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Mechanisms in noise-induced permanent hearing loss: an evoked otoacoustic emission and auditory brainstem response study

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Abstract

In this study 22 patients (44 ears) with noise-induced permanent hearing loss were audiologically evaluated using transient-evoked otoacoustic emissions (TEOAE) and auditory brain-stem response (ABR). Twenty-one normal subjects (42 ears) without exposure to occupational noise were used as controls. Based upon the hearing loss at 4, 3, 2 and 1 kHz on the pure-tone audiogram, they were classified into four groups. In group 1 (eight ears), emissions were present in all ears but their TEOAE-noise level and their reproducibility (percentage) proved to be weak. The auditory brain-stem response (ABR) indicated that the I/V amplitude ratio, the latency values of wave V and the I–V intervals fell within the normal range in all ears. In Group 2 (14 ears), 40 per cent had no emissions, whereas the remaining ears showed weak emissions. The ABR revealed that in all ears the I/V amplitude ratio became small while wave V peak latency as well as I–V intervals were within the normal range. In Group 3 (10 ears), emissions were absent in 50 per cent, while in the other ears the emissions were very weak. The ABR revealed that the I/V amplitude ratio, which could be calculated in the 60 per cent in which wave I was present, was smaller than in Group 2. Wave V latency as well as I–V intervals were within the normal range. In Group 4 (12 ears), none of the ears showed emissions. The ABR indicated that the I/V amplitude ratio was much smaller when wave I was present (27 per cent) as well as I–V interval values being within the normal range. Wave V absolute latency value (ΔV index) indicated a positive index in 17 per cent of this group (two ears) when wave I was absent. In the present study a dynamic process from cochlear outer hair cells to cochlear neurons was seen, correlating with an increasing hearing loss.

Key words: Hearing loss, noise induced; Audiometry, evoked response; Acoustic emissions

Introduction

Noise-induced auditory damage is characterized by sensorineural hearing loss. It is frequently referred to as a '4 kHz notch' (Bess and Humes, 1995). Many techniques have been used to study the morphological lesions caused by noise damage in the animal. There is general agreement that it is associated with degeneration of outer and inner hair cells (Ward *et al.*, 1981). There is, however, also evidence of damage to the dendritic nerve endings surrounding the inner and outer hair cells (Spoendlin, 1971). Changes have also been described in the spiral ganglion and the auditory nerve (Bredberg, 1968). Damage of the central auditory nervous system has been described by Morest and Bohne (1983).

The presence of evoked otoacoustic emission (EOAE) indicates mechanically active mechanisms in the cochlea. Active contraction of the outer hair cells is counteracting the high level of mechanical damping provided by the structure and composition of the cochlea. The outer hair cells are involved in this process (Kemp, 1980). The presence of the EOAE thus provides direct indication of the integrity of preneural cochlear receptor mechanisms (Prasher *et al.*, 1995; Vinck *et al.*, 1996; Xu *et al.*, 1998).

ABR is an electrophysiological procedure. Diagnostic inferences from the ABR are determined by comparing wave latencies (conduction time) and amplitudes (voltage). ABR has been proven to be useful in the differential diagnosis of sensorineural hearing loss by providing information that can objectively indicate whether the lesion is situated at the cochlear or the retrocochlear level. These parameters include absolute latencies, interpeak latency intervals, interaural differences between the absolute latencies and interpeak latency intervals, and amplitude ratio. The ΔV index has also been proposed as a diagnostic tool of sensorineural hearing loss by Prosser and Arslan (1987). This is calculated only on the basis of the wave V latency of

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the examined ear, hence allowing a strictly monaural evaluation of the ABR evoked at a single suprathreshold intensity.

The aim of the present paper is to try to verify the location of the pathological process in noise-induced permanent hearing loss by means of EOAE and ABR.

Materials and methods

Subjects

Twenty-two males (44 ears) with bilateral sensorineural hearing loss entered the study. Their ages ranged between 37 and 64 years (mean: 51 years). They had been exposed to noise in various types of industries: railways, electrical plant, metalwork, textile production, and paper manufacture. The equivalent continuous noise level being more than 85 dB (A) during a great deal of their working time. The duration of the individual noise exposure varied between 10 and 40 years (mean: 26 years). There was no history of exposure to ototoxic drugs, there was no chronic ear disease nor family history of hereditary deafness, and there were no radiological signs of retrocochlear involvement. All patients exposed to noise presented a pure sensorineural hearing loss as confirmed by pure-tone audiometry. Based upon hearing losses at 4, 3, 2, and 1 kHz ($\geq$25 dB HL), patients were classified into four groups: Group 1 (hearing loss only at 4 kHz) consisted of four patients (eight ears) with an average age of 39 years, Group 2 (hearing loss at 3 and 4 kHz) consisted of seven patients (14 ears) with an average age of 42 years, Group 3 (hearing loss at 2, 3, and 4 kHz) consisted of five patients (10 ears) with average age of 51 years, and Group IV (hearing loss at 1, 2, 3 and 4 kHz) of six patients (12 ears) with average age of 55 years.

