.6* (16.9)	11.1 (3.6)	7.7 (4.2)	5.5 (5.1)	10.4 (6.8)	12.0 (8.2)

*Significantly different from corresponding referent value (Student's t test, $p<0.001$).

Table 4: Frequency of hearing impairment among exposed subjects in relation to length of employment

Length of employment (yr)	Normal hearing		Hearing impairment	
	No.	%	No.	%
0-9	29	33.7	2	3.7
10-19	35	40.7	21	38.9
>20	22	25.6	31	57.4
Total	86	100	54	100

with hearing impairment significantly increases (Chi-square test for trend, $p<0.001$). The prevalence of hypertension was studied between both groups and the data are presented in Table 5.

Table 5: Distribution of hypertensive and normotensive subjects between exposed and unexposed individuals.

Subjects	Exposed group		Unexposed group	
	No.	%	No.	%
Hypertensive	28	20	18	12.8
Normotensive	112	80	122	87.2
Total	140	100	140	100

The number of hypertensive subjects among the exposed individuals was significantly higher than its corresponding value in the unexposed group (One side Chi-square test, $p=0.05$, relative risk, 1.6).

To assess the effect of age on hypertension, the distribution of hypertensive subjects of both groups was studied in individuals younger and older than 45 years (Table 6).

As shown, the number of hypertensive subjects among exposed individuals younger than 45 was significantly higher than that in the unexposed group. Conversely, most of the hypertensive subjects in the unexposed group were older than 45 (Chi-square test, $p=0.008$, relative risk 2.44). After adjusting for age, using Mantel-Haenszel test of association (Table 7), the positive relationship between noise exposure and hypertension was still significant ($\chi^2_{MH}=4.39$, $p=0.04$).

Table 6: Distribution of hypertensive subjects among both groups by age

Subjects	<45 years old Hypertensive		>45 years old Hypertensive		Total	
	No.	%	No.	%	No.	%
exposed	19	67.8	9	32.2	28	100
unexposed	5	28.0	13	72.0	18	100

Table 7: Distribution of hypertensive and normotensive subjects adjusting for age

Blood pressure	<45 years old			>45 years old		
	Exposed	Unexposed	Total	Exposed	Unexposed	Total
Hypertensive	19	5	24	9	13	22
Normotensive	13	37	50	99	85	184
Total	32	42	74	108	98	206

Discussion

There should be very few, if any, socioeconomic and demographic differences between exposed and referent subjects as they were from the same industry with almost identical level of education, sex, ethnic background, age, weight, height and length of employment.

Additionally, both groups were similar as far as the number of smokers was concerned; they were non-alcoholic and no excessive intake of dietary sodium was recorded for them. Therefore, as there were no significant differences in the major confounding variables of age, sex, weight, cigarette smoking and other socioeconomic and ethnic factors, an increased prevalence of hearing impairment and symptoms such as vertigo and tinnitus as well as hypertension among exposed subjects are likely to be the direct result of, and may well be explained by, exposure to noise.

Given the intensity of noise and length of exposure, the prevalence of hearing loss has been reported to be 16 to 73% among noise-exposed population.[18-27] Consistent with these reports, in our study, the prevalence of occupational noise-induced hearing loss among the exposed subjects was found to be 38.5%.

The prevalence of hypertension among subjects exposed to noise was significantly higher than that among unexposed employees. In fact, it was found that the risk of contracting hypertension in noise-exposed individuals was 1.6 folds higher when compared to their unexposed counterparts (Table 5).

The data presented in Table 6 demonstrate that age is unlikely to be a contributing factor in the increased prevalence of hypertension observed in the exposed individuals. In fact, if age had a part in this phenomenon, one would expect that a majority of hypertensive subjects to be very old or at least older than their unexposed counterparts. However, this was not found to be the case and the opposite was true. Table 6 shows that most (67.8%) of the noise exposed hypertensive subjects were younger than 45 years. In contrast, most of (72%) the unexposed hypertensive individuals were older than 45 years. This implies that among the exposed group noise is likely to be causally linked with hypertension. Conversely, in the unexposed group, hypertension is probably age related. While being consistent with a previous study by Fogari R, et al,[28] this finding is further supported by the data presented in Table 7, showing that after adjusting for age, using Mantel-Haenszel test of association, the positive relationship between noise exposure and hypertension was still significant (χ^2_{MH}=4.39, p=0.04).

Similarly, dietary sodium intake is also unlikely to play a part in the significantly increased prevalence of hypertensive subjects among the exposed employees. Three lines of evidence support this proposition.

First, sodium intake of a majority of (92.1%) the exposed individuals was either low or in the normal range. Second, only a small proportion (7.9%) of the exposed population claimed to have excessive (high dietary) intake of sodium and this was not significantly different from the corresponding value, 6.4%, in the unexposed population.

Third, if sodium had a part in hypertension, its effect would be expected to be observed in the referent subjects because the number of the exposed individuals using low sodium diet was significantly higher (51 vs. 18) than that of their unexposed counterparts (Chi-square test, p<0.0001). Likewise, the data presented in Tables 1 and 2 indicate that weight and cigarette smoking could be ruled out as confounding variables in the observed association between noise exposure and hypertension.

The exact mechanism(s) by which noise induces hypertension is not clear. However, in some studies higher levels of circulating catecholamines and higher urinary excretion of these vasoconstrictors during working hours have been shown in 60 subjects exposed to noise ≥90 dB as compared to 60 control subjects. This has been implicated as the biological pathway by which noise may exert its effect on blood pressure.[12] While this appears to be a plausible explanation, it can only demonstrate the acute effects of noise.[11]

The findings of this study collectively indicate that exposure to high levels of noise significantly increases the prevalence of hearing impairment (sensorineural hearing loss). Additionally, symptoms such as tinnitus, vertigo, sleep and speech perception disturbances and difficulty in recognition of the words and voices are associated with hearing loss and are much more prevalent among noise exposed subjects than in their unexposed counterparts.

Similarly, long term exposure to noise might be considered as a risk factor of arterial hypertension, although additional studies with larger sample sizes and more sufficient follow up on employed populations exposed to more intense noise are clearly required to conclusively demonstrate the presence or lack of a causal relationship between noise exposure and hypertension.

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