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Case Series

Noise induced hearing loss in low frequencies in employees in a hospital microbiology department

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ABSTRACT

Noise induced hearing loss (NIHL) is regarded as a serious problem and one of the most recorded occupational disorders in Europe and in the rest of the world and amounts to between 7% and 21% of the hearing loss. Aim of this study is to explore the development and the prevalence of low frequency noise-induced hearing loss (NIHL) in a hospital, especially in microbiology laboratory workers. Generally it is known that 4 KHz is the main NIHL frequency. Despite current theories, our study suggests for the first time the impact of low frequency noise in hearing loss among laboratory workers. According to the results, the population examined, namely the employees at the Microbiology Department of the Hospital, showed lower hearing levels compared to the control group, who had no history of occupational exposure to noise. There are many other studies which suggest that prolonged exposures to high noise levels have negative physiological and psychological effects on workers. The finding of the correlation of noise frequency with the frequency of the generated hearing loss is involved in the controversy about the pathophysiology of noise effect.

Keywords: Noise induced hearing loss, Low frequencies, Laboratory employees, Cochlear hair cell damage, Physiological and psychological effects

INTRODUCTION

Noise induced hearing loss (NIHL) is regarded as a serious problem and one of the most recorded occupational disorders in Europe and in the rest of the world and amounts to between 7% and 21% of the hearing loss.[1,2] It is remarkable that occupational noise-induced hearing loss refers to men actually at percentages rising to 94%.[3] Over 10% of adults suffer from hearing damage of some kind; a fact that renders the matter a major problem for public health.[4]

Studies on hearing in various professions suggest that hearing loss due to workplace noise was a significant problem in the 1960s and 1970s in industrialized countries, whereas hearing loss has been a less frequent problem in subsequent decades. The reduced incidence of hearing loss is probably a result of decreased noise exposure, improved regulation and use of protective equipment, but the evidence for this is still limited.[5] This positive trend does not apply to developing countries, where exposure to high levels of noise at work is still significant. Common occupations that are well studied, for example groups of higher risk of NIHL are found in the armed forces, the engineering industry, building and construction, and agriculture.[3] There is strong relationship between occupation and hearing loss. Male construction workers may be one of the groups with the most pronounced NIHL with an average hearing loss of 9 Db in the 3-6 kHz frequency range and 6 dB for the average of 0,5, 1,2 and 4 Hz , compared to non-noise-exposed male teachers 6. Employees who seem to have little or no risk of harmful noise exposure at work are people employed in school, day care, transportation, musicians, police, kindergarten

employees and firefighters.[3] According to Martin et al (1975) the prevalence of a hearing loss defined as the average >25 db for the 0,5-2 KHz area ranged from 14-32% in exposed >50 years among smell workers compared to 4% of the control group which was unexposed employees for the same factory.[7] Furthermore continous noise and impulse noise can damage hearing if exposure is high emough. Impulse noise is probably more harmful than continous noise at the same level of noise exposure of 85-90 db. The reason for this according to Clifford and Rogers (2009) is an overload of both the hair cells and the cellular antioxidant system at high exposure levels. Higher exposure levels may also produce a mechanical damage in the cochlear.[8]

According to Glonig et al provided that at a frequency of 4000 Hz hearing corresponds to normal levels, it is almost certain that the rest of the hearing function is normal, actually at percentages rising to 98%. On the other hand, Schneider et al found that the frequency most susceptible to hearing damage is not 4000, but 6000 Hz.

Occupational risks at hospitals concern both accidents and occupational diseases related to exposure to risk factors.

In the USA, the incidence of accidents per 1000 full-time employees corresponds to 65-80 cases per year, 20-30% of which resulted in absence from work for at least three days; a percentage so high that it reached the first place within the services sector. In the USA, the EU and Australia the occupational diseases incidence index in hospitals is the highest one in the services sector, counting 50 new cases of occupational diseases per 10000 employees every year.[9] The most common occupational diseases at hospitals are musculoskeletal diseases, contact dermatitis, pulmonary diseases, infections, anxiety-burnout syndrome, sleeping disorders and cardiovascular diseases. The risk factors are ergonomic: patient handling-posture and standing work-repetitive movements, biological: Microbes, chemical: carcinogenic chemical substances, organizational: rolling work hours, on-call service, psychosocial relations with patients and the public, cooperation.