Twenty-one normal subjects (all males, 42 ears) with average age of 41 years were used as controls. They consisted of three age subgroups; Group A consisted of eight normal subjects (16 ears) with ages from 26 to 40 years (mean: 37 years), Group B consisted of six normal subjects (12 ears) with ages from 41 to 45 years (mean: 43 years), and Group C of seven normal subjects (14 ears) with ages from 46 to 55 years (mean: 53).

Pure-tone audiometric (PTA) thresholds at 500, 1000, 2000, 3000, 4000, 8000 Hz were measured in all subjects using an Interacoustics AC5 clinical audiometer.

All subjects had a normal otoscopic examination. Middle-ear function in all subjects (control group and patient group) was evaluated through a set of immittance measurements with the Grason-Stadler GSR-33 middle-ear analyser (version II). Type A tympanograms were found, and ipsilateral acoustic reflexes were elicited at 80 to 100 dB HL at 500 and 1000 Hz. The contralateral acoustic reflexes were elicited at 90 to 110 dB HL at 500 and 1000 Hz.

Otoacoustic emissions

TEOAE were recorded using the ILO 88 otoacoustic emission analyser (V 4.2) with a nonlinear click stimulus of 80 μs electrical duration, presented at a repetition rate of 50 Hz and a sound pressure level (SPL) of 85 ± 5 dB. Each response was recorded using 260 sweeps. A foam tip was used to seal the probe in the external ear canal, and a stable stimulus was present without excessive ringing.

The IL088 system provides a number of useful descriptions of the averaged waveforms. TEOAE level was defined by averaging into two separate buffers (A and B). The level of the residual noise is provided by subtracting one waveform from the other. The extent to which the two waveforms are correlated is expressed in a total reproducibility percentage.

Off-line data analysis was performed on broadband waveforms and octave bands, centred at 1000, 2000, 3000, and 4000 Hz. Three parameters (TEOAE-noise, reproducibility, and TEOAE/noise ratio) were used. The TEOAE-noise parameter was calculated by subtracting the noise level from the TEOAE level for broadband signals. Reproducibility (percentage) was registered for broadband and octave band emissions. The TEOAE/noise ratio was computed for different frequency bands. If the TEOAE energy was 3 dB greater than the noise level, the reproducibility was greater than 50 per cent (Prasher *et al.*, 1995), and if the TEOAE-to-noise ratio (SNR) was greater than 2 dB, the emission was considered to be present.

The otoacoustic emission criteria were classified into three categories: normal, weak, and absent emissions. The normal region is mean ± SD. The weak emission is between the lower limit of the normal region and the lower limit of the present emission.

Auditory brainstem response

ABR waveforms were obtained at stimulation rates of 21.3/s with 80 dB nHL and 90 dB nHL unfiltered alternating clicks in the control group and 90 dB nHL unfiltered alternating clicks in the patients. The analog Butterworth filter was set to 200 and 1500 Hz, and the window time was set to 10 ms. Amplification and averaging were performed using a Nicolet Pathfinder I.

The view switch was set to see raw noise input. All subjects had a high degree of muscular relaxation as reflected by the peak-to-peak sweep amplitude which was within screen boundaries.

ABR value criteria were:

(a) I/V amplitude ratio; this was classified into three possible outcomes; normal ratio (mean ± SD), small ratio (below the normal region), and zero ratio.
(b) I-V interpeak latency; this could be normal (mean ± 2 SD) and prolonged (beyond the normal region).
(c) Wave V absolute latency value; this was corrected for the hearing loss according to Prosser's ΔV index. The ΔV index was

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TABLE I

SUMMARY OF THE MEAN AND SD PARAMETERS OF THE PURE-TONE THRESHOLD (AVERAGE 0.5–8 KHZ), AUDITORY BRAINSTEM RESPONSES, AND TRANSIENT-EVOKED OTOACOUSTIC EMISSIONS IN CONTROL GROUP A, B, AND C

Normal subjects Parameters		Control group Group A	Group B	Group C	ANOVA F	*P*-value
PTA 0.5–8 kHz (dB)	Mean	9.4	10.1	11.0	2.83	0.07
	± SD	2.4	2.3	2.6		
I/V Ratio (μv)	Mean	0.60	0.59	0.58	0.07	0.92
	± 1 SD	0.16	0.13	0.11		
I–V IPL (ms)	Mean	4.08	4.09	4.10	0.52	0.59
	± 2 SD	0.24	0.24	0.25		
V Latency (ms)	Mean	5.51	5.52	5.53	0.11	0.89
	± 2 SD	0.22	0.22	0.21		
Total TEOAE (dB)	Mean	12.9	12.5	11.6	0.42	0.65
	± 1 SD	3.82	3.79	3.70		
Total Repro (dB)	Mean	94.5	94.0	93.0	0.40	0.67
	± 1 SD	2.96	2.93	2.82		

