An Electrocochleographic Study of Acute Low-Tone Sensorineural Hearing Loss

（急性低音障害型感音難聴の蝸電図による研究）

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INTRODUCTION

Low tone sensorineural hearing loss with acute onset but without vertigo, of unknown origin has been considered as a single disease entity, termed "acute low tone sensorineural hearing loss (ALHL)" (1-4).

ALHL occurs commonly in the fourth and fifth decades and is predominant in females. Patients complain of a sense of fullness in the ear and autophonia, rather than a hearing impairment. Hearing returns to normal within several days after onset in most cases. However, there is recurrence of hearing loss without vertigo in about one-third of all patients and with vertigo in another ten percents; the latter are eventually diagnosed as having Meniere's disease. Significant differences with respect to subjective symptoms and clinical findings at the first examination are not found between patients with and without recurrent attacks (2,3).

The etiology of ALHL is as yet unknown. With the glycerol test, hearing loss in ALHL has been reported to improve in more than two-thirds of all patients (3), and in all patients examined in another study (5). Previous electrocochleographic (ECoG) studies on 6 patients with ALHL revealed high amplitudes of both an action potential (AP), also known as N1, and a negative summating potential (-SP)

with almost normal cochlear microphonics, which resembled ECoG findings in patients with early Meniere's disease (4).

In order to clarify the pathophysiology of ALHL, we conducted an ECoG study on the amplitudes of both -SP and AP and the -SP/AP ratio, in comparison with cases of normal hearing and those having Meniere's disease.

SUBJECTS AND METHOD

Subjects

Twenty-four patients of hearing impairment diagnosed with ALHL were examined and compared with 15 normal hearing controls and 7 patients having Meniere's disease with moderately impaired hearing. An ECoG was performed on the patients with ALHL before their hearing loss improved and on the Meniere patients during an attack.

The diagnostic criteria for ALHL was set as shown in Table 1. For the diagnosis of Meniere's disease, the criteria proposed by the Meniere's Disease Research Committee of Japan was applied. The average audiograms for the ALHL and Meniere patients are shown in Figure 1.

Method

1. Electrode

The needle electrode was a polyurethane-coated stainless needle, 0.2 mm in diameter. The tip of the electrode was placed on the promontory anterior to the round window niche, by penetrating the postero-inferior portion of the eardrum under local anesthesia. An

1. Acute low tone sensorinueral hearing loss
   1) Total hearing threshold of 0.125, 0.25 and 0.5kHz $\geqq$ 100dB and total hearing threshold of 2, 4 and 8kHz $\leqq$ 60dB
   2) No evidence of conductive deficit
   3) Idiopathic hearing loss with acute onset
2. No episode of dizziness or prior hearing loss
3. No neurological abnormalities in either history, physical examination or laboratory test

Table 1. Diagnostic criteria for ALHL

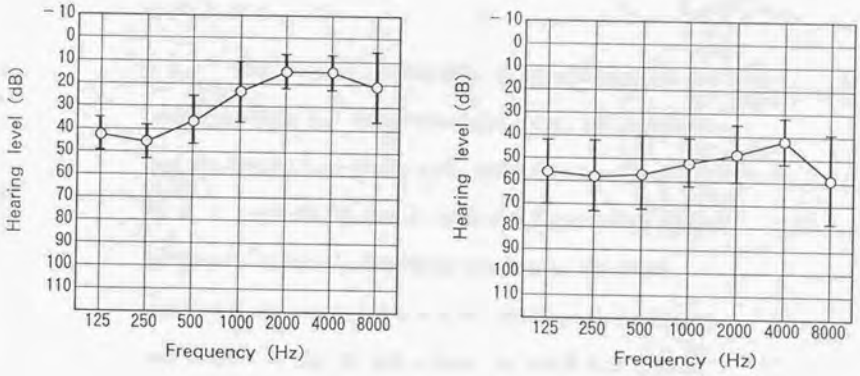


Figure 1. Hearing level (mean value ± 1 SD) in 24 patients with ALHL (left) and 7 patients with Meniere's disease (right).

expandable foam ear plug, containing a central tube connected to a remote earphone, was then inserted in the external meatus to keep the electrode in place (Fig. 2). Silver chloride disk-type electrodes were applied as the reference and ground electrodes on the ipsilateral mastoid and forehead, respectively. The impedance between the promontory and reference electrodes was always mainteined within 10 kilo-ohms (kΩ).

