The Latency of Distortion Product Otoacoustic Emissions in Ears with Hearing Impairment

Junghak Lee* · Soojin Cho · Jinsook Kim*****

ABSTRACT

Distortion Product Otoacoustic Emissions (DPOAEs) can be measured in the external ear canal two fold: amplitude and latency, but most DPOAE studies deal with amplitude aspects. The purpose of this study was to investigate the latency of the 2f1-f2 DPOAEs in ears with hearing losses and to see if it could be a clinically useful method to distinguish normal from abnormal ears. For this purpose, DPOAE latency were measured as a function of frequency from 1 to 8 kHz in 30 ears with conductive and sensorineural hearing losses (SNHLs). DPOAEs were recorded with Otodynamic Analyzer ILO92. Results showed that the latency decreased as the frequency increased up to 8 kHz. The mean values of DPOAE latency for ears of SNHLs were shorter at all frequencies when they were compared to the mean values of normal ears. The latency in ears of conductive hearing losses was shorter than normal ears at the selective frequencies, as well. The results support the hypothesis that latency values are shorter in pathological ears.

Keywords : DPOAEs, DPOAE latency, Conductive hearing loss, SNHL.

1. INTRODUCTION

Otoacoustic emissions (OAEs) first described in 1978 (Kemp) are responses in the form of acoustic energy detected in the external ear canal. Although studies at present have not definitively demonstrated the source of OAEs, it is widely accepted that OAEs are produced by the movement of outer hair cells in the cochlea. There are two general categories of OAEs: Spontaneous OAEs (SOAEs) and stimulus evoked OAEs. The latter is further subclassified on the basis of the kind of stimulus used such as transient evoked OAEs (TEOAEs), stimulus frequency OAEs (SFOAEs) and distortion product OAEs (DPOAEs). Using especially TEOAEs and DPOAEs, as the objective and

* Dept. of Otolaryngology, Hallym University
** Dept. of Otolaryngology, Inha University
*** Graduate School of Social Welfare, Hallym University

noninvasive methods representing cochlear function, the clinical applications continue to be identified for screening of hearing loss and monitoring subtle cochlear changes during administration of ototoxic drugs or from noise exposure (Lonsbury-Martin & Martin, 1990).

Much of the interest in the clinical uses of DPOAEs centers around three perceived advantages of DPOAEs over other types of OAEs. The first is that unlike TEOAEs, DPOAEs are frequency specific (O'Mahoney, 1993). Regarding the stimulus, TEOAE applies a short duration click which contains all frequencies and stimulates the entire cochlea. For DPOAEs, the stimuli are two discrete pure tones of different frequencies (f1 and f2). Because each frequency has its "characteristic place", there are two areas on the basilar membrane that are maximally stimulated. However, it does not necessarily follow that the areas maximally stimulated are the areas generating the DPOAEs. According to Brown and Kemp (1984), the site of generation is near the f2 frequency place on the basilar membrane, but other workers (Martin et al., 1987; Probst & Hauser, 1990) have assumed that the site of generation is at the geometric mean of f1 and f2. Nevertheless, the implication is that each part of the cochlea can be characterized by looking at the amplitude of DPOAEs and that an "objective audiogram" can be constructed. Indeed, there appear to be good correlation between the DPOAE amplitude and the pure tone audiogram (Probst & Hauser, 1990). The second is that they can be elicited from cochlea with a greater degree of hearing losses. While TEOAEs are not usually detected in ears with losses greater than 30 dBHL, DPOAEs are detectable in ears with losses up to 55 dBHL. A possible explanation for this could be that the stimulus to elicit the DPOAE is a "stronger" stimulus. Further, DPOAEs can be recorded in ears with middle ear dysfunction such as perforated tympanic membrane or otitis media with effusion depending on the position and size of the perforation and the severity of the effusion (Ueda, et al., 1998). The third is that DPOAEs can be recorded from all normal ears.

DPOAEs are cochlear emissions that result from the simultaneous presentation of two pure tones (f1 and f2). Among different DPOAE components such as 2f1-f2, 3f1-2f2, and 2f2-f1, the 2f1-f2 distortion product is usually greatest in amplitude of the DPOAEs measurable in the external ear canal (Probst, 1990). The 2f1-f2 DPOAEs have properties of latency and amplitude. Both are believed to be conferred by some aspect of cochlear mechanical function. But, thus far, almost all of the reported work on DPOAEs deals with aspects of the amplitude.