Despite the fact that numerous entities have been identified as occupational problems in hospitals, there is lack of studies for occupational NIHL in hospital workers. There is one study that correlates anesthetic gas with hearing loss. Carbon disulfide is a colorless, flammable, poisonous liquid, CS2. It is used as a solvent, and is a counterirritant and has local anesthetic properties. Carbon disulfide was shown to reduce hearing in the low frequencies in a study of 346 rayon wool workers, where 105 were exposed to equivalent noise levels of 80–90 dB, 132 were exposed to a combination of noise and carbon disulfide, and the rest were not exposed. The hearing loss was about 10 dB in both exposed groups compared to the nonexposed.[10] Furthermore there is a recent exploratory study of noise exposure in educational and private dental clinics. There are potential effects of dental noise on dental students' and staffs hearing, particularly in participants in pediatric clinics.[11]

Our study suggests for the first time the impact of low frequency noise in hearing loss among laboratory workers.

Aim of this study is to explore the development and the prevalence of low frequency noise-induced hearing loss (NIHL) in hospital, especially in microbiology laboratory workers.

CASE SERIES

This is a case control study included all employees at the microbiology laboratory of Ippokrateio General Hospital.

The study took place in the Hippocratio General Hospital and concerned the exposure to noise of all of the employees in the Microbiology Department (48 persons), the place where there was undoubtedly the greatest likelihood of a noise effect on the employees.

There was a control group of 30 control subjects consisting of employees in other departments of the hospital with similar sex and age ratios, in order to minimize the effect of the confounding factors. The noise recorded ranged between 82 and 87 dB. The work areas corresponding to the specialized laboratories: biochemistry, immunology, hormonology, blood donation-crossmatching, cultures, coagulations.

We compared the employees at the microbiology laboratory of Ippokrateio General Hospital (group one) with a control group (control group two). Inclusion criteria were exposure to occupational noise alone, hearing loss and the statistical association between occupational noise and hearing loss.

The exclusion criteria were set in order to eliminate the potential effect of possible confusing factors such as age, noise exposure outside professional life, diseases related to the occurrence of hearing loss, and otologic history. In order for the data to be collected, a questionnaire was compiled, and filled out following a personal interview with each of the study participants. The questionnaire included questions regarding the following matters: Age, general state of health-personal anamnesis, subjective feeling of hearing loss, history of vertigo-tinnitus, otologic history, use of ototoxic factors, hereditary history of hearing loss, job position, years of previous experience, probable previous experience at another job position, place of residence, presence of noise at residence or in activities during leisure time, sensitization around noise issues, use of protective means against noise. An ENT examination followed, for the exclusion of mechanical (earwax blockage, serous otitis media, foreign matter) or other factors causing hearing loss. Then an audiogram was conducted, all by the same examiner and using the same audiographer. This choice aimed at eliminating possible systemic errors and measurement errors.

A system featuring an auditory barrier-special acoustic chamber was used to place the person under examination for the purpose of total acoustic isolation from his/her environment. Noise dosimeters are required to comply with the American National Standards Institute (ANSI) Specification for Personal Noise Dosimeters S1.25–1991 (R1997), which states that dosimeters should be suitable for measurement of impulsive, intermittent, and continuous noise. The Occupational Safety and Health Administration (OSHA) and the National Institute of Occupational Safety and Health (NIOSH) proposed directions which state that no unprotected noise exposure exceeding 140 decibels (DB SPL) may be permitted under any circumstances.