2. Acoustic Stimulation

The acoustic stimuli (Fig. 3) were alternating polarity clicks (0.1 msec rectangular pulse) and condensation and rarefaction tone-bursts with center frequencies of 0.5, 1, 2, 4, and 8 kHz (rise-fall time of 0.5 msec and a plateau of 8 msec). A Nicolet Pathfinder was used as the sound-generating apparatus, and a Nicolet tubal insert earphone was coupled to the ear with a foam tip, containing a sound channel of 1.93 mm diameter tubing. The repetition rate was 11.1/sec, which was increased to 99.9/sec for differentiating between the -SP and AP. Intensity of the acoustic stimuli was expressed in decibels re normal subjective threshold (dBnHL) and was varied from 85 dB to 25 dB in 10 dB steps. The frequency spectra of the acoustic stimuli measured at the end of the 1.93 mm tubing are shown

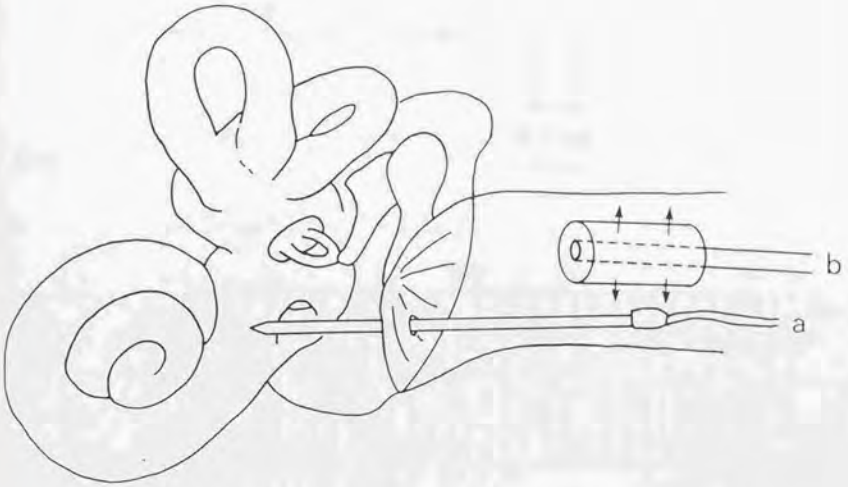


Figure 2. Schematic drawing of the placement of the needle electrode (a), which was inserted through the tympanic membrane on the promontory and kept in place by an expandable foam ear plug (b).