Kemp and Brown (1983) suggested that DPOAE latency, taken with amplitude, would offer further valuable information about cochlear processes such as the travelling wave propagation along the cochlea. The latency represents the time delay between the stimuli being delivered to the ear canal and the DPOAE being detected

back in the canal. The phase gradient method has already been used to evaluate DPOAE latency. In order to measure DPOAE latency through the phase gradient method, a small change of f2 which will increase the frequency of the generated DPOAE is applied as illustrated in Figure 1. Because the frequency of the generated DPOAE depends on the stimulating frequencies, DPOAEs of slightly different

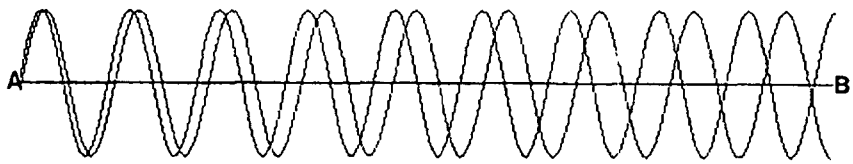

Figure 1. Demonstration of the principle of the "Phase Gradient Method" to measure time delay (latency) of a wave. Two waves of slightly different frequencies start in phase, at point A. By the time they reach B they are out of phase by an amount proportional to the frequency difference of the two waves.

frequencies, i.e. different phase are generated. This phase difference can be calculated as the time taken, i.e. latency, according to the following formula.

Time(latency) = change in phase angle in radians/
change in frequency in radians per sec

but $\omega = 2\Pi f$,

where ω is frequency in radians/sec and f in cycles/sec.

When radians are converted to degrees, the above equation becomes;

Time = change in phase angle in degrees/360° /change in f (Hz)

Therefore, time can be calculated because the change in angle can be measured and change in frequency is known (O'Mahoney, 1993).

When DOPAE latency was tested as a potential clinical tool, Lee et al. (1997) found that the latency was decreased as frequency increased up to 6 kHz with high test reliability in 4-6 kHz for 38 normal ears. This latency pattern could be altered in a pathological ear due to the affected travelling wave pattern. Experimentally, the research on the DPOAE latencies of the damaged ears was attempted once with 11 ears as a minor part of the whole research but the DPOAE latencies were successfully recorded in only 4 ears (O'Mahoney, 1993). The limited data showed tentatively different DPOAE latency from the normal ears. Considering the lack of energy input by the pathological outer hair cells, the latency could be shorter for a given frequency in a pathological cochlea. Because the energy of the wave is absorbed more basally, the stimuli would not travel as far on the basilar membrane. Toward

this end, the present investigation is aimed at gaining a better understanding of DPOAE latencies in the hearing impaired, conductive and sensorineural hearing losses.

2. METHODS

Subjects

30 ears, with mild to moderate hearing losses, were tested. They include 10 ears of conductive hearing losses (Female:5, Male:5) and 20 ears of sensorineural hearing losses (Female:10, Male:10). The age range of subjects was 28 to 58. For the ears with conductive hearing losses, air conduction hearing thresholds were better than 50 dBHL at the standard audiometric frequencies between 0.25 and 8.0 kHz. The differences between air and bone conduction hearing thresholds were 10 dBHL or more and the tympanograms were B or C type indicating middle ear dysfunction. For the ears with sensorineural hearing losses, they also had air conduction hearing thresholds better than 50 dBHL and the differences between air and bone conduction hearing thresholds were 10 dBHL or less at the standard audiometric frequencies between 0.25 and 8.0 kHz. The tympanogram was A type and the normal tympanic membrane was confirmed through the otoscopic examination showing normal middle ear function.

Materials

DPOAE latency was measured using the Otodynamics ILO92 OAE systems. This equipment was placed in a sound isolated booth controlling ambient noise lower than 30 dBA and latency measurements were performed using the same equipment in this room. An ILO-B type probe with three ports-2 loudspeakers and 1 microphone- was inserted in the external ear canal. The two stimulating tones (f1 and f2) were delivered via probe speakers to the external ear canal and the resulting sound field was detected by the probe microphone. Air and bone conduction pure tone audiometry was performed using Madsen Orbiter 922 audiometer in the sound isolated booth. Tympanometry was operated using Madsen Zodiac 901 middle ear analyser.

Procedures

Subjects were seated in the sound isolated room and examined bilaterally with an otoscope to determine the presence of cerumen or tympanic membrane perforation. Pure tone audiometry and immittance were conducted consequently. The probe was housed in the disposable and pliable foam eartip and fitted in the individual ear. The value of the ear canal volume, shown just beside the "Checkfit" curve while testing, was observed for the stable probe fitting.