On the basis of the exclusion criteria, 4 patients were excluded from the study. Two of them had a known otologic history; the first patient because she reported being exposed to noise at her residence due to proximity to the central avenue, and the second patient because she was diagnosed with otosclerosis during the test. The employees who met the final criteria were 48 in number. 43 of them were female and 5 male. The average age was 39.58 years (standard error 1.28). The average years of previous experience was 14.2 (standard error 1.36). We compared the employees (group one) with a control group (control group two). Statistically, there is no considerable difference in age distribution between the two groups (P=0.2020). Firstly, the results of our study indicate a considerable difference, from a statistical point of view, in terms of the threshold of hearing at 250 and 500 Hz between the employees and the control group both for the right and the left ears, while no considerable difference, from a statistical point of view, was observed in terms of the threshold ranging between 1000-8000 Hz. Secondly, there is no considerable difference, from a statistical point of view, in terms of the threshold of hearing at 250 Hz and 500 Hz when comparing the right and left ear of each employee. Thirdly, there is a considerable difference, from a statistical point of view, in terms of the threshold of hearing at 250 and 500 Hz for both ears among employees having previous experience above or below 10 years. Fourthly, there is a considerable difference, from a statistical point of view, in terms of the threshold of hearing in all frequencies among employees above or below 40 years of age.

Table 1: A detailed table with the above mentioned results follows.

Frequencies (Hz) right ear and/or left ear	Group One Mean threshold of hearing (dBL)	Control Group mean threshold of hearing (dBL)	P value
250 Hz right	20.42	8.95	<0,0001
250 Hz left	10.53	19.90	<0,0001
500 Hz right	19.38	8.68	<0,0001
500 Hz left	10	16.77	<0,0002
1000-8000 Hz right	10.52	9.47	<0.1931
1000-8000 Hz left	10.99	9.51	<0.0595
All frequencies (Hz) right between >40 years old years and <40 years old	24.20	16.25	<0.0001
All frequencies (Hz) left between employees >40 years old and <40 years old	9.68	15.25	<0.0001
250 Hz and 5t00 Hz right and left between emploeers with previous experience ≥10 years and <10 years	14.88	23.48	<0.0001
250 Hz between right and left	20.42	19.90	<0.3412
500 Hz between right and left	19.38	18.13	<0.0898

DISCUSSION

According to our study's results, the population examined, namely the employees at the Microbiology Department of the Hospital, showed lower hearing levels compared to the control group, who had no history of occupational exposure to noise. One of the major issues of the study was the exclusion of those persons whose history included states and diseases that might cause hearing loss. That was achieved through the use of a standardized questionnaire used for patients admitted to the ENT clinic, as well as through a physical examination.

Generally it is known that 4 KHz is the main NIHL frequency.[12] This is the frequency that is considered susceptible to the effect of noise, and which theoretically is impaired first during the chronic process of noise effect on hearing is that of 4KHz. Actually, there are studies claiming that normal hearing at this frequency proves the absence of sensorineural hearing loss in the rest of the frequencies too, at a percentage approximating 98%.[13] The reason behind the fact that the frequency of 4KHz constitutes the most susceptible target for the effect on hearing is subject to analysis and various theories. It is generally acknowledged, as also mentioned in the chapter regarding the effect of noise on hearing, that the chronic

oscillation of the inner hair cells causes strain through various mechanisms that appear to act simultaneously and cumulatively, such as anatomic strain, malfunction of the stria vascularis and potassium ions, as well as disorder in terms of the function of neurotransmitters. It is also acknowledged that the areas of the cochlea feature a distinct natural frequency that allows them to become activated on a case-by-case basis depending on the frequency of the auditory stimulus treaching the cochlea. These two factors, the chronic nature of the effect and the natural frequency, constitute tools for explaining the clinical finding with regard to the onset of the effect of noise at the frequency of 4 KHz. Among the probable patterns proposed are, on the one hand, the fact that 4KHz correspond to the natural frequency of the anatomical structure of the cochlea, and on the other hand, the fact that the anatomical structure of the cochlea at the level of basic membrane thickness and, by extension, resistance is the one corresponding to 4 KHz.

Our findings suggest NIHL in low frequencies.

This study, apart from first suggesting that noise may qualify as an occupational risk employees at Microbiology Departments, also presents one further piece of evidence in the attempt to understand and explain the effect of noise on hearing as a whole, at a pathophysiological level.