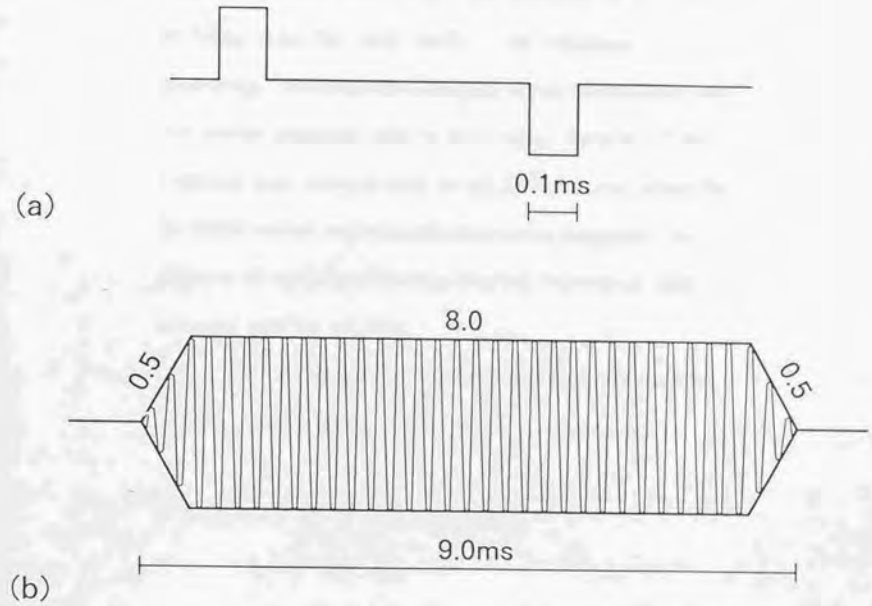


Figure 3. Waveform of the acoustic stimuli used in this study: (a) alternating click; (b) 4kHz tone-burst.

in Figure 4.

3. Recording and Averaging System

Responses were amplified and summated using a Nicolet Pathfinder with a bandpass of 3 Hz to 1.5 kHz. A computer system allowed the average waveforms to be filed on floppy disks for later retrieval and additional processing, including the averaging of the condensation and rarefaction responses used in this study. Averages of 300 responses were obtained with an artificial-reject algorithm. To obtain summed condensation-rarefaction responses, the averages at each porality were obtained separately, then averaged together off line.

A block diagram of the apparatus used for the ECoG in this study is shown in Figure 5.

4. Measurement of -SP and AP Amplitudes

A negative summating potential appears as a hump on the leading edge of the average SP-AP complex waveform evoked by a click, and as a broad current shift in the baseline evoked by a tone-burst (Fig. 6).

Both the -SP and AP peak amplitudes were measured from a pre-stimulus baseline (Fig. 7). The amplitudes of the

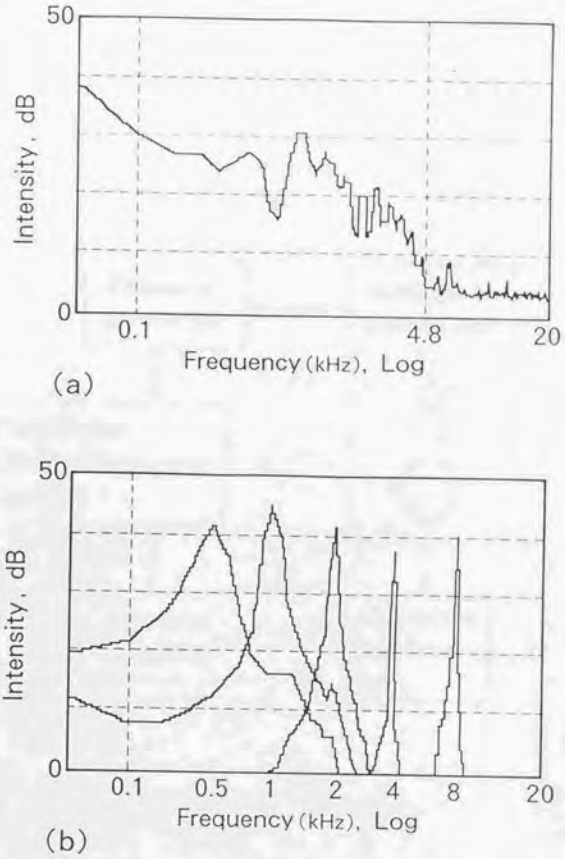


Figure 4. Frequency spectra of the acoustic stimuli used in this study: (a) click; (b) tone-burst with a center frequency of 0.5, 1, 2, 4 and 8 kHz from the left to the right.

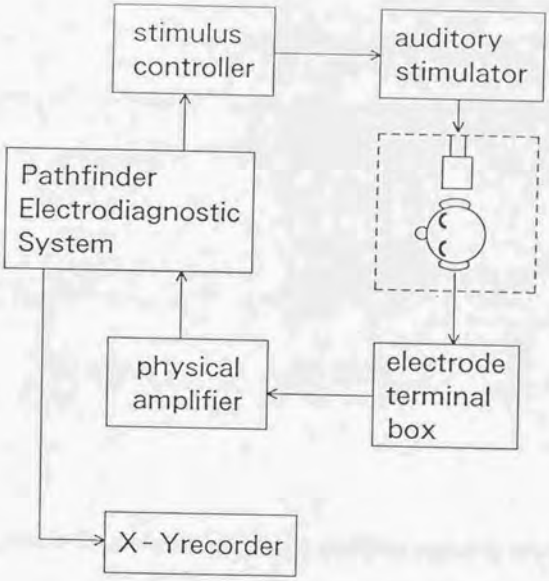


Figure 5. Block diagram of the apparatus for an ECoG used in this study.

