HEARING LOSS IN WORKERS EXPOSED TO DIFFERENT TYPE OF NOISE

Ewa ZAMYSŁOWSKA–SZMYTKE, Piotr KOTYŁO, Marek BĄK, Adrian FUENTE, Mariola ŚLIWIŃSKA–KOWALSKA

Nofer Institute of Occupational Medicine Department of Physical Hazards Św. Teresy 8, 91-348 Łódź, Poland e-mail: zamysewa@imp.lodz.pl

(received March 21, 2007; accepted April 18, 2007)

It's well documented that noise damages peripheral part of the auditory tract (cochlea). Only few investigations were performed to assess central hearing disturbances caused by noise on animal.

The aim of the study was to evaluate the site of hearing damage in 2 groups of individuals exposed to different types of noise. First group consisted of 62 dockyard workers exposed to impulsive noise with co-exposure to hand-arm vibration while second group included 76 bottle glass factory workers exposed to continuous steady-state noise. Results were referred to 86 control subjects exposed neither to noise nor vibrations.

Pure-tone audiometry, immitance audiometry, Auditory Brainstem Response (ABR) and cognitive-event relate auditory evoked potentials (wave P-300) were performed in all subjects.

Audiometric results revealed the poorest hearing level in dockyard workers among all groups. The bottle factory workers had also significant hearing impairment at high frequencies as compared to controls. Although the wave V at the ABR was prolonged in the dockyard workers this change could reflect sensorineural hearing thresholds shift and retrocochlear damage. The latency of P-300 wave was prolonged in dockyard workers exclusively suggesting a cortical effect of exposure to impulsive noise.

Conclusions. Exposure to high level impulsive noise in combination with hand-arm vibration may cause hearing deficit greater than expected. Abnormalities involve peripheral and central auditory system. More observations are necessary to confirm these findings.

Keywords: pure-tone audiometry, immitance audiometry, ABR, cognitive-event.

1. Introduction

Noise-induced hearing loss is well known as sensorineural hearing deficit that begins at the higher frequencies and develops gradually as a result of chronic exposure to excessive sound level. There is very few papers on central damage caused by noise exposure. On the other hand noise is known to activate the pituitaryadrenal-cortical axis and the sympathetic-adrenal-medullary axis [3]. Changes in stress hormones including epinephrine, norepinephrine and cortisol are frequently found in acute and chronic noise experiments. Chronic stress impairs synaptic plasticity in the hippocampal area; in addition, it induces selective atrophy within this region and severely disrupts working memory and behavioral flexibility [4]. Moreover, studies performed on the groups of workers employed in the aeronautical industry revealed morphological vascular changes of the central nervous system and cognitive impairment (P300 latency significantly longer) in subjects exposed to low frequency noise [5].

Central hearing loss is generally thought to be extremely rare compared to the sensorineural or conductive types of hearing loss, but recent studies have shown that it is much more common than previously appreciated. The diagnosis is usually difficult since pure tone audiogram often remains normal. Patients usually exhibit poor scores on speech reception threshold or word recognition scores. Also, the P300 wave shows the modifications in neuronal activity which takes place during the cognitive process. The P300 is a positive wave which arises when an attended stimulus is detected. Its parameters depend on a number of variables, in particular the subject's mental state, the task that has to be accomplished, the significance of the stimulus, and the degree of attention. P300 responses get strong contribution from higher-order structures in the central nervous system (hippocampus, amygdala, limbic system, thalamus, frontal/temporal lobes). The sensitivity of P300 in detecting lesions of central auditory nervous system has been reported to be about 80% [1]. Latency of P300 can be used as measure of speed of information processing in the central nervous system.

The aim of the study was to assess the site of hearing damage and the cognitive function in workers exposed to the noise of different intensity and character and in two different working conditions.

2. Material and methods

Two groups of workers exposed to noise were examined: 63 dockyard workers; and 76 bottle and glass factory workers. The reference group consisted of 83 office and maintenance workers with no exposure to noise above admissible level of 85 dBA. Mean age doesn't differ between study groups (p < 0.05) as described in Table 1.

	N	Mean	Std. Deviation	Minimum	Maximum
Dockyard workers	62	41.5484	9.91055	23.00	57.00
Bottle factory workers	76	38.9605	7.95394	23.00	55.00
Control	83	39.6024	8.22539	23.00	56.00
Total	221	39.9276	8.66730	23.00	57.00

Table 1. Groups by age.

