

MEASUREMENTS OF CLICK-EVOKED OTOACOUSTIC EMISSION IN INDUSTRIAL WORKERS WITH NOISE-INDUCED HEARING LOSS

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Abstract. Click-evoked otoacoustic emission (c-EOAE) was analysed in a group of 122 males (= 244 ears) occupationally exposed to industrial noise levels of 89–94 dB (A). The highest intensity of cochlear responses was observed at 1 and 2 kHz, while at the higher frequencies (3–4 kHz), c-EOAE spectrum was contracted proportionally to the level of the hearing loss and duration of occupational exposure to noise. If hearing loss in pure-tone audiometry exceeded 30 dB HL at 1 kHz and 40 dB at 2 and 4 kHz, c-EOAE was absent.

The analysis of the relationship between c-EOAE spectrum and hearing threshold in pure-tone audiogram showed correlation ($R = 0.43$ to 0.48 , at $p < 0.01$) between distribution of emission energy and hearing threshold at the same range of frequencies.

It was found that the c-EOAE amplitudes as well as the values of c-EOAE energy in the noise-exposed people were lower by about 3 dB SPL than in the controls, especially at 4 kHz even in cases of pure-tone hearing thresholds ≤ 20 dB HL.

This study indicates that, owing to c-EOAE, it is possible to detect a slight noise-induced cochlear lesion which may be overlooked in pure-tone audiometry.

INTRODUCTION

Long years of animal studies of acoustic trauma have resulted in a relatively precise determination of noise-induced structural changes in the inner ear (3,30,34,40,41,46).

It has been evidenced that outer hair cells (OHCs), in which degenerative processes occur earliest, are most sensitive to noise (3,40,46).

Bohne and Rabitt (3), describing qualitative changes, indicated that mechanical damage of OHCs stereocilias and of the inner hair cells (IHCs), were typical pathomorphological manifestations in noise-induced cochlear impairments. In the case of prolonged exposure, changes in vascular stria, spiral ganglion and auditory nerve fibres may also occur (30,34,41,45).

Recent experiments have revealed that sometimes there may be no close relationship between the degree of acoustic trauma damage to hair cells of Corti's organ and the extent of hearing impairment, as hair cells are able to regenerate if the basal and supporting cells have remained undamaged (13,14).

Thus, a hypothesis has been proposed saying that electrophysiological changes which occur in the cochlea may prove to be a more sensitive indicator of noise-induced hearing loss than morphological changes which are better correlated with the duration of the exposure than with the shift of the hearing threshold in pure-tone audiometry (2,29,45,46).

In the light of the most recent findings, OHCs play the major role in the active cochlear micromechanism. Owing to their specific ability of slow/fast motility, OHCs participate in the active cochlear amplification of the acoustic stimulus and monitor the activation of IHCs which are responsible for perception of acoustic phenomena. It has also been evidenced that OHC movements probably generate otoacoustic emission (25,49).

The discovery of otoacoustic emission (OAE) as well as formation of systems (ILO 88 and ILO 92) facilitating the reproducibility of OAE measurements have provided a new non-invasive instrument for an objective evaluation of the OHC activity-related cochlear function. This method has already been successfully applied as a screening test in newborns (6,8,43).

At present, the possibility of using OAE measurements in monitoring drug ototoxicity and noise exposure effects is the subject of particular interest (9,23,50).

Since the early 1980s, numerous reports on the use of otoacoustic emission, particularly distortion product otoacoustic emission (DPOAE), in the assessment of cochlear sensitivity to acoustic overstimulation in animals have been published (10,16,17,31,42,44,47,50).

In majority of the studies, the effect of short (from 1 to 120 min), single exposure to the high levels of noise (from 90 to 110 dB SPL) producing reversible changes in outer hair cells manifested by temporary changes in the DPOAE amplitude and temporary threshold shift (TTS) was examined (16,17,42,47,50).

Various valuable information on spontaneous (SOAE), transiently evoked (TEOAE), and distortion product otoacoustic emissions were also obtained from the human-ear studies in people with normal hearing and with pathological changes, including cochlear and retrocochlear hearing loss (e.g. Meniere's disease, presbycusis, acoustic neuroma) (5,6,7,12,29,39).

However, sufficient data on TEOAE in ears under prolonged repeated occupational exposure to industrial noise possibly leading to permanent threshold shift (PTS) are still not available.

Thus, the aim of this work was to investigate the incidence of click-evoked otoacoustic emission (c-EOAE) in the ears of patients with noise-induced hearing losses at different stages of their development, as well as to assess the usefulness of the method in early detection of hearing impairments.

MATERIALS AND METHODS

The study comprised 122 male workers employed in a cotton plant and in a piston ring factory. They were exposed 8 hours daily to continuous broad-band noise. The mean noise levels at workplaces ranged from 89 to 94 dB(A). Only those

people who had shown a normal otoscopic picture of the eardrum, and who had not reported during the interview either past diseases of the ear, or exposure to hearing-impairing agents other than noise were enrolled in the audiological examinations.

Adopting the duration of noise exposure as a criterion, the subjects were divided into four groups: group I — < 1–5 years of exposure; group II — 6–10 years; group III — 11–20 years and group IV — more than 20 years of exposure.

The control group consisted of 48 non noise-exposed otologically healthy people. A general characteristics of the subjects is given in Table 1.

Table 1. Characteristics of the subjects

Group	Exposure duration (yrs)	Number of subjects (n)	Mean age	Mean exposure duration
I	< 1–5	n = 43 (= 86 ears)	32.34 ± 9.74	2.93 ± 1.69
II	6–10	n = 32 (= 64 ears)	38.49 ± 6.60	8.41 ± 1.60
III	11–20	n = 25 (= 50 ears)	42.52 ± 7.60	16.48 ± 3.95
IV	> 20	n = 22 (= 44 ears)	51.36 ± 5.09	29.09 ± 4.69
Controls group	0	n = 48 (= 96 ears)	22.70 ± 2.30	0

In addition to the primary click-evoked otoacoustic emission measurements, all the subjects underwent a battery of audiological tests including pure-tone audiometry, impedance audiometry, and auditory brainstem response (ABR) recording.

The tests were intended to identify the extent and site of hearing impairment, as well as to exclude possible conductive hearing losses and “objectivize” the hearing threshold in cases of suspected malingering.

Pure-tone air and bone audiometric examination was performed in a sound-proof chamber under standard acoustic conditions according to the relevant ISO recommendations using an OB 822 Madsen audiometer with TDH-39 earphones. Short increment sensitivity index (SISI) test was also applied, and the score between 60 and 100% was considered to be indicative of cochlear hearing loss, between 0 and 25% of *laesio nervi cochlearis*, whereas in the cases of values > 25% and < 60% the score closer to 60% was treated as the indicator of cochlear loss.