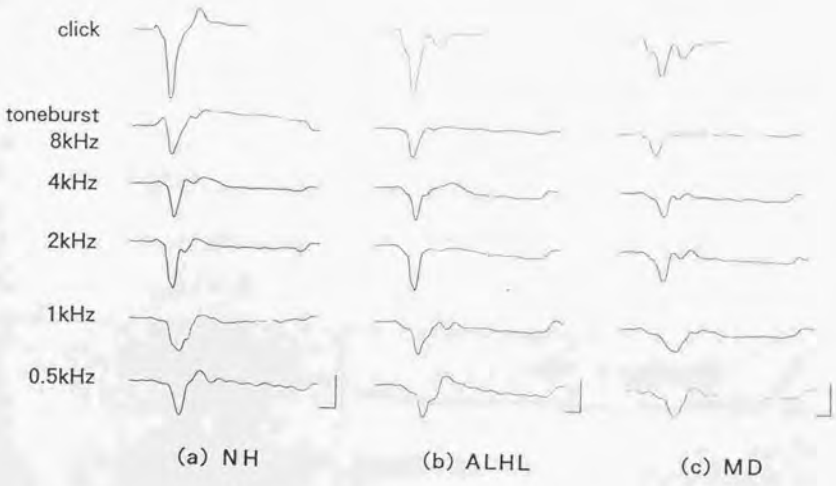


Figure 6. Typical SP-AP complex waveforms evoked by a click and a toneburst at the intensity of 85 dB in normal hearing (left), ALHL patient (center) and Meniere patient (right). The acoustic stimuli from the top to the bottom are in order an alternate click and a tone-burst with 8, 4, 2, 1 and 0.5 kHz. The horizontal scale indicates durations of 1 ms; the perpendicular scale, 5 μV.

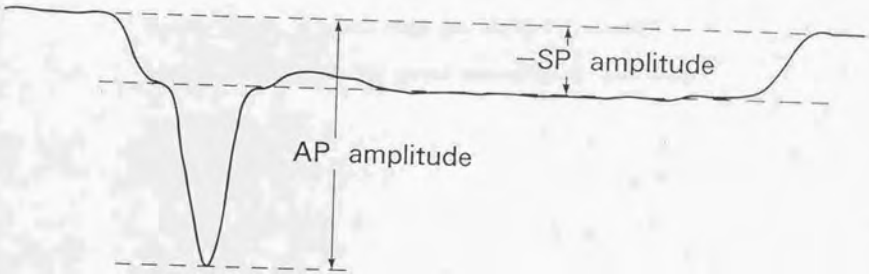


Figure 7. Schmatic drawing of a measurement of the AP and -SP amplitudes.

-SP were measured at both 11.1 and 99.9/s rates, and these measurements were averaged. The AP was measured at a 11.1/s rate.

5. Comparison between Groups

The amplitude of -SP and AP and the ratio between them were measured at an intensity of 85 dB and then compared for cases of ALHL, Meniere's disease and normal hearing. In addition, the number of patients with -SP amplitude or -SP/AP ratio above a normal range was studied in both the ALHL and Meniere groups. A normal range was set as + 2 standard deviations (SD) above the normal mean at 85 dB (confidence coefficient, 95%).

RESULTS

1. -SP Amplitude

Summating potentials in the ALHL and normal hearing cases were commonly found over 45 dB using a click, and over 55 dB using a tone-burst. The increase in the -SP amplitude in the ALHL cases was as gradual as that in the normal hearing subjects at low intensities and somewhat steeper over 75 dB. In the Meniere subjects, -SP appeared over 55 dB using both a click and a tone-burst and increased rapidly (Fig. 8). In all of the groups, a click evoked a greater -SP than a toneburst.