The individual working life exposures to noise were evaluated on the basis of collected subjects' work histories and exposure data in different working conditions. The total lifetime equivalent noise level (dose) was higher in dockyard workers (mean exposure level 138.7 \pm 4.6 dBA) than in bottle factory workers group (mean 136.1 \pm 4.9 dBA). The average exposure level was also higher in this group (92.2 \pm 3.3 dBA vs. 89.6 \pm 4.5 dBA), the differences were statistically significant (p < 0.05). Moreover, the work characteristics was different in the groups. The most of shipyard workers reported a least short-time everyday or sporadic exposure to hand-arm vibration at daily exposure value normalized to 8-hour reference period A(8), ranged from 0.4 to 4.1 m/s² (POEL = 2.8 m/s²). They were also exposed to impulse noise working as assemblers, grinders, welders etc. On the other hand, glass factory workers were neither exposed to impulse noise nor hand-arm vibration.

Medical inclusion criteria were as following: time of employment in exposure > 6 months, normal otoscopy, tympanogram type A, no history of ear diseases and ear surgery or severe head trauma in anamnesis. During medical examination the questionnaire was filled. The questionnaire included detailed inquiries on present and previous employment exposure to noise, medical history, physical features, life-style, military service, and exposure to ototoxic factors beyond occupational environment.

Audiometric tests

Hearing examination was performed at least 16 hours after the last exposure to noise in the sound proof booth meeting the requirements of ISO 6189:1983.

The following tests were included:

- Standard pure-tone audiometry (air conduction 1–16 kHz, bone conduction 1– 4 kHz), with the clinical audiometer AC-40 model from Interacoustics Co.
- Immitance audiometry (tympanometry, ipsi- and contralateral stapedial reflex) with Zodiac 901 model from Madsen. Tympanometry used single frequency with a probe signal of 85 dBSPL continuous tone at 226 Hz.
- auditory brainstem evoked responses (ABR) was registered with Nicolet Spirit 2000 & Spirit 2000 Lite model from Nicolet Biomedical. A 100 μ s, 90 dB HL click stimulus was presented at the rate of 20 Hz. The analyzed parameters were the latencies of the waves I, III and V.
- A cognitive event related potential (wave P-300) was recorded using the same equipment and the same electrode configuration as with the ABR for ease of recording. The stimulus oddball paradigm was used with target frequency of 500 Hz and non-target more frequent stimuli of 1 kHz (mode A); and with target frequency of 1500 Hz and non-target more frequent stimuli of 1 kHz (mode B); One hundred stimuli were presented once every second in a random sequence of target and non-targets (target/non-target ratio 30/70) at a comfortable listening level of 70 dB HL.
- Interrupted speech audiometry sentences presented at listening level 60 dB over hearing thresholds.

3. Results

Hearing thresholds were significantly worse in dockyard workers as compared to the bottle factory workers and control in whole range of frequency tested (Fig. 1). In bottle workers group mean values of hearing thresholds were higher than in control at frequencies 4 and 6 kHz in a right ear and 4, 6, 8 kHz in a left ear.

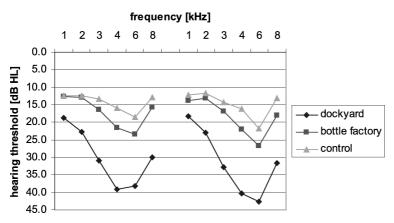


Fig. 1. Pure tone audiometry (PTA) thresholds in study groups.

Auditory Brainstem Response (ABR) results revealed longer latencies for all waves in dockyards workers group but the significant differences were seen only for wave V (right and left ear). Although one-way anova test showed significant difference among three groups for wave III (left ear), no significant differences were seen in Tukey's post hoc analysis between groups (Fig. 2).

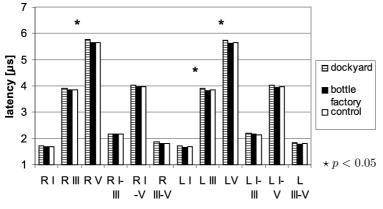


Fig. 2. ABR latencies of waves I, III and V and interpeak duration.

These data to the some degree was confirmed by higher percentage of subjects in dockyard group with absence of contralateral reflexes when ipsilateral reflexes were

present (27%) as compare to bottle factory workers (8%) and control group (14%), which may be the sign of possible abnormalities at the brainstem level.

One way anova showed significant differences among the three groups in P300 test. Tukey's post hoc analysis confirmed differences in latency between dockyards and control in both types of mode used (Fig. 3a, b). There were no statistical differences between the latency in dockyards workers and bottle factory workers or bottle factory workers and control group.