Impedance audiometry (ZO 20—20 Madsen equipment) included measurements of ear canal volume, tympanic pressure, static compliance, gradient and acoustic reflex threshold (ART). ART was determined at four frequencies: 500, 1000, 2000 and 4000 Hz following ipsilateral and contralateral pure-tone stimulation with intensity between 70 dB and 115 dB; it was assumed that the gap between ART and pure-tone thresholds less than 60 dB indicated the incidence of Metz-recruitment (32).

The audiometric thresholds were verified against auditory brainstem responses (ABRs) obtained by means of the Polish EPTEST system. The frequency-specific ABRs produced by digitally synthesized tone pips stimuli of 1 000, 2 000 and 4 000 Hz were recorded as average after 2 048 repetitions. The lowest intensity of stimulus at which V wave could be identified was taken as a threshold level.

Click-evoked otoacoustic emission (c-EOAE) was measured and analysed according to the method developed by Bray and Kemp (8), using the ILO 88 Otodynamics system consisting of a probe with a miniature loudspeaker and microphone placed in the ear canal, a generator of acoustic stimuli, a pre-amplifier and an averaging unit. Prior to the examination, the electronic measuring and sound-generating systems were calibrated according to the manufacturer's instructions. The single channel probe suitable for adult subjects was provided with a soft overlay in order to assure its complete adherence to the walls of the ear canal. Correct placement of the probe in the canal (noise level) was automatically monitored during the test.

A set of four clicks with a frequency of 50 Hz, intensity of 76–87 dB SPL, and duration of 80 msec, was used as the acoustic stimulus. The stimulus level was each time automatically compensated, depending on individual dimensions of the ear canal. Each set of stimuli comprised three clicks with the same polarity and amplitude, whereas the fourth click was characterized by three times higher amplitude and reverse polarization. Owing to the application of the above stimuli, and by means of special averaging technique, undesirable linear elements were rejected and the cochlear response nonlinear components maintained.

C-EOAE responses were averaged over 260 repetitions, filtered within a broad band (from 500 Hz to 6 kHz), and then analysed during 2.5–20 msec following initial acoustic stimulation (each stimulus was followed by a 2.5 msec blockage). Responses to the stimuli were averaged as two 20 msec buffers, A and B, to determine the correlation and repeatability of the cochlear responses. According to the criteria proposed by Hauser (21), the result of c-EOAE measurement was assumed positive if the stability of the measurement probe was not less than 80%, and the response correlation was above 70%. The presence or absence of c-EOAE was identified (an otoacoustic emission was assumed to occur when the intensity was at least 3 dB above the background noise), and the amplitude and response spectrum were evaluated. The ILO-88 ILOSTAT programme (Interpretation Statistics) developed by Otodynamics Co. was used for the quantitative assessment; it facilitated the interpretation and statistical analysis of the highest stimulation value in dB SPL, stability of the probe position, c-EOAE magnitude in dB-SPL in the whole range of frequencies (500–5000 Hz), level of background noise in the same range of frequencies and the reproducibility value (REPRO) in % for each c-EOAE measurement.

In view of inter-individual differences and the differences between ears of the same individual reported by some authors (7,37), the measurements of c-EOAE were taken in each ear separately.

In total, results of c-EOAE measurements obtained in 122 subjects (= 244 ears) exposed to noise were compared and assessed versus 48 (= 96) otologically healthy controls.

STATISTICAL ANALYSIS

The test and control groups were characterized according to mean values of relevant parameters and their standard deviations.

One-way analysis of variance with multiple comparison test was used to compare mean values between noise-exposed groups and the controls.



The results obtained in the noise-exposed subjects ($n = 122$) were compared with those in the controls ($n = 48$) using Student's t test (for equal and different variances). The relationships between the hearing thresholds in pure-tone audiogram and mean values of c-EOAE measurements were assessed by means of linear regression model at the significance level 0.05 for all tests. In order to estimate the age adjusted relationships, analysis of covariance with age as covariate was used.

RESULTS

Pure-tone audiometry

Pure-tone audiometry showed sensorineural hearing loss in all 122 subjects exposed to noise; in 67 subjects (54.91%) it was manifested by cochlear hearing loss concurrent with recruitment, and in 55 subjects (45.09%) the hearing loss was classified as cochlear nerve lesion.

Table 2. Mean hearing loss in pure-tone audiogram in the study groups

Group (years of exposure)	Ear	Hearing threshold in pure-tone audiometry (mean values)				
		Frequency				
		250 Hz	500 Hz	1000 Hz	2000 Hz	4000 Hz
I <1–5	R	12.56 ± 4.42	12.91 ± 5.15	16.47 ± 5.86	20.35 ± 7.82	26.51 ± 9.16
	L	11.78 ± 3.36	12.97 ± 4.56	14.88 ± 5.57	18.69 ± 8.12	24.28 ± 10.22
II 6–10	R	14.37 ± 4.71	14.06 ± 5.15	17.18 ± 5.67	21.56 ± 8.65	30.00 ± 12.89
	L	15.00 ± 6.22	15.00 ± 6.22	18.13 ± 6.93	22.19 ± 10.07	32.50 ± 13.68
III 11–20	R	16.20 ± 5.64	17.60 ± 6.78	20.80 ± 10.38	27.00 ± 11.27	42.40 ± 14.66
	L	17.60 ± 7.92	20.00 ± 9.35	22.80 ± 12.42	30.00 ± 13.69	45.80 ± 15.52
IV >20	R	30.91 ± 12.31	33.41 ± 14.67	37.50 ± 15.94	50.68 ± 14.08	63.18 ± 13.05
	L	31.82 ± 13.41	32.73 ± 13.95	38.86 ± 16.32	53.64 ± 16.49	62.04 ± 14.19

R – right
L – left

Table 2 illustrates mean values of the hearing thresholds in pure-tone audiogram at frequencies ranging from 250 to 4 000 Hz in four groups of subjects in relation to the period of employment. A diagram indicating the extent of hearing loss is plotted in Figure 1.