For the DPOAE latency measurements, the two continuous pure tones at f1 and f2 were generated separately and delivered through two separate transducers within the external canal. A microphone collects cochlear emissions. Latency calculations were performed with the ILO92 'latency' protocol, which obtained phase measures of 2f1-f2 with fixed f1 and f2 increasing in four small frequency steps. The f2 frequency was dependent on the f1 frequency and varies at 12 Hz intervals for low frequencies around 1,000 Hz and at 48 Hz for high frequencies around 8,000 Hz. Each f2 frequency sweep average was based on 48 time samples, with epochs of 13 sec. Following the accumulation of the average, a Fast Fourier Transform (FFT) analysis was performed to examine the amplitude/phase properties of the DPOAE. A simple linear-regression function was computed for the slope of the DPOAE-phase response as a function of frequency in order to derive DPOAE latency.

For the strongest response, the level of the primaries were set around 75 dBSPL for f1 and 65 dBSPL for f2 and the f2/f1 frequency ratio was ranged from around 1.20 to 1.24 (Gaskill & Brown, 1990). Noisy data above 8 mPa were rejected. The presence of a DPOAE was determined automatically using the criterion inherent in the ILO92 system, i.e. it was necessary for a response to be 0 dB above the mean noise +2 standard deviation in order to be a DPOAE.

3. RESULTS

The 2f1-f2 DPOAE latency values decreased as the frequency increased from 1 to 8 kHz in the ears with sensorineural hearing losses. The standard deviation was the highest at 1 kHz and the lowest at 6 kHz indicating the highest and lowest variability, respectively (Table 1). The DPOAE latency of the male subjects were longer than that of the female subjects in frequencies between 1 and 8 kHz except 4 kHz as illustrated in figure 2. However, the gender effect was not statistically significant (unpaired t-test: $p>.05$).

Compared to the normal data of Lee et al. (1997), because of the same recording parameter and the similar subject selection, the latency for the ears with sensorineural hearing losses were systematically shorter in all frequencies. With 38 normal ears, the authors reported the DPOAE latencies 10.70, 7.74, 5.10, 3.18, 3.28 ms at 1, 2, 4, 6, 8 kHz, respectively. These values were shortened in ears with the sensorineural hearing losses. It was calculated as 64, 70, 67, 85, 48% at 1, 2, 4; 6, 8 kHz, respectively revealing the largest shortened amount at 8 kHz.

Table 1. The average DPOAE latency in ears with sensorineural hearing losses as a function of frequency (kHz)

		Frequency(kHz)				
		1	2	4	6	8
Total	Mean	6.89	5.42	3.42	2.69	1.56
	SD	3.01	1.88	1.24	0.92	1.2
	Min	1.67	2.22	0.54	0.45	0.12
	Max	12.79	8.75	5.16	3.57	4.58
Female	Mean	6.48	4.95	3.49	2.46	1.45
	SD	3.06	1.71	1.33	0.87	1.07
Male	Mean	7.56	6.16	3.29	3.04	1.73
	SD	3.08	2.05	1.16	0.94	1.46

(SD : Standard deviation)

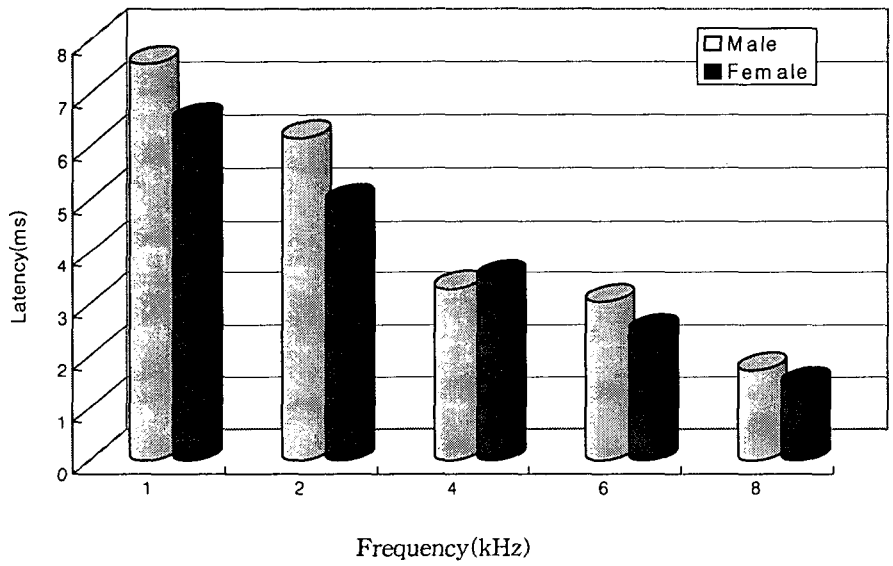

Figure 2. The DPOAE latencies of the female vs male subjects of sensorineural hearing losses as a function of frequency (kHz)