The ALHL group showed a significantly larger -SP amplitude than the normals with both types of acoustic stimuli. The Meniere group also had a significantly larger amplitude than the normals, except for 2 and 8 kHz tone-bursts. Although the -SP amplitude of the ALHL group was larger than that of the Meniere group, this finding was not significant (Table 2).

The detection rates of cases with an -SP amplitude above the normal range were 54% in the ALHL group and 29% in the Meniere group using a click. These results were almost the same as those for tone-bursts with lower frequencies; the number of subjects with a -SP amplitude above the normal

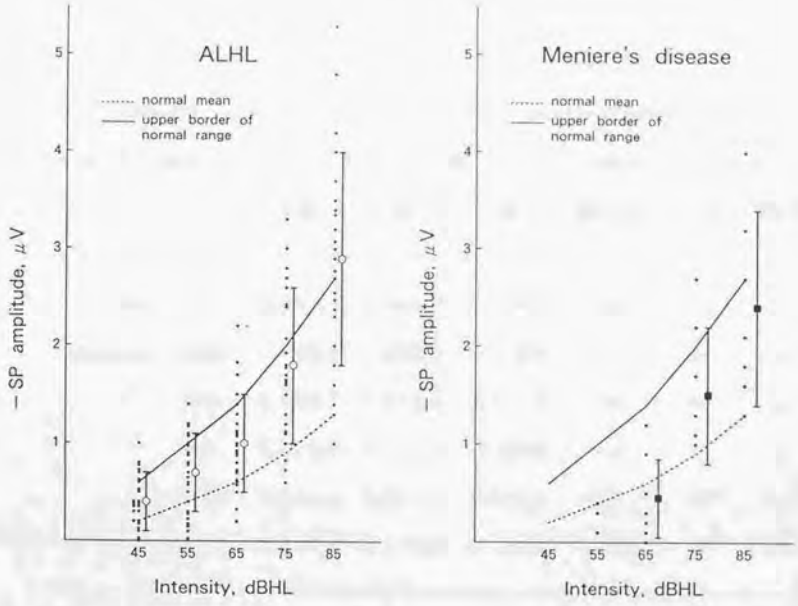


Figure 8. The change in the -SP amplitude in the ALHL group (left) and the Meniere group (right) as a function of stimulus intensity. Individual data are plotted as mean values ± 1 SD. A dotted line indicates the normal means; solid line: upper border of the normal range (normal mean + 2 SD); abscissa: stimulus intensity, dBnHL; ordinate: -SP amplitude, μV; stimulus: a click.

type of stimulus	-SP amplitude			statistical analysis		
	ALHL	MD	NH	ALHL:MD	ALHL:NH	MD:NH
click	2.9±1.1	2.4±1.0	1.3±0.7	ns	**	*
toneburst 0.5kHz	1.5±0.8	1.2±0.7	0.6±0.3	ns	**	*
1kHz	1.7±0.7	1.4±0.8	0.7±0.3	ns	**	*
2kHz	2.0±0.9	1.7±1.0	1.1±0.6	ns	**	ns
4kHz	2.0±0.9	1.8±1.1	1.2±0.7	ns	**	**
8kHz	0.9±0.7	0.7±0.7	0.3±0.4	ns	**	ns

Table 2. -SP amplitudes at 85dB in acute low tone sensorineural hearing loss (ALHL), Meniere's disease (MD) and normal hearing (NH). The mean values ± 1 SD are calculated. Two asterisks indicate $p<0.01$; one asterisk: $p<0.05$; ns: not significant.

range decreased at 4 and 8 kHz (Table 3).