The results of our analysis of the growing extent of hearing loss at three frequencies (1, 2 and 4 kHz) for groups II, III and IV compared with the results obtained for group I (shortest period of employment) revealed that the differences between mean hearing thresholds were least significant between groups I and II (from 0.71 to 8.22 dB), slightly higher between groups I and III (4.33 to 21.52 dB) and highest between groups I and IV (21.03 to 37.76 dB). Statistical analysis (using one-way analysis of variance with multiple comparison test) of the results showed significant differences ($p < 0.001$) between mean hearing thresholds

Pure tone audiogram

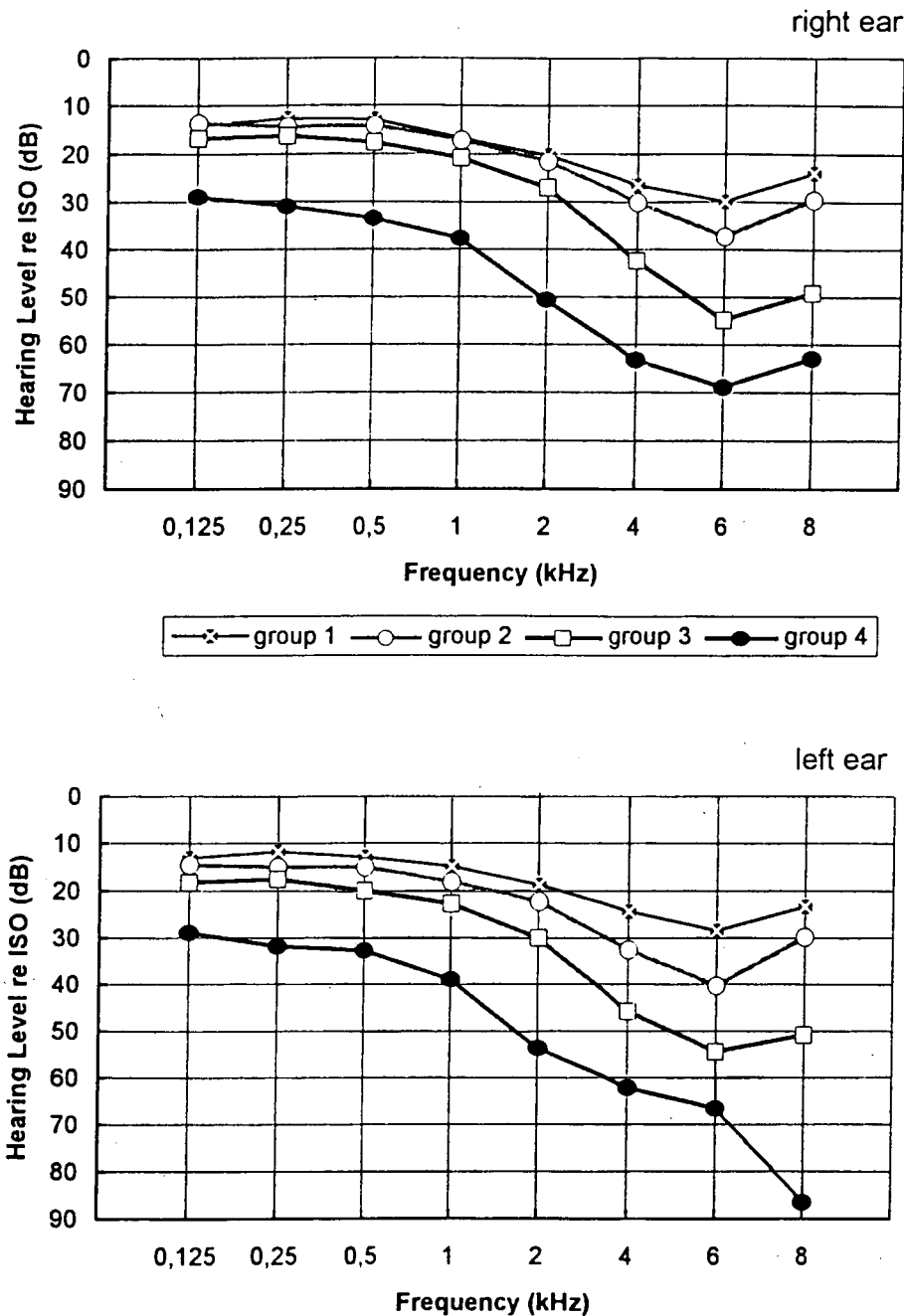


Fig. 1. Mean pure-tone hearing threshold as a function of noise exposure duration
Group I 0–5 years of exposure; Group II 6–10 years; Group III 11–20 years; Group IV over 20 years of exposure.

in group III (11–20 years of employment) or group IV (more than 20 years of employment) and hearing thresholds in two other groups of subjects employed for periods not longer than 10 years. The covariance analysis of the data with age as covariate revealed that the hearing loss increase was stronger related with the period of employment under noise exposure than with the age of the subjects ($p = 0.001$).

Impedance audiometry

The A configuration of tympanogram was found in all subjects, and mean values of ear canal volume, tympanic pressure, static compliance and gradient remained within the range considered to be normal. The incidence of acoustic reflex threshold at least at two frequencies (500 Hz and 1 kHz) was recorded in 96/122 subjects, and that was in agreement with the extent of hearing loss observed in pure-tone audiogram. The values of mean acoustic reflex threshold ranged from 79.54 ± 15.26 dB to 95.00 ± 14.47 dB, increasing proportionally to the extent of hearing loss.

An analysis of the relationship between acoustic reflex threshold and pure-tone threshold revealed a gap lower than 60 dB in 67 subjects (54.91%), ranging from 24.38 ± 9.21 dB to 30.29 ± 11.25 dB, which confirmed the presence of recruitment.

Examination of auditory brainstem responses (ABR)

The comparison between pure-tone hearing thresholds at 1, 2 and 4 kHz and ABR thresholds showed that mean electrophysiological thresholds were higher than audiometric ones by about 5–10 dB n HL at 1 kHz, 10–15 dB n HL at 2 kHz, and 20 dB n HL or more at 4 kHz; however, these differences were less significant in the group of patients with cochlear hearing loss and more significant in the cochlear nerve lesion group.

Click-evoked otoacoustic emission (c-EOAE) measurements

The measurements of c-EOAE in the test group (244 ears) revealed that it was present (at least at one frequency) in 221/244 ears examined, and completely absent in 23 ears.

Table 3 shows mean values of c-EOAE findings in the full frequency range and at 1, 2, 4 and 5 kHz (for each ear separately) in the groups of subjects exposed to noise and in the controls. Table 4 illustrates the results of the analysis of REPRO (%) values in the test groups at the same (1 to 5 kHz) frequency band.

An analysis of amplitudes of c-EOAE responses in the individual noise-exposed groups showed that their mean values (identified for both ears) accounted for 9.21 ± 4.14 dB SPL in group I; 6.47 ± 4.78 dB SPL in group II; 3.20 ± 5.60 dB SPL in group III; and 0.67 ± 4.86 dB SPL in group IV. The mean value of c-EOAE amplitude in groups I–IV was reduced by about 3 dB SPL, and the reduction was related with the growth of hearing loss in pure-tone audiogram attributable to longer employment under noise exposure.