		Octave band emissions							
		1 kHz		2 kHz		3 kHz		4 kHz	
Control group		SNR dB	Repr %	SNR dB	Repr %	SNR dB	Repr %	SNR dB	Repr %
Group A	Mean	11.8	93.5	15.4	95.9	14.4	94.2	11.3	89.4
	± 1 SD	2.1	2.5	4.7	2.6	4.3	4.5	4.8	6.5
Group B	Mean	12.1	93.4	15.6	95.0	14.0	94.4	11.1	90.4
	± 1 SD	2.0	2.6	4.2	2.1	4.2	4.2	4.5	5.3
Group C	Mean	11.8	93.9	15.2	95.9	14.2	94.6	11.1	90.2
	± 1 SD	2.2	2.5	4.3	2.7	4.5	4.6	4.6	6.1

ANOVA = Analysis of variance: single factor
PTA = Pure-tone threshold average
TEOAE = Transient-evoked otoacoustic emissions
Repro = Reproducibility
p-value>0.05 N.S.

calculated according to the formula: ΔV = Lp(90)–Ln(90–x) where Lp(90) represents the wave V latency of the pathological ear at 90 dB nHL, Ln(90–x) represents the wave V latency as predicted from a normal intensity-latency function, and x represents the patient's pure-tone hearing loss as an average of the frequencies 2 and 4 kHz (Prosser and Arslan, 1987). The wave V absolute latency (ΔV index) was positive or negative.

Results

Table I presents the mean and SD parameters of the pure-tone threshold average (PTA), ABR, and TEOAE in the 21 normal subject sample, who were classified into three age groups (A: 26–40 years, B: 41–45 years and C: 46–55 years). The F test (Anova: single-factor) was performed to determine if the age effects of PTA, ABR and, total TEOAE, and (ABR). Our results are summarized in Table I. Although the mean data revealed slight differences with increasing age, our statistical data showed no significant age effects of pure-tone thresholds average, total TEOAE and ABR in our normal sample. All control groups (A, B and C) show 100 per cent presence of emissions in the broadband and frequency-band, and all normal subjects have the wave I of ABR.

Figure 1 shows the mean values of pure-tone audiometric thresholds of the normal subjects (Groups A, B, and C) and the four groups of patients with noise-induced permanent hearing loss. Table II shows the mean, standard deviation, and range of pure-tone thresholds in the control group (A, B, and C) and four groups of patients exposed to different noise times.

Figure 2 illustrates a typical TEOAE and ABR recording in one patient with noise-induced permanent hearing loss in group III. This patient had no significant level of emissions with respect to the noise level, and the ABR waveform had no wave I and a normal wave V peak latency.

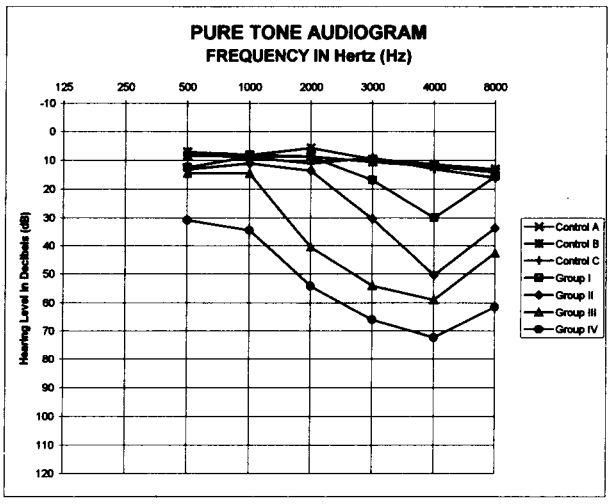

FIG. 1

The mean values of pure-tone audiometric thresholds of the control group (A, B, and C) and the four groups of patients with noise-induced permanent hearing loss (I, II, III, and IV).

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TABLE II

PURE-TONE THRESHOLDS AT 0.5, 1, 2, 3, 4 AND 8 KHZ IN CONTROL GROUPS (A, B AND C) AND 4 GROUPS OF PATIENTS EXPOSURE TO NOISE (I, II, III, AND IV)