Out of 10 ears with conductive hearing losses, 4 ears at 1 kHz, 1 ear at 2 kHz, 2 ears at 6 kHz, and 4 ears at 8 kHz could not reach the criterion for the DPOAE latency response due to the middle ear status. The 2f1-f2 DPOAE latency values measured also decreased as the frequency increased. The standard deviation was the lowest at 8 kHz indicating the highest variability (Table 2). The gender effect could not be compared because the amount of the data was too small.

The latency for the ears with conductive hearing losses were shorter in all frequencies except 6 kHz, when compared to the normal data of Lee et al. (1997).

The latency values in ears with the conductive hearing losses were 60, 71, 88, 108, 15 % at 1, 2, 4, 6, 8 kHz, respectively. The latency was even longer in ears with conductive hearing losses at 6 kHz and was extremely short at 8 kHz. This irregularity of the latency decrease can be interpreted in two ways. One is that insufficient data were collected. The other is that the DPOAE is difficult to measure in ears with conductive hearing losses.

Table 2. The average DPOAE latency in ears with conductive hearing loss as a function of frequency (kHz)

	Frequency(kHz)				
	1	2	4	6	8
Mean	6.44	5.46	4.5	3.44	0.79
SD	2.8	2.8	1.9	1.37	0.67
Minimum	2.85	0.86	1.24	0.77	0.06
Maximum	9.37	10.3	7.46	5.13	1.42

(SD : Standard deviation)

When the latency was compared between the ears with sensorineural and conductive hearing losses, the result showed a similar pattern. Shorter DPOAE latencies were observed at 1 and 8 kHz and longer DPOAE latencies at 4 and 6 kHz for ears with the conductive hearing losses (Figure 3).

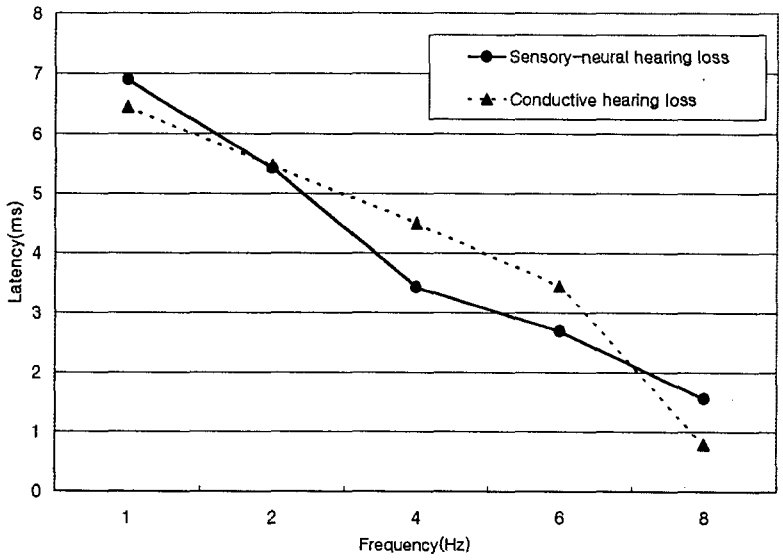

Figure 3. The DPOAE latency of conductive vs sensorineural hearing losses as a function of frequency (kHz)

4. DISCUSSION

Importantly, the 2f1-f2 DPOAE latency pattern for the hearing impaired appeared in the present study. 2f1-f2 DPOAE latency decreased when the stimulus frequency increased along the cochlear duct. Although our results are based on damaged ears, the latency pattern obtained from pathological ears is similar to that from the normal ears. The known distribution of frequencies could still be applied. That is the maximum excitation of the basilar membrane shifts from apical toward more basal regions as stimulus frequency increases.