Table 3. Mean values of the amplitude and c-EOAE spectrum at frequencies from 1 kHz to 5 kHz in the groups of subjects exposed to noise and in the control group

Group (years of exposure)	Ear	Amplitude of c-EOAE (dB SPL)	Spectrum c-EOAE (dB SPL)				
			Frequency				
			1 kHz	2 kHz	3 kHz	4 kHz	5 kHz
I <1–5	R	9.64± 4.41	–5.77±4.73	–9.04±4.87	–12.30±5.49	–15.58± 5.02	–21.92±3.97
	L	8.72± 3.99	–6.45±4.95	–9.28±4.69	–13.89±4.70	–18.26± 4.21	–20.95±2.34
II 6–10	R	7.35± 3.99	–7.47±4.52	–11.41±4.79	–16.48±4.81	–17.83± 4.22	–
	L	5.60± 5.38	–7.62±5.04	–11.92±5.58	–14.19±4.80	–19.07± 5.59	–21.87±3.43
III 11–20	R	3.21± 5.16	–9.30±3.81	–12.35±4.86	–15.53±4.77	–15.70±15.70	–
	L	3.18± 6.11	–8.75±5.36	–11.86±3.06	–15.15±2.97	–19.00± 6.22	–
IV >20	R	–0.16± 4.44	–11.24±4.85	–14.08±3.63	–	–	–
	L	1.50± 5.22	–10.16±5.11	–11.00	–21.90	–	–
Controls	R	12.42± 4.15	–4.25±4.83	–6.27±4.19	–8.97±5.03	–13.53± 6.03	–20.17±5.43
	L	12.72±14.27	–4.97±3.45	–8.53±4.87	–10.57±4.97	–13.09± 7.62	–20.78±4.08

R – right
L – left

Table 4. Mean values reproducibility (%) at frequencies from 1 kHz to 5 kHz in the group of subjects exposed to noise and in the control group

Group (years of exposure)	Ear	REPRO (%) by frequency					Whole-REPRO (%)
		1 kHz	2 kHz	3 kHz	4 kHz	5 kHz	
I <1–5	R	78.86±23.66	79.33±28.55	55.84±41.07	33.35±42.24	9.77±27.33	79.26±20.52
	L	74.88±22.75	78.36±24.48	54.12±40.21	34.24±42.27	10.33±28.58	75.45±19.43
II 5–10	R	73.37±29.60	70.47±33.48	48.44±39.37	22.81±37.45	2.44±13.79	72.31±24.91
	L	70.22±32.31	56.94±37.17	40.37±42.27	27.22±41.29	13.25±31.46	67.87±27.05
III 11–20	R	51.60±36.46	56.20±39.08	24.84±37.79	6.76±23.47	–	54.44±32.28
	L	54.92±34.22	44.56±41.47	18.32±33.58	3.44±17.20	–	50.92±31.07
IV >20	R	51.59±33.95	32.95±37.34	–	–	–	46.73±29.22
	L	44.00±36.05	20.64±36.09	4.00±18.76	–	–	40.91±32.12
Control	R	86.64±13.48	94.36± 5.31	88.47±18.47	83.62±25.08	37.47±44.19	90.07± 8.62
	L	82.00±17.15	90.50± 9.09	88.56±15.35	79.69±26.45	43.94±43.04	84.70±11.34

R – right
L – left

In the group of people with recorded incidence of c-EOAE, the highest response intensity was observed at 1 and 2 kHz, whereas at higher frequencies the emission energy in some test subjects decreased, or declined completely. Graphical representation of c-EOAE data and REPRO (%) values at 1 to 5 kHz frequency band in the noise-exposed groups and in the controls is shown in Figure 2.

Both ears

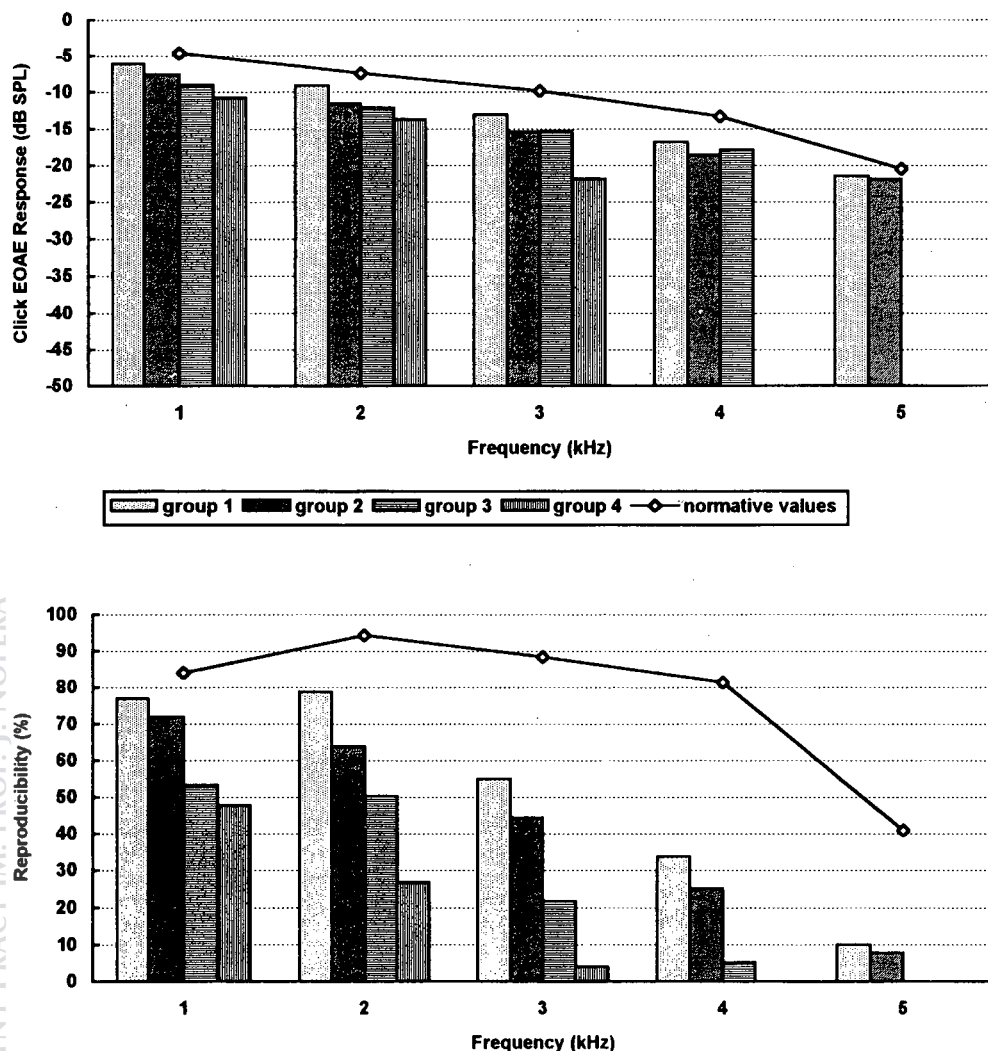


Fig. 2. Mean values of c-EOAE spectrum and REPRO (%) at 1 kHz to 5 kHz frequency range in the noise-exposed subjects versus the controls.