Noise exposure times (years)		0.5 kHz (dB)	1 kHz (dB)	2 kHz (dB)	3 kHz (dB)	4 kHz (dB)	8 kHz (dB)
Control group A	Mean	8.4	8.2	5.8	9.6	11.4	13.0
Average 0	SD	2.1	2.0	1.8	2.9	3.8	4.3
	Range	5–15	5–15	5–10	5–10	5–20	5–20
Control group B	Mean	7.1	8.1	8.6	10.6	12.1	14.1
Average 0	SD	2.3	2.1	2.5	2.9	4.1	4.8
	Range	5–10	5–15	5–10	5–10	5–15	5–25
Control group C	Mean	8.0	9.0	11.1	9.1	13.0	16.0
Average 0	SD	2.3	3.1	2.5	2.8	4.2	5.3
	Range	5–10	5–15	5–10	5–10	5–25	5–30
Group I	Mean	12.5	8.8	8.8	16.9	30.0	15.6
Average 15	SD	2.7	2.3	2.3	2.6	7.1	5.6
	Range	10–15	5–10	5–10	15–20	25–40	10–25
Group II	Mean	13.2	11.1	13.6	30.4	50.4	33.6
Average 23	SD	3.2	2.1	3.6	7.7	13.8	17.7
	Range	10–20	10–15	10–20	24–55	40–95	10–80
Group III	Mean	14.5	14.5	40.5	54.0	59.0	42.5
Average 29	SD	2.8	3.7	8.6	7.7	11.7	15.3
	Range	10–20	10–20	25–50	40–65	40–80	20–60
Group IV	Mean	30.9	34.5	54.1	65.9	72.3	61.4
Average 37	SD	7.4	6.5	21.9	18.7	15.2	10.7
	Range	20–40	25–45	30–90	45–95	55–95	45–80

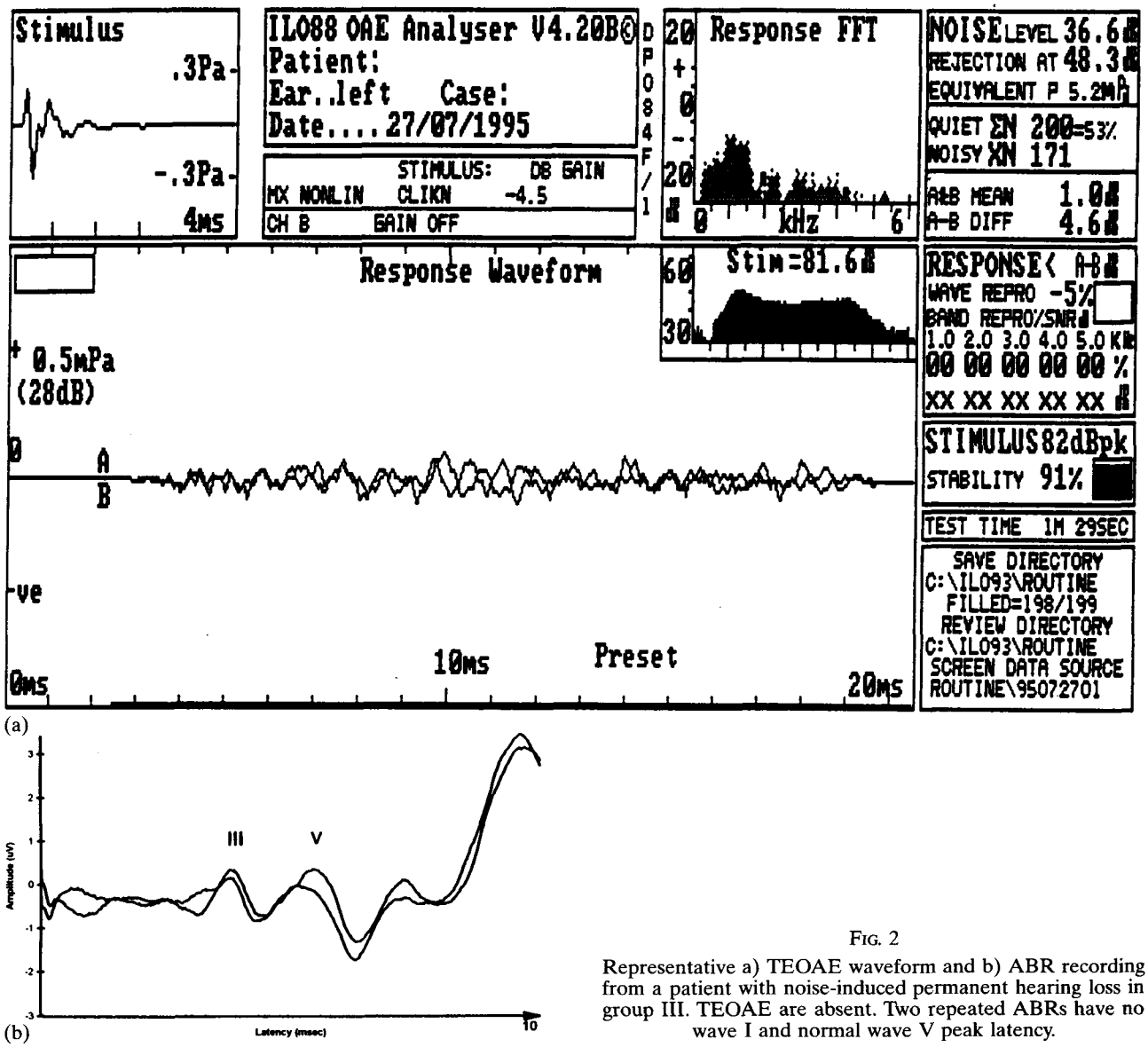

FIG. 2

Representative a) TEOAE waveform and b) ABR recording from a patient with noise-induced permanent hearing loss in group III. TEOAE are absent. Two repeated ABRs have no wave I and normal wave V peak latency.