However, as expected, our results were much shorter than the normal latency values of many studies (Kimberley et al., 1993; O'Mahoney & Kemp, 1995; Lee et al., 1997). As the DPOAE latency appears to relate more closely to the progression of the travelling wave along the cochlea, it seems reasonable to propose that the latency reflects an altered traveling wave pattern by the pathological ear. Among investigations of the normal latency values mentioned above, exhibited discrepancy can be explained by the different type of DPOAE latency measuring method used, by the difference in the level of primaries, and by differences in primary amplitude separation. In order to use DPOAE latency in the clinical setting, the parameter of latency recording needs to be further studied.

The results reported in the present work are consistent with those of O'Mahoney (1993). He reported the lower mean value of DPOAE latency in ears with cochlear pathology. The latency from the pathological ears was 66% that of the normals, with a range from 46 % to 91 %. The difference was statistically significant only at three frequencies. He added that the interpretation of the data must be guarded due to the small amount of data collected. The results of the present study were compared to the normal data of Lee et al. (1997), on the basis of the same recording parameter and similar subject selection. Latency for the ears with sensorineural hearing losses was 67 % ranging from 48 % to 85 % and 68 % ranging from 15 % to 108 % for the ears with conductive hearing losses. The percentages are all in good accordance for the ears with the cochlear pathology. However, when ears with conductive hearing loss are compared, special caution should be taken due to the different kind of pathology and insufficient data selection. More evidence needs to be gathered to support the impression that pathological ears differ in latency from normal ears and to reach the conclusion on the usefulness of applying DPOAE latency as a clinical tool.

Furthermore, in the present study, only 5 out of 10 ears could successfully record DPOAE latency at all experimented frequencies in the case of conductive hearing losses. This supports the findings of the studies of OAE amplitude. Because DPOAEs are transmitted from the cochlea to the ear canal via the middle ear, the transmission

properties of the middle ear directly influence the responses. Several studies have shown the influence of middle ear status on the amplitude of TEOAEs (Kemp et al., 1990; Veuillet et al., 1992; Naeve et al., 1992), SOAEs (Kemp, 1981), and DPOAEs (Hauser et al., 1993). In general, middle ear dysfunctions reduce measure emission amplitudes and sometimes eliminate the response entirely. Changes in OAE characteristics can be viewed in the following three features of middle ear functioning. First, the ear is a complex network with multiple pathways for the flow of energy. Energy that flows through the shunt elements does not reach the inner ear. This indicates that there is vibratory energy in the system that is not transmitted to the inner ear. Second, the system has an input impedance that represents the opposition offered by the total system to the flow of energy. Because the input impedance is influenced by components that are or are not in the pathway to the cochlea, the input impedance is a poor indicator of hearing sensitivity. Third, the middle ear can transmit sound bidirectionally and the forward and backward transmission characteristics are different. OAE characteristics are subject to the effects of forward transmission on the spectral content of the effective stimulus reaching the inner ear and the effect of the frequency response of the middle ear on OAE spectral characteristics (Robinette & Glattke, 1997).

Moulin & Kemp (1996) reported an increase of the DPOAE latency following both positive and negative pressure applied in the outer ear canal. The effect was greater at frequencies below 2 kHz. As varying the air pressure in the outer ear canal resulted in modifying the stiffness of the tympanic membrane and the mobility of the ossicular chain, an analogous influence could be expected for ears with conductive hearing loss. However, our results revealed a decrease of the DPOAE latency in ears with middle ear dysfunction. It was postulated that the change in the middle ear status might affect the amplitude of DPOAE directly but not the latency of DPOAE. Finally, caution needs to be exercised in drawing conclusion due to the limited data amount.

5. SUMMARY AND CONCLUSION

The primary goal of the present study was to explore the effects of hearing impaired, conductive and sensorineural hearing losses, on DPOAE latencies. 30 ears, 20 ears with mild to moderate sensorineural hearing loss and 10 ears with mild to moderate conductive hearing loss were examined using a phase gradient method. Similar to the normal DPOAE latency, the overall DPOAE latency pattern revealed a decrease in latency as the stimulus frequency increased. The latency values of the damaged ears were much shorter than those of the normal ears. The available data

supports the hypothesis that latency would be decreased in pathological ears. This certainly suggests that further investigation of this hypothesis is warranted.

REFERENCES

Gaskill, S. A. & A. M. Brown. 1990. "The behavior of the acoustic distortion product, 2f1-f2 from the human ear and its relation to auditory sensitivity." *Journal of Acoustical Society of America,* 88, 821-839.