An analysis of c-EOAE spectrum in individual groups (I–IV) revealed a clear-cut contraction of otoacoustic emission band at high frequencies (particularly at 4 kHz) which progressed with the increase in hearing loss and longer periods of employment under noise exposure. However, if pure-tone hearing loss exceeded 30 dB HL at frequencies of 1 Hz and 40–dB HL at frequencies of 2 and 4 kHz, the incidence of c-EOAE was not observed in any of the test subjects. Figures 3 and 4 illustrate examples of individual findings.

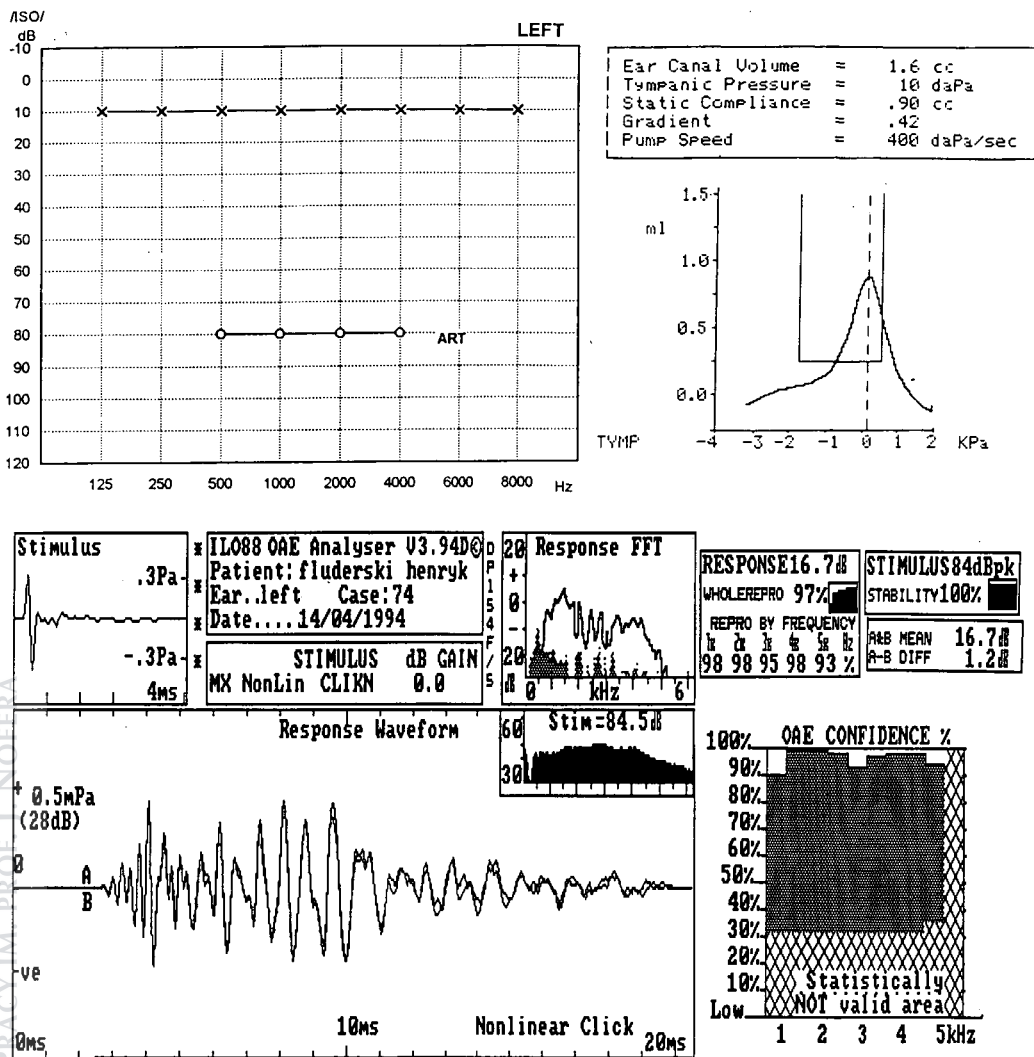


Fig. 3. C-EOAE and pure-tone hearing threshold in a 25-year-old man with normal hearing (subject from control group)

A. Pure-tone hearing threshold is 10 dB HL. B. Typanometric findings within the range considered to be normal. C. C-EOAE responses with the highest level of 3.1. dB SPL at frequency of 1000 Hz and amplitude 16.7 dB SPL. D. Results of REPRO analysis.

In the control group (96 ears) of otologically healthy people, c-EOAE incidence was observed in 96/96 ears in the range of frequencies from 1 to 4 kHz and in 44/96 ears at the frequency of 5 kHz. The highest response intensity was found at 1 and 2 kHz. The values of emission energy decreased at higher frequencies (3–4 kHz) reaching the lowest level at 5 kHz, and at this frequency it was recorded only in 42.5% of ears. The amplitude of c-EOAE response showed very high differences between individual subjects, ranging from 4.6 to 23.2 dB SPL, with mean value (for both ears) 12.57 ± 10.65 dB SPL.

The results of c-EOAE measurements in the controls ($n = 48 = 96$ ears) were compared with those in group I ($n = 43 = 86$ ears), < 1–5 years employment period. It was found that mean values of response amplitude were higher by 3 dB SPL in the control group than in group I of noise-exposed subjects, whereas mean level of emission energy was lower by 2–3.5 dB SPL in the latter than in the former, and at frequencies 3 and 4 kHz the differences were significant ($p < 0.01$); however, at 1 and 2 kHz these differences were statistically insignificant.