There is an evidence that noise may damage the lower and medium frequencies as well as the higher frequencies. 200 of 1000 patients with noise-induced hearing loss showed loss of hearing at 250 Hz and 500 Hz of 20 dB or more 14.

It is known the effect of low frequency noise to hearing levels. Intense, LF sound causes cyclic changes of indicators of inner ear function after LF stimulus offset.

Drexl et al showed that the mechanical amplification of hair cells (OHCs) is significantly affected after the presentation of LF sound.[15] They concluded that a temporary disturbance of OHC calcium homeostasis caused activity changes of outer hair cells and furthermore low frequency induced hearing loss.

For decades LF sound, i.e sound with frequencies lower than 250 Hz has been considered to bypass the inner ear because human hearing thresholds for frequencies below 250 Hz are high.

Wiegrebe et al also concluded that LF sounds, in contrary to current beliefs, strongly stimulate the human cochlea and affect active micromechanics in the human inner ear.[16] LF sounds significantly affects outer hair cells: a 90 s, 80 dB LF sound induced slow, concordant and positively correlated frequency and level oscillations of spontaneous otoacoustic emissions that lasted for about 2 min after LF sound offset.

Normally a short LF sound exposure of just 90s can elicite cochlear responses that the recovery process significantly exceeds the exposure duration. The higher sensitivity of human for LF sounds is true despite the fact that the LF sound has a sensation level of only about 60 dB and is not perceived as uncomfortably loud .It is known that in low frequency sound hearing in most mammals is poor or non-existent.

Kemp and Kevanishvili et al recorded click-evoked OAEs after exposure to LF sound with up to 105 dB and also found level changes in the cochlear status and as a consequence in human hearing ,although not exceeding 1–2 dB, with a time course very similar to previous studies.[17,18]

Bian and Watt and Bian analysed SOAEs in humans during exposure to LF sounds with maximum levels of 50 dB (A), i.e. at least 30 dB fainter than in the current study. Coupled to the phase of the LF tone, the SOAEs showed a periodic level decrease and frequency increase.[19,20]

Thus the exposure to lower frequency sound (i.e. less than 500 Hz) causes slow changes of OHC mechanical properties and gain of the cochlear amplifier. Both could explain the level alterations of SOAE we observed. Moreover Brief exposures to LF tones have also been shown to induce endolymphatic hydrops, possibly altering cochlear mechanics to an extent which can cause, or contribute to, the SOAE changes we observed.[21] It is likely that both of them (endolymphatic volume changes and SOAE changes) share the same origin and are a result of LF-induced changes of cochlear homeostasis.

Patuzzi suggested that LF tones induce changes in the Ca2+ homeostasis of OHCs.[22] He argued that only stimulation with LF sound can produce receptor potentials large enough to depolarize the OHC to such an extent that voltage-gated Ca2+ channels at the base of the OHCs are opened, triggering Ca2+-induced Ca2+-release and -uptake, which can become unstable and, as a consequence, cause oscillation of Ca2+ levels.[23]

The result of the noise effect on hearing includes both temporary and permanent threshold shift (NITTS-noise induced temporary threshold shift and NIPTS-Noise induced permanent threshold shift respectively) and the acoustic trauma. The prevailing theory for the pathophysiology of the sound effect on hearing involves two phases: the static and the dynamic. The dynamic begins during the auditory stimulation and results in anatomical and functional changes in the hair cells of the organ of Corti, which may be permanent or temporary. It is accepted that there is a marginal exposure limit to noise. Below this level, there is a biochemical and perhaps reversible deterioration of the cochlea, while above that level there is considerable mechanical and irreparable damage. After the acoustic stimulus, the ear structure can be either completely restored, whereby the condition is called temporary threshold shift (NITTS-noise induced temporary threshold shift) or partially restored. If the