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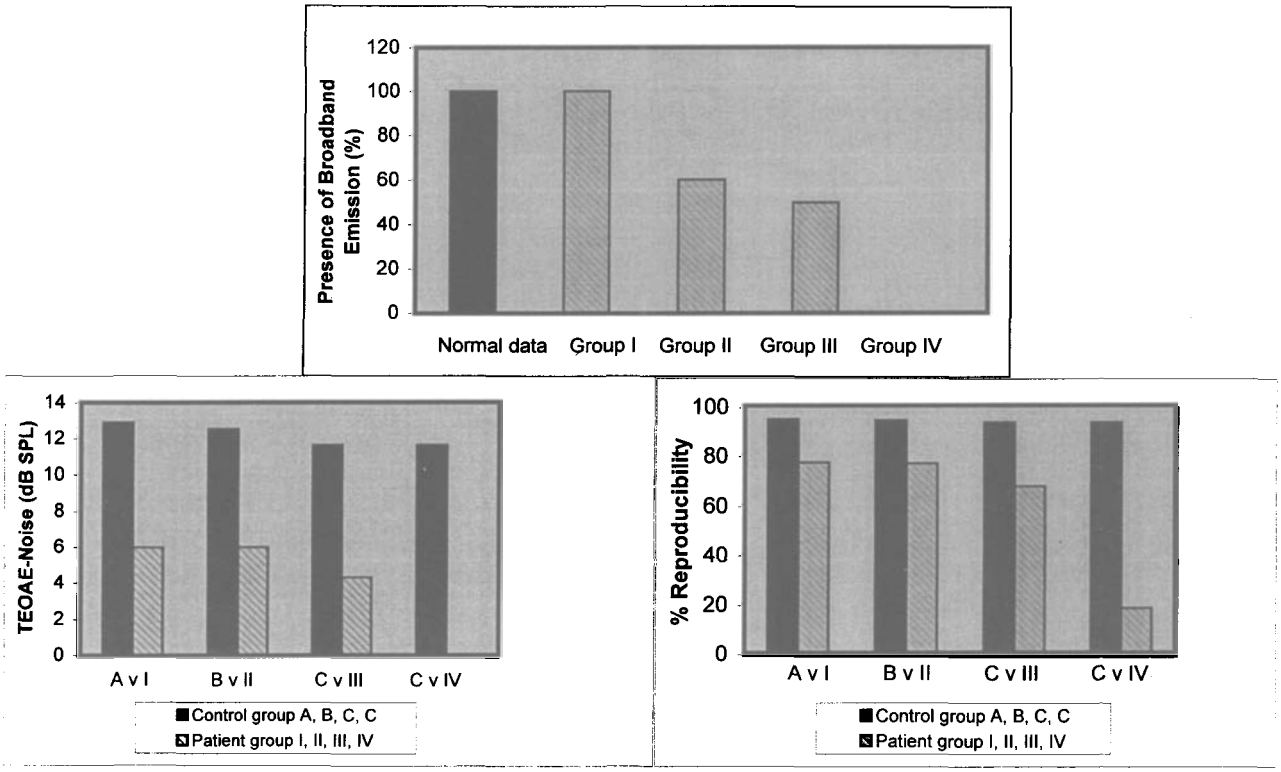

FIG. 3

Analysis of the broadband TEOAE waveform in each control group (A, B, and C) and appropriate age-related patient group (I, II, III, IV) a) presence of broadband emission, b) mean total TEOAE-noise, and c) mean total percent reproducibility.

Analysis of the broadband TEOAE waveform (presence of broadband emissions, TEOAE-noise, and reproducibility percentage) in each patient group (I, II, III, and IV) and appropriate age related control groups (A, B, and C) are described in Figure 3. The octave band emissions (presence of octave band emissions, SNR, and percentage reproducibility), centred at 1000, 2000, 3000, and 4000 Hz, are shown in Table IV. The presence of TEOAE was judged by three independent audiologists. An increase in the hearing loss from Group I to Group IV showed a decrease in presence of broadband emissions. As far as the analysis of the frequency-specific emissions is concerned, a slight correlation between the presence of octave band emissions and the behavioural frequency-specific thresholds was noted (Table IV).