Hauser, R., R. Probst. & F. P. Harris. 1993. "effects of atmospheric pressure variation on spontaneous, transiently evoked, and distortion product otoacoustic emissions in normal human ears." *Hearing Research.* 69, 133-145.

Kemp, D. T. 1978. "Stimulated acoustic emission from within the human auditory system." *Journal of Acoustical Society of America,* 64, 1386~1389.

Kemp, D. T. 1981. "Physiologically active cochlear microphone: a source of tinnitus." *in Tinnitus, CIBA Foundation Symposium.* 85, 54-81.

Kemp, D. T. & A. M. Brown. 1983. "A comparison of mechanical nonlinearity in the cochlea of man and gerbil from ear canal measurements." *Hearing : Physiological bases and psychophysics.* 82-88.

Kemp, D. T. & A. M. Brown. 1984. "Suppressibility of the $2f_1-f_2$ stimulated acoustic emissions in gerbil and man." *Hearing research.* 13, 29-37.

Kemp, D. T., S. R. Ryne. & P. Bray. 1990. "A guide to the effective use of acoustic emissions." *Hearing Research.* 2, 213-232.

Kimberley, B. P., D. K. Brown & J. J. Eggermont. 1993. "Measuring human cochlear traveling wave delay using distortion product emission phase response." *Journal of Acoustical Society of America,* 94, 1343~1350.

Lee, J. H., S. H. Lee, Y. B. Kim & M. S. Park. 1997. "The latency of distortion product otoacoustic emissions in normal ears." The Korean Journal of Otolaryngology, 40(3), 347-352.

Lonsbury-Martin, B. L. & G. K. Martin. 1990. "The clinical utility of distortion product otoacoustic emission." *Ear and Hearing,* 11(2).

Martin, G. K., R. Probst., S. A. Scheinin., A. C. Coats. & B. L. Lousbury-Martin. 1987. "Acoustic distortion products in rabbits. II. Sites of origin revealed by suppression and pure-tone exposures." *Hearing Research.* 28, 29-44.

Moulin, A & D. T. Kemp. 1996. "Multicomponent acoustic distortion product otoacoustic emission phase in humans. I. General Characteristic." *Journal of Acoustical Society of America,* 100(3), 1617~1639.

Naeve, S. L., R. H. Margolis., S. C. Levine & E. M. Fournier. 1992. "Effect of ear-canal air pressure on evoked otoacoustic emissions." *Journal of Acoustic Society American.* 91, 2091-2095.

O'Mahoney, C. 1993. "Investigating the latency of the 2f1-f2 distortion product otoacoustic emission in normal and pathological using the phase gradient method." A research report submitted for the degree of master of science of the university London.

Probst, R. 1990. "Otoacoustic emission overview." *Advances in otorhinolaryngology.* 44, 1~91.

Probst, R. & R. Hauser. 1990. " Distortion product otoacoustic emissions in normal and hearing-impaired ears." *American Journal of Otolaryngology.* 11, 236-243.

Robinette, M. S. & T. J. Glattke. 1997. *Otoacoustic Emissions: Clinical applications.* New York: Thieme.

Ueda, H., S. Nakata, & M. Hoshino. 1998. "Effect of effusion in the middle ear and perforation of the tympanic membrane on otoacoustic emission in guinea pigs." *Hearing Research,* 122, 41-46.

Veuillet, E., L. Collet. & A. Morgan. 1992. "Differential effects of ear-canal pressure and contralateral acoustic stimulation on evoked otoacoustic emissions in humans." *Hearing Research.* 61, 47-55.

Received : Jan. 3, 2000.
Accepted : Feb. 26, 2000.

▲Junghak Lee
94-200 Yongdungpo-Dong Yongdungpo-Gu, Seoul, Korea (150-030)
Hangang Sacred Heart Hospital, Dept. of Otolaryngology, Hallym University
Tel: +82-2-2639-5482
Fax: +82-2-678-2521
e-mail: leejh@sun.hallym.ac.kr

▲Soojin Cho
7-206 Sinhung-Dong Samga Chung-Gu, Inchon, Korea (400-103)
Inha University Medical Center, Dept. of Otolaryngology
Tel: +82-32-890-2434
Fax: +82-32-890-2430
e-mail: en7474@hanmail.net

▲Jinsook Kim
37-12 Chamwon-Dong Seocho-Gu, Seoul, Korea (137-743)
Hearing-Speech-Language Institute, Hallym University
Tel: +82-2-3446-2475
Fax: +82-2-3446-2479
e-mail: jskim@sun.hallym.ac.kr