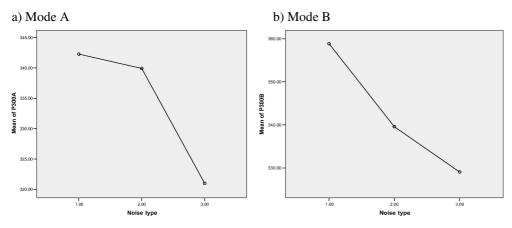


Fig. 3. P300 latencies in test using two modes of stimuli application. Groups 1 – dockyards; 2 – bottle factory workers; 3 – control. Mode A target/non target 500/1000 Hz and mode B 1500/1000 Hz.

The percentage of subjects with abnormal interrupted speech test results was the highest in dockyard workers (33 subjects, 62%) when compared to bottle glass factory workers (20 subjects, 26%) and control (15 subjects, 18%).

4. Discussion

This study shows that hearing thresholds in dockyard workers are signifficantly worse than in bottle factory workers group. The difference in thresholds approximately reaches 18 dB at 4 and 6 kHz, and arises mostly probably from different exposures. The former group was exposed to noise of higher intensity calculated both as a dose and average value than the latter one (92.2 vs. 89.6). Moreover the type of exposures was different, as dockyards were working with the pneumatic tools generating noise impulses and hand-arm vibration while the bottle factory workers were exposed to steady-state noise only. Impulse noise causes evidently more severe hearing loss than steady state noise. Even through the correction factor of 5–12 dB HL at 4 kHz [5] was implied for impulsiveness of noise, still hearing loss in dockyard workers was more pronounced than in steady-state noise exposed group.

ABR abnormalities shown in this study may correspond to elevated hearing thresholds. It is known that the latencies of the ABR waves depend on the severity of sensorineural hearing loss [7], what could be the cause in our investigation. On the other hand the adjustment for hearing loss is recommended when hearing impairment exceeds 50 dB HL at range 2–4 kHz. However that condition is rare in our study individuals. We cannot exclude that longer latency of V wave may be the sign of retrocochlear damage. It's also supported by the stapedial reflexes absence in several workers.

P300 is related to cognitive and neuropsychological brain function that is relatively independent of educational influences and it seems to be independent to hearing thresholds variations as the stimulus is well perceived [9]. Both types of measurement in our study (mode A and B) revealed significantly longer latency in dockyard workers is than in control. The differences between two noise groups were more marked when the mode B was used, but still insignificant. From the results of this study it seems that noise influence the cognitive function and the central auditory processing. The coexistence of several disadvantageous factors (impulse noise, high level of noise exposure and hand arm vibration) in dockyard workers may be particularly harmful. More observations are necessary to confirm these findings.

Acknowledgments

This study was supported by EU within the 6-th European Framework Project under the Marie Curie Host Fellowship for the Transfer of Knowledge "NoiseHear" (Contract No MTKD-CT-2004-003137).

References

- [1] MUSIEK F.E., BARAN J.A., PINOHEIRO M.I., P300 results in patients with lesion of the auditory areas of the cerebrum, JAAA, 3, 5–15 (1992).
- [2] BABISCH W., *The noise/stress concept, risk assessment and research needs*, Noise and Health, **4**, 16, 1–11 (2002).
- [3] OLESHKEVICH L. A., SHABUNINA N. D., Effect of noise of moderate intensity on the functional status of the sympathetic-adrenal system, Gig Sanit., 12, 93–95. (1989).
- [4] CERQUEIRA J. J. et al., The prefrontal cortex as a key target of the maladaptive response to stress, J. Neurosci., Mar 14; 27, 11, 2781–2787 (2007)
- [5] GOMES L. M., PIMENTA M., BRANCO C., Effects of occupational exposure to low frequency noise on cognition, Aviation, Space, and Environmental Medicine, 70, A115-8. (1999).
- [6] STARCK J., TOPPILA E., PYYKKO I., Impulse noise and risk criteria, Noise Health, 5, 20, 63–73 (2003).
- [7] NOORHASSIM I., KAGA K., NISHIMURA K., Pure-tone audiometry and auditory brainstem responses in noise-induced deafness, Am. J. Otolaryngol., 17, 1, 31–35 (1996)
- [8] HALL J. W., RUPP K. A., Auditory brainstem response: recent developments in recording and analysis, Adv. Otorhinolaryngol., 53, 21–45 (1997)
- [9] FJELL A., WALHOVD K. B., Effects of auditory stimulus intensity and hearing threshold on the relationship among P300, age, and cognitive function, Clinical Nerophysiology, 114, 799–807 (2003).