restoration is incomplete or completely absent, then the situation enters a stationary phase in which the anatomical and by extension the acoustic-physiological damages are irreversible. A feature of NITTS is that after the cessation of the noise effect, hearing returns to the previous levels within a few minutes or within several weeks the most. NITTS is caused by noise >80 dB. Above these limits, the size of NITTS increases with the intensity and time of exposure to noise, even though there is evidence that the exposure above 8 to 16 hours does not further increase the size of NITTS. Moreover, the intermittent noise causes less NITTS compared to the constant noise with the same energy. According to the classic distinction, damages are distinguished into temporary, permanent, degenerative and reparable. However, due to the nature of the organ anatomy, it is very difficult to study in vivo the pathophysiological mechanisms. There is no imaging method of the cochlea, or a measurable indicator of the bloodstream associated with either the functional state of the cochlea or the patient's level of hearing. Besides these, there are numerous confounding factors. Significant factors are the age and the consequent presbycusis, whose degree varies depending on diathesis and heredity. NIPTS typically occurs to employees who work under continuous or repeated strong noise for a long time. It is supposedly and arbitrarily believed by some researchers that NIPTS is the result of continuous successive NITTS. NIPTS is the result of the noise intensity levels and the exposure time. The typical appearance of NIPTS shows maximum loss at the level of 6-8 KHz frequencies, with a slower fall in adjacent frequencies. The loss is faster during the first 10-15 years of exposure while consequently the rate slows down. There seems to be considerable variation in the employees' sensitivity or predisposition to develop NIPTS. The term acoustic trauma is limited to the effect of a single or relatively few exposures to noise levels >160 dB and the damage caused is permanent. Series of studies have shown correlation between hearing loss and thinning of the hair cells, disruption of the stria vascularis structure, overexpression of proteins secreted by the calcium pumps, detection of GABA protein receptors (neurotransmitter). The glutamine secretion (basic neurotransmitter) has been proposed as the promoting mechanism of hearing loss caused by noise, due to the subsequent loading of calcium ions and the entering of chlorine ions along with water into the cells. This results in the discharge and the disabling of the synaptic membrane of the nerve cells. It is also known that NO synthase has been detected in the inner ear, while there are indications that it is related to ototoxicity. In a study in 1997, Rous et al. demonstrated that the NO synthase, a metabolite, damages both the outer and the inner hair cells when injected into the round window. Its role in the pathophysiology of the noise effect on hearing is therefore being speculated.

A systemic review induced includes all the workers who seems to have noise-induced hearing loss. Occupational noise exposure causes between 7-21% of the hearing loss among workers highest in the developing countries and it is age-related. Impulse noise is more deleterious to hearing than continuous noise. Occupational groups at high risk of NIHL are the military, construction workers, agriculture and others with high noise exposure such as industrial workers, offshore workers (oil and gas production at sea), professional divers, steel, cotton, pulp, metal, aluminum workers, fire fighters, shipyard workers, railway workers, farmers, musicians and kindergarten employees.[3]

Furthermore, most research findings show that shift – and night work are associated with cardiovascular, musculoskeletal and neurological disorders as well as work-related injuries among health care workers.[24,25] Among the most frequent viral occupational infections are those transmitted by blood such as HBV, HCV and HIV. The majority of occupational exposures were reported by nurses and medical technicians. The most common type of exposure is the needlestick injury occurring during the blood sampling and during surgical procedure.[26] Analysis of data of an epidemiological study of occupational diseases in Italy and Argentina revealed a relationship between nationality, exposure to occupational risks and the prevalence and incidence of selected pathologies.[27]

The limitations of this study include the relatively small number of participants, given that in order for noise to be established as an occupational health hazard for other professions, the studies submitted included hundreds or even thousands of patients; the inability to fully eliminate the effect of confusing factors such as the effect of noise outside the hours and place of work, as well as the lack of records on individual sound exposure of employees due to the inability to obtain the required equipment.

In conclusion there are many studies which suggest that prolonged exposures to high noise levels have negative physiological and psychological effects on workers.[28]

CONCLUSION

Occupational hearing loss is a common condition that involves large and heterogeneous groups of employees with a very broad scope for prevention. This was the first recording of occupational hearing loss in employees in a Hospital Microbiology Department and particularly after exposure to low frequency noise despite the current theories. The finding of the correlation of noise frequency with the frequency of the generated hearing loss is involved in the controversy about the pathophysiology of noise effect.

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