In addition, quantitative analysis of TEOAE waveforms was performed. The total TEOAE-noise (noise level subtracted from the TEOAE level) and total reproducibility percentage was already significantly weak in all cases in Group I ($p<0.005$, t-test), whereas the sample ages showed no significant difference between control Group A (average 37 years) and Group I (average 39 years) (Table III). There were no significant differences of TEOAE-noise and total percentage reproducibility

TABLE III

EACH SUBGROUP OF PATIENTS (I, II, III, AND IV) COMPARED WITH APPROPRIATE AGE-RELATED SUBGROUP OF NORMAL SUBJECTS (A, B, AND C) FOR TOTAL TEOAE, TOTAL REPRODUCIBILITY, I/V AMPLITUDE RATIO, I–V INTERPEAK LATENCY AND V LATENCY

Control group (years) V Patient group (years)	Ages p-value	Total TEOAE p-value	Total Repro p-value	I/V ratio p-value	I–V IPL Patient subgroup	V latency Patient subgroup
A v I (mean 37 v 39)	N.S.	<0.005	<0.005	>0.1	WN range	WN range
B v II (mean 43 v 42)	N.S.	<0.005	<0.005	<0.001	WN range	WN range
C v III (mean 53 v 51)	N.S.	<0.005	<0.005	<0.001	WN range	WN range
C v IV (mean 53 v 55)	N.S.	<0.001	<0.001	<0.0005	WN range	2 ears: PI

TEOAE = Transient-evoked otoacoustic emissions
Repro = Reproducibility
IPI = Interpeak latency
V = Versus
WN = Within normal range (mean ± 2 SD)
PI = Positive ΔV index
p-value>0.05 N.S. (t-tests)

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TABLE IV
OCTAVEBAND EMISSIONS IN PATIENTS WITH NOISE-INDUCED PERMANENT HEARING-LOSS CLASSIFIED IN FOUR GROUPS

Octaveband emission data			Group I (8 ears)		Group II (14 ears)		Group III (10 ears)		Group IV (12 ears)
1 kHz	Presence of TEOAE (%)		100%		60%		50%		0%
	SNR (dB)	Mean	6.5		8.3	–1.2*	5.3	–2*	0*
		Minimum	3.0		3.0	–2	3.0	–5	–2
		Maximum	10.0		11.0	0	11.0	0	1
	Repro (%)	Mean	79.3		83.3	12*	76.3	0*	24.0*
		Minimum	56.0		50.0	0	66.0	0	0
		Maximum	92.0		92.0	40	90.0	0	49.0
2 kHz	Presence of TEOAE (%)		100%		60%		20%		0%
	SNR (dB)	Mean	8.8		7.2	–2*	4.5	–1*	–0.5*
		Minimum	3.0		3.0	–3	4.0	–3	–5
		Maximum	12.0		13.0	2	5.0	0	0
	Repro (%)	Mean	86.1		80.5	0*	72.5	0*	0*
		Minimum	64.0		55.0	0	70.0	0	0
		Maximum	93.0		95.0	0	75.0	0	0
3 kHz	Presence of TEOAE (%)		50%		21.5%		0%		0%
	SNR (dB)	Mean	4.2	–1*	3.6	–1.3*	–1*		0*
		Minimum	2.0	–3	2.0	–4	–4		0
		Maximum	9.0	1	6.0	0	0		0
	Repro (%)	Mean	68.7	11*	69.6	3*	4*		0*
		Minimum	53.0	0	57.0	0	0		0
		Maximum	89.0	46	79.0	37	40		0
4 kHz	Presence of TEOAE (%)		12.5%		0%		0%		0%
	SNR (dB)	Mean	5.0	–0.4*	–1.4*		0.6%		–1*
		Minimum	5.0	–2	–4		–2		–5
		Maximum	5.0	0	0		0		0
	Repro (%)	Mean	79.0	0*	0*		0*		0*
		Minimum	79.0	0	0		0		0
		Maximum	79.0	0	0		0		0

SNR = Signal (TEOAE)-to-noise ratio
Repro = Reproducibility
* = Mean values in the ears with absent TEOAE

between Group I and Group II ($p>0.05$, t-test). From Group III on they became even weaker ($p<0.01$, t-test) (Figure 3).

The quantitative analysis of the different frequency bands are listed in Table IV. For the patients with hearing loss only at 4 kHz (Group I), the data for signal to noise ratio (SNR) and reproducibility percentage appeared to be different at the different frequencies. In Group II with hearing loss at 3 and 4 kHz, there was no emission for the 4000 Hz band. For the 3000, 2000, and 1000 Hz bands, the mean SNR value in this group ranged from 3.6 to 8.3 dB SPL, and mean reproducibility ranged from 69.6 per cent to 83.3 per cent. As far as Group III was concerned, the absent emissions occurred at 4000 and 3000 Hz bands. For the 2000 and 1000 Hz bands, the mean SNR ranged from 4.5 to 5.3 dB SPL, and mean reproducibility ranged from 72.5 per cent to 76.3 per cent. For patients with hearing losses at all frequencies (4, 3, 2 and 1 kHz) in Group IV, there were no emissions at any of the frequency bands.