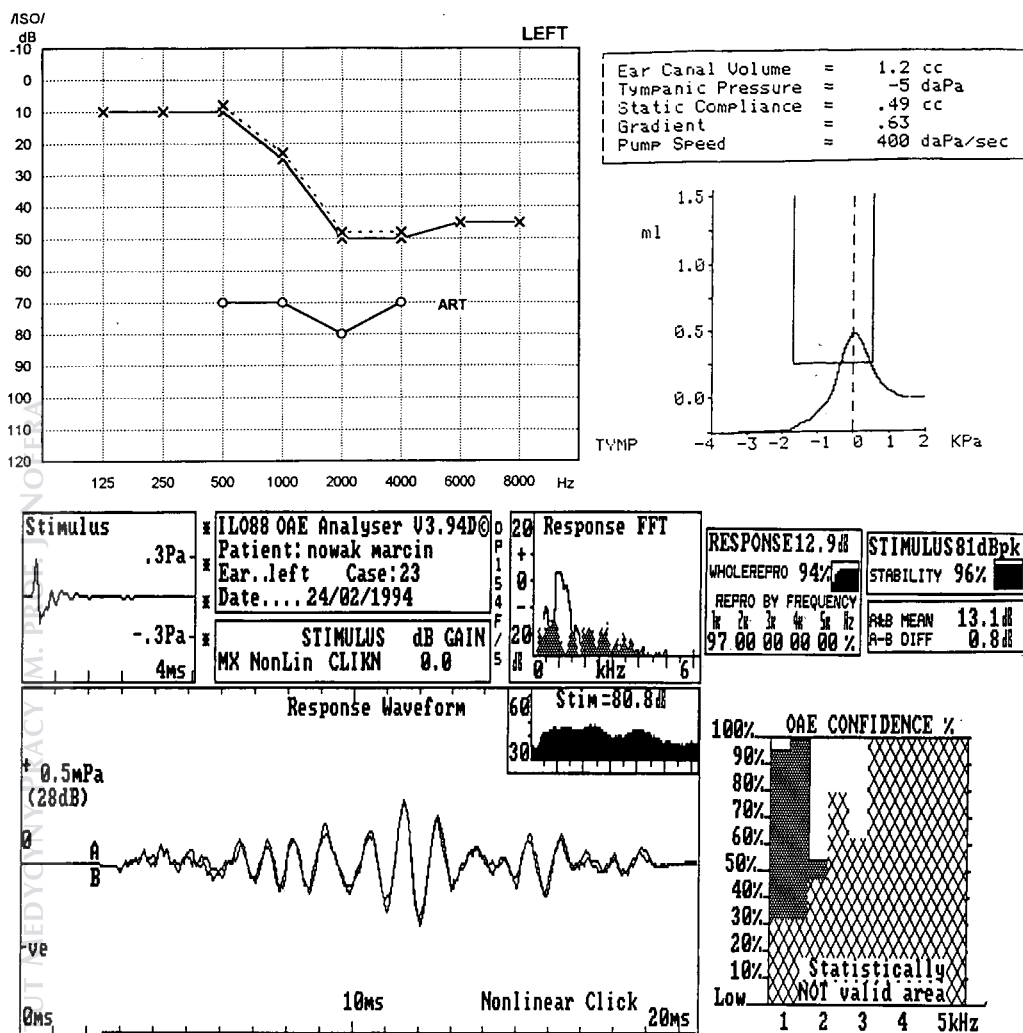


Fig. 4. C-EOAE and pure-tone hearing threshold in a 45-year-old man exposed for 20 years to noise at a mean level of about 93 dB (A).

A. In pure-tone audiogram — a profound noise-induced hearing loss ranging from 10–50 dB HL. B. Tympanometric findings within the range considered to be normal. C. A clear-cut contraction of c-EOAE spectrum. C-EOAE responses at a level of energy emission ranging from -0.4 dB SPL to 0.2 dB SPL within frequency band of 500 Hz to 1000 Hz; amplitude 12.9 dB SPL. D. Results of REPRO analysis.

Assessment of cochlear response correlation in c-EOAE measurements, based on REPRO analysis, revealed correlation coefficient of $87.27 \pm 10.43\%$ in the control group and $77.38 \pm 19.96\%$ in the noise-exposed group I, the correlation being highest at 2 kHz and lowest at 4 and 5 kHz.

In order to evaluate the sensitivity of c-EOAE measurements in early detection of cochlear sensitivity to noise, the results of examination of 41 ears in group I whose pure-tone hearing threshold was ≤ 20 dB HL at frequencies ranging from 250 Hz to 4 kHz were subjected to an additional analysis, and this group of ears was classified as subgroup Ia. Mean age of subjects in subgroup Ia was 29.14 ± 7.98 years.

Table 5 gives mean values of pure-tone hearing threshold at selected frequencies ranging from 250 Hz to 4 kHz in the control group and in subgroup Ia.

Table 5. Mean hearing loss in pure-tone audiogram in the control group and in 41 ears of subjects exposed to industrial noise for 0.5 years (group Ia) with hearing threshold ≤ 20 dB HL

Frequency	Hearing threshold in pure-tone audiometry (mean values)	
	Controls (96 ears)	Group Ia (41 ears)
250 Hz	2.50 ± 3.32	10.48 ± 2.69
500 Hz	2.60 ± 3.32	11.46 ± 3.39
1000 Hz	3.38 ± 3.51	12.80 ± 4.19
2000 Hz	3.39 ± 3.62	15.36 ± 4.92
4000 Hz	4.47 ± 3.73	17.31 ± 2.88
Mean age	22.70 ± 2.30	29.14 ± 7.98

Table 6 presents mean values of c-EOAE measurements in the frequency band from 0.5 to 5 kHz, and mean values of c-EOAE amplitudes in the control group versus those in subgroup Ia. It can be seen that mean values of emission energy at 0.5–2.5 kHz were almost the same in both groups, but at frequencies of 3–5 kHz, these values were lower by 2–4 dB SPL in noise-exposed subjects of subgroup Ia, and at 3.5 kHz and 4 kHz the differences were significant ($p < 0.01$). The mean value of the magnitude of c-EOAE amplitude was higher in the control group by about 3 dB SPL than mean amplitude in ears of subjects in the noise-exposed subgroup Ia.

The comparison of c-EOAE measurements in the control group with those carried out in the other groups of noise-exposed subjects (II–IV) indicated that mean levels of emission energy differed in both groups, especially at 2, 3 and 4 kHz. It also revealed lower values in noise-exposed subjects, the lowering being related with the extent of hearing loss in pure-tone audiogram, duration of exposure, and age. The influence of age on the extent of hearing loss and the magnitude of c-EOAE response in the exposed groups and in the controls was assessed by covariance analysis with age as covariate. The results evidenced that the age and effect of noise had a significant impact on the extent of hearing loss in pure-tone audiogram and click-evoked otoacoustic emission spectrum. However, the increase of hearing loss as well as the reduction of the amplitude and contraction of c-EOAE spectrum were related more with occupational exposure to noise than with the age of the subjects ($p = 0.001$).