2. AP Amplitude

The loudness-intensity curves for the APs evoked by clicks were obtained for each group (Fig. 9). Action potentials were found even at 25 dB in the ALHL group as well as in the normal hearing controls, but the detection threshold was about 55dB in the patients with Meniere's disease. The ALHL group showed different response slopes between the low and high intensities, which somewhat resembled the curves for the normal hearing controls. In contrast, the Meniere group had only a steep curve. A similar tendency was found using a toneburst, though at 0.5 kHz, the ALHL group had a steeper slope at low intensities and a somewhat higher threshold than for the other frequencies.

The mean values $\pm$ 1 SD in the AP amplitude at 85 dB are summarized in Table 4. Though the ALHL group had slightly smaller AP amplitudes than the normal hearing controls with both acoustic stimuli, these differences were not significant. In contrast, the AP amplitudes of the Meniere group were significantly smaller than those of both the normal hearing controls and the ALHL patients.

type of stimulus	-SP amplitude(%)		-SP/AP ratio(%)	
	ALHL	MD	ALHL	MD
click	54	29	63	86
toneburst 0.5kHz	54	43	54	71
1kHz	59	29	67	86
2kHz	38	29	63	86
4kHz	21	14	29	57
8kHz	25	14	4	29

Table 3. Detection rate for abnormally increased -SP in acute low tone sensorineural hearing loss (ALHL) and Meniere's disease (MD) using the -SP amplitude and -SP/AP ratio.

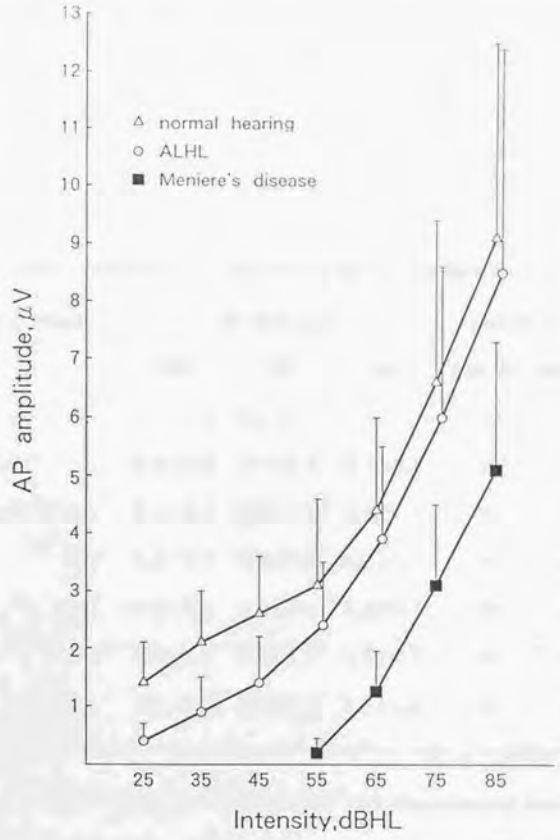


Figure 9. The AP amplitude change in the ALHL group (○), the normal hearing controls (△) and the Meniere group (■) as a function of stimulus intensity. Mean values + 1 SD in each group are shown. Abscissa: stimulus intensity, dBnHL; ordinate: AP amplitude, μV; stimulus: a click.

type of stimulus	AP amplitude			statistical analysis		
	ALHL	MD	NH	ALHL:MD	ALHL:NH	MD:NH
click	8.5±3.9	5.1±2.2	9.1±3.4	**	ns	**
toneburst 0.5kHz	4.3±2.0	2.6±1.2	4.4±1.9	**	ns	**
1kHz	5.6±2.4	3.0±1.4	5.8±2.2	**	ns	**
2kHz	5.7±2.2	3.4±1.6	6.5±2.4	**	ns	**
4kHz	6.0±2.5	3.6±1.7	6.6±2.4	**	ns	**
8kHz	4.4±2.0	2.3±1.2	4.1±1.8	**	ns	**

Table 4. AP amplitudes at 85dB in acute low tone sensorineural hearing loss (ALHL), Meniere's disease (MD) and normal hearing (NH). The mean values ± 1 SD are calculated. Two asterisks indicate $p<0.01$; ns: not significant.

3. -SP/AP Ratio

In both