Figure 4 shows the presence of wave I and the I/V amplitude ratio of the ABR data in each patient Group (I, II, III, IV) and appropriate age related to each control Group (A, B, and C). The presence of wave I was 100 per cent in Group I and II, and decreased with increasing hearing loss.

The I/V amplitude ratio in all ears with a slight hearing loss (Group I) showed no significant change in comparison to the normal value of I/V amplitude ratio in control Group A (Table III). With increasing hearing loss, the I/V amplitude ratio became significantly smaller ($p<0.001$ between control Group B or Group I and Group II, $p<0.05$ between Group II and III, $p<0.05$ between Group III and IV; t-test) in these cases where wave I was present (Figure 4). The I-V interpeak latencies fell within the normal range (M ± 2SD) in all cases (Table III). However, two ears with severe hearing loss (Group IV) at 2 and 4 kHz (average 75 dB), in which wave I was absent, revealed a positive ΔV index, calculated by comparing the latency values of wave V obtained in normal-hearing subjects (Figure 5). In all other ears, the ΔV index was negative. The ΔV index was calculated from all normal subjects and all patients, there were no significant differences between the two groups (with respect to age (t-test, $p>0.05$).

Discussion

Up to now, the effect of noise on the ear of animals has been studied by means of light microscopy, scanning electronmicroscopy, and immune histochemistry. The pathological process seems to start as outer hair cell damage which is progressively followed by damage of the inner hair cell and later of the cochlear neurons (Saunders and Rhyne, 1970). Morest and Bohne (1983) reported that the auditory nerve as well as the central auditory nervous system may be damaged too. Human material for histopathological examination is difficult to obtain.

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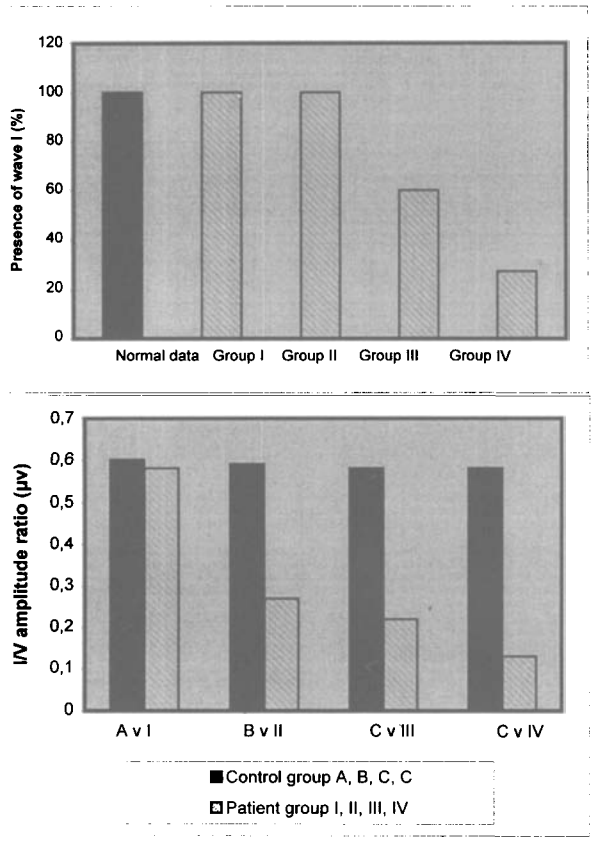

FIG. 4

a) presence of wave I in normal control and four groups of patients with noise induced hearing loss, b) mean I/V amplitude ratio with presence of wave I in each control group (A, B, and C) and appropriate age-related patient group (I, II, III, IV).

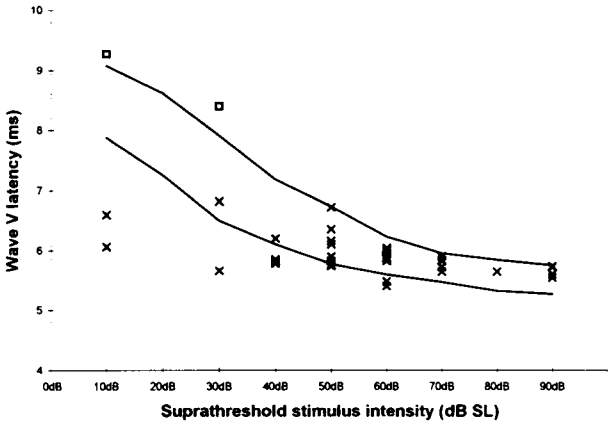

FIG. 5

Wave V latencies in relation to the suprathreshold stimulus intensity (dB SL) in all patients with noise-induced permanent hearing losses (positive ΔV and × negative ΔV). Continuous lines represent the normal range of variation for total control groups. The square symbols indicate a positive delta index for two ears with severe hearing loss at high frequency (2–4 kHz; average 75 dB) in group IV.