Table 6. Mean values of c-EOAE measurements at frequencies from 1 kHz to 5 kHz in the control group and in 41 ears of subjects exposed to industrial noise for 0–5 years (group Ia) with hearing threshold ≤ 20 dB HL (A). Mean values REPRO (%) at frequencies from 1 kHz to 5 kHz in both groups under study (B)

A	Frequency (kHz)	Spectrum c-EOAE (dB SPL)	
		Controls (96 ears)	Group Ia (41 ears)
	1	-3.16 ± 4.25	-4.50 ± 4.96
	2	-9.89 ± 5.00	-9.66 ± 4.22
	3	-10.21 ± 5.46	-12.00 ± 4.32
	4	-15.83 ± 5.68	-18.92 ± 4.96
	5	-21.78 ± 5.43	-26.90 ± 0.84
	Amplitude c-EOAE (dB SPL)	12.47 ± 10.65	9.21 ± 4.14
B	Frequency (kHz)	Reproducibility (%)	
		Controls (96 ears)	Group Ia (41 ears)
	1	84.17 ± 15.63	79.73 ± 21.80
	2	94.36 ± 5.31	82.90 ± 23.38
	3	88.52 ± 16.78	66.58 ± 37.40
	4	81.52 ± 25.75	46.65 ± 42.42
	5	40.92 ± 43.46	8.39 ± 26.01

In the noise-exposed groups, correlation between the hearing threshold in pure-tone audiogram at three frequencies (1, 2 and 4 kHz) and c-EOAE spectrum was assessed by linear regression (level of significance 0.05 for all tests). Correlation was found to occur at frequencies 1 kHz ($R = -0.45$, $p < 0.01$), 2 kHz ($R = -0.48$, $p < 0.01$) and 4 kHz ($R = -0.39$, $p < 0.01$).

DISCUSSION

Evoked otoacoustic emission, known also as transiently evoked otoacoustic emission (TEOAE), is a signal generated in the cochlea in response to short stimuli such as clicks and short sine wave pulses (24,25,37). Since TEOAE occurs in almost all (98–100%) ears with normal hearing and provides information on the normal or abnormal functioning of outer hair cells (OHCs), it has become widely used in clinical examinations (6,7,12,37,39).

It has been evidenced that OHCs are extremely sensitive to harmful factors, such as ototoxic drugs, noise or hypoxia, and selective damage of these cells results in hearing impairment of 30 or even 50 dB even if inner hair cells (IHCs) are left intact (7,9,23,29,37,39,46).

The aim of our study was to assess the feasibility of the click-evoked otoacoustic emission (c-EOAE) measurement in detection and diagnosis of noise-induced hearing loss (NIHL) at various stages of its development.

A selected group of workers characterized by different duration of noise exposure and age, and non noise-exposed controls were the subjects of the study.

The majority of the test population, namely 75 out of 122 subjects (= 150/244 ears) were 22–45 years old, and the duration of their occupational noise exposure did not exceed 10 years. A smaller test group comprised 47 people (= 94 ears), on average 45 years old, but none older than 60 years, with a noise-exposure history of 11 to 20 years or longer.

Before the c-EOAE measurements, the battery of audiological tests was used for assessment of hearing in all subjects.

Pure-tone audiometry showed sensorineural hearing loss in all 122 people exposed to noise; in 67 subjects (54.91%) it was attributable to cochlear lesion, and in the remaining 55 subjects (45.09%) to the damage of the auditory nerve fibres (probably at the IHC level).

Cochlear location of hearing loss was found in the majority of subjects 67/75 (134/150 ears) with occupational exposure to noise below 10 years, and in 8/75 people (16/150) a damage of the auditory nerve was observed. In all subjects exposed to noise for more than 10 years (groups III and IV), 47/47 (= 94 ears) hearing impairment was located in the auditory nerve. That is in good agreement with observations of other authors (45,46) who have reported that the first 10 years of exposure to noise induce selective damage of cochlear hair cells. After that period the incidence of Metz recruitment declines because of secondary degeneration of nervous fibres and ganglion cells.

The assessment of the trend of the hearing impairment development related to the duration of occupational noise exposure provided evidence that mean hearing loss increased with the period of employment under noise exposure. The greatest changes in the hearing threshold were observed at 4 kHz, moderate at 2 kHz, and slight changes at frequencies below or close to 1 kHz. It was estimated that after next 10 years of occupational exposure, mean hearing loss in the test population was increased by about 10 dB, particularly at the frequency of 4 kHz. Similar results were reported by other authors (45).

The c-EOAE measurements in the subjects exposed to noise (244 ears) revealed the incidence of this emission, (at least at one frequency) in 221/244 ears examined and its complete absence in 23 ears.

The comparison between the results obtained in people exposed to noise (221 ears) and in the control group (96 ears) with normal hearing showed significant differences in such parameters as amplitude, emission energy and the range of response spectrum. In both groups (exposed and control) the highest intensity of c-EOAE response was observed at 1 and 2 kHz, whereas at higher frequencies emission energy declined, the decline being most evident in the noise-exposed subjects. These results correspond with those of other authors (7,36,37,39). Bonfils et al. (7) reported that in the majority of healthy ears, the highest intensity of TEOAE response had been recorded at frequencies between 1 and 2 kHz; also in the ears with sensorineural hearing loss, the same range of frequencies was most important if the TEOAE incidence was to be retained (5,36,39).

Our evaluation of mean values of the amplitude of c-EOAE responses and mean values of emission energy at frequencies of 1–5 kHz and in the whole range of frequencies from 0.5 to 5 kHz revealed that the amplitude of c-EOAE in people exposed to noise was considerably lower than in the control group, and the emission energy in the exposed group at higher frequencies (3 and 4 kHz) decreased or declined completely. The spectrum of c-EOAE responses became constricted in

proportion to the extent of hearing loss and duration of occupational exposure. That was particularly remarkable at high frequencies.

The c-EOAE incidence was not observed in any test subject if hearing loss exceeded 30 dB HL at 1 kHz and 40 dB HL at 2 and 4 kHz. Similar data were reported by other authors (7,12,25,36,37,39) who found that TEOAE incidence was recorded neither in ears with hearing loss higher or equal to 40 dB HL at 1 kHz nor if the mean value of the hearing threshold in pure-tone audiogram was higher than 35 dB HL at 0.5, 1, 2 and 4 kHz.