In the present study, patients with noise-induced permanent hearing loss were examined using mechanical and electrophysiological tests. The behavioural thresholds at 1000, 2000, 3000 and 4000 Hz were compared to ABR and TEOAE. Our results revealed that the total TEOAE-noise and percentage reproducibility were already weak in patients with a mild noise-induced hearing loss only at 4 kHz, while I/V amplitude ratios, latency values of V, and I–V intervals still fell within the normal range. This demonstrates that damage from exposure to noise first takes place in the outer hair cells. This is in agreement with the pathological findings in animals, as was reported by Lim (1986). It also illustrates that the measurement of TEOAE is a sensitive method to detect outer hair cell damage.

For hearing losses extending to 3 kHz, not only TEOAE-noise and reproducibility percentage became weak, but in addition the I/V amplitude ratios in the ABR became small. Wave I in the ABR serves as an indicator of the peripheral auditory output, whereas wave V in the ABR represents brainstem activity. When placing a recording electrode closer to the cochlear neuron or distal auditory nerve, the wave I greatly increases the probability of observing this potential. These data also revealed that type I spiral ganglion cells (cochlear neuron) in the rat are responsible for generation and propagation of the auditory nerve action potentials (Moore *et al.*, 1996). The identification of wave I can give interesting information about the cochlear function. In high-frequency cochlear impairment, wave I is often absent while wave V peak latency is normal (Musiek *et al.*, 1984). The finding of reduced amplitude ratios may be useful in differentiating cochlear from retrocochlear pathology. Indeed Museik *et al.* (1984) reported patients (with cochlear lesions) who showed normal wave V latency results but with wave I amplitude values that were significantly smaller than those of wave V. When I/V amplitude ratios become small, this probably indicates patients with cochlear lesions (cochlear neuron).

With increasing hearing loss (Group III), the TEOAE-noise and percentage producibility became much weaker, and also the I/V amplitude ratio became much smaller. In the worst cases (Group IV), two ears with severe hearing loss at 2 and 4 kHz (average 75 dB) revealed a positive ΔV index, which probably indicated the more damaged cochlear function including cochlear neurons at high frequency. Prosser's ΔV takes into consideration the latency of wave V in the pathologic ear for a 90 dB nHL click as a function of sensation level for pure tone thresholds in the 2 to 4 kHz range, as compared with wave V latency for a normal ear (Prosser and Arslan, 1987).

Almadori *et al.* (1988) reported that the use of Prosser's ΔV index did not reveal a positive ΔV index in any case with noise-induced permanent hearing loss. In their study, however, none of the ears suffered from a severe hearing loss of more than 70 dB, which may explain the absence of a positive ΔV index in their series.

There is general agreement that hearing ability decreases in relation to duration of noise exposure (Rop *et al.*, 1979). Our data showed clearly that hearing loss decreases with increasing noise expo-

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sure times in exposed patients, the equivalent continuous noise level being more than 85 dB (A) (Table II).

The patient group with ages ranging between 37 and 64 years (mean: 51 years) showed a hearing loss caused by the degenerative changes of aging (presbycusis). In order to exclude the effect of age on hearing disability in the patient group, the normal subject group selected was of a similar age distribution. The 21 normal subjects sample was classified into three age groups (A: 26–40 years, B: 41–45 years and C: 46–55 years). We found that the mean data revealed a slight difference with increasing ages but the F-test (Anova: single-factor) showed no significant age effects of PTA, total TEOAE and ABR in our normal sample. In order to further exclude the age effect in the patient group, each subgroup of patients was compared with the appropriate age-related control subgroup (Table III). We found that the total TEOAE-noise and total reproducibility percentage in Group I when compared to data from the control group A revealed a significant change ($p<0.005$, t-test), whereas the sample ages showed no significant difference between control Group A (average 37 years) and Group I (average 39 years) ($p>0.05$, t-test). A comparison of the I/V amplitude ratio revealed a significant difference between control Group B and group II ($p<0.001$ t-test), whereas the sample ages of the two groups (average 43 years, average 42 years) showed no significant difference ($p>0.05$, t-test). In addition, presbycusis presents a slightly or moderately linear gradually descending pure-tone threshold, whereas all patients in our study had a typical 4 kHz notch as confirmed by pure-tone audiometry (Figure 1). From these data, we confirm that the hearing losses in our patients are due to noise alone.

In our study progressive damage from outer hair cells to cochlear neuron with growth and extent of the hearing loss was found with the help of TEOAE and ABR techniques. This method can detect a dynamic pathological process after long-term noise exposure in patients with permanent hearing loss.

With increasing hearing losses and extension of the involvement from 4 to 1 kHz in pure-tone audiometry, the objective TEOAE figures (presence of TEOAE, TEOAE-noise, percentage reproducibility and SNR) became lower and the objective ABR data (presence of wave I and I/V amplitude ratios) showed lower figures.

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