Experimental and model studies carried out thus far on the monitoring of the effects of noise exposure have shown that the lowering of the cochlear response amplitude proves to be the first consequence of suprathreshold acoustic stimulation, and this applies to all kinds of otoacoustic emission (10,17,19,23,35,39,48,50).

The influence of acoustic overstimulation on otoacoustic emission has been best determined for distortion product otoacoustic emission (DPOAE) (1,10,18,31,42,44,50). Stainback (42) indicated that even a slight cochlear dysfunction induced by a small stimulation of 100 dB SPL for one hour may lead to the reduction of DPOAE amplitude at a relevant frequency interval.

There are only few papers published on TEOAE in ears of people with occupational noise-induced hearing loss (NIHL) (26,27,39).

Kvaerner, Engdehl et al. (27) evaluated the results of pure-tone audiometry and TEOAE in 13 workers exposed to industrial noise of 85–90 dB (A) for 7 hours during 3 working days. Analysis of the results before and after exposure showed the reduction in the amplitude of TEOAE response as well as temporary threshold shift at frequencies of 4 and 6 kHz (27).

One of the objectives of our study was to assess whether c-EOAE is more effective than pure-tone audiometry in early identification of noise-induced changes in the cochlea.

Therefore, in order to evaluate the c-EOAE sensitivity in recognizing early and slight NIHL, group I (< 1–5 years of employment) composed of the largest number of subjects ($n = 43 = 86$ ears) was chosen as the most suitable. This group comprised people with cochlear sensorineural hearing loss only (with the incidence of recruitment), including 41 ears with hearing loss ≤ 20 dB HL at frequencies ranging from 250 Hz to 4 kHz (subgroup Ia), and 45 ears with hearing loss not exceeding 30 dB HL at frequencies ranging from 3 to 4 kHz. The influence of age on the extent of hearing loss and the magnitude of c-EOAE was smaller in this group than in groups II and IV because it was composed of the youngest subjects, aged 22–42 years (mean age 32.34 ± 9.74).

According to the data by Sulkowski (45) and Dieroff (15) obtained from field audiometric surveys of large noise-exposed populations, NIHL gradually progressed in a phasic way as a function of noise levels and duration of exposure, and was characterized by certain regularity. An early stage of NIHL, observed during the first 3 years of noise exposure, was manifested by a clear-cut notch at the frequency range of 3–6 kHz, with the maximum usually at 4 kHz. Such findings were obtained in group I (duration of employment not longer than 5 years). Furthermore, the comparison of mean values of the c-EOAE amplitudes obtained in 41 ears of subgroup Ia with those in the controls showed that the former were lower by about 3 dB SPL, and that the mean value of emission energy at frequencies 3.5 and 4 kHz was lower by approximately 2–4 dB SPL.

It may suggest that c-EOAE measurements allow the detection of slight noise-induced disorders of the OHC function which may be overlooked in pure-tone audiogram, as the hearing threshold seems to be at the border of normal hearing acuity; a noticeable reduction of the c-EOAE amplitude and an evident decline of emission energy especially at a frequency of 4 kHz even at ≤ 20 dB pure-tone hearing thresholds appear to manifest advantages of such measurements for identifying early stages of noise-induced hearing loss. An opinion like that is shared by Collet et al. (11).

In our present study the duration of exposure to noise, and not the age of subjects, was adopted as a criterion for selecting the test groups. Nevertheless, in the final evaluation of results, the effect of age on both the extent of hearing loss in pure-tone audiogram and the incidence of c-EOAE were also taken into account. The statistical analysis (analysis of covariance with age as covariate at $p = 0.001$) of the findings showed that the increase in hearing loss as well as the decline in the amplitude level and the constriction of c-EOAE spectrum was related more with the duration of occupational exposure to noise than with the age of the subjects.

It is generally recognized that the effects of aging complicate the assessment of noise effects and precise distinction between NIHL and presbycusis component is difficult and not always feasible because of considerable variations of individual age-related hearing losses which may result from many factors, including genetic and metabolic ones (12,22,45).

Age-related incidence of c-EOAE was investigated by Bonfils et al. (5). The authors carrying out examinations in various age groups (from > 10 to < 60 years) reported that TEOAE was recorded in all ears of subjects aged under 60 but only in 35% of ears in subjects above 60 years old. Moreover, the TEOAE incidence was more related with the degree of hearing loss in those people than with their age.

In our study, correlation between the pure-tone hearing threshold and the c-EOAE amplitude and spectrum was analysed at three frequencies of 1, 2 and 4 kHz (linear regression significance level = 0.05). The spectrum of c-EOAE response correlates with the pure-tone hearing threshold but this correlation is stronger at 1 and 2 kHz ($R = -0.45$; $R = -0.48$, $p < 0.01$) and slightly weaker at 4 kHz ($R = -0.39$, $p < 0.01$). The findings are in good agreement with data of other authors. Probst and Harris (38) reported strong correlation (0.77) between the distribution of emission energy and the pure-tone hearing threshold in the same range of frequencies.

To sum up the results of our study it should be stated that the c-EOAE measurements may prove to be a sensitive and objective tool for detecting early symptoms of noise-induced hearing loss. Although c-EOAE cannot be used for quantitative determination of hearing loss, it can be used for recording its presence. Moreover, if c-EOAE is identified, then it is most likely that the hearing threshold in the given frequency band is ≤ 30 dB HL. The method can be applied together with other objective tests in the complex evaluation of hearing in medical/legal cases, particularly in malingers.

The discussed results of c-EOAE measurements in the group of 122 subjects exposed to noise were additionally verified by distortion product otoacoustic emission testing, and the findings will be presented in a separate report.

CONCLUSIONS

1. In people exposed to noise, the amplitude and c-EOAE emission energy are evidently lower than in non-exposed people with a similar pure-tone hearing threshold.
2. The incidence of c-EOAE can be detected in ears with noise-induced hearing loss, if the loss does not exceed 30 dB HL at 1 kHz and 40 dB HL at frequencies of 2 and 4 kHz.
3. The largest amplitudes of c-EOAE are recorded at frequencies of 1 and 2 kHz, whereas at higher frequencies (3–4 kHz) emission spectrum becomes constricted proportionally to the extent of hearing loss and the duration of occupational exposure to noise.
4. The spectrum of c-EOAE response correlates with the pure-tone hearing threshold, but this correlation is stronger at frequencies of 1 and 2 kHz ($R = -0.45$; -0.48 , $p < 0.01$) and slightly weaker at a frequency of 4 kHz ($R = -0.39$, $p < 0.01$).
5. The c-EOAE measurement can be applied as a valuable complement to pure-tone audiogram in the detection of slight cochlear impairments induced by exposure to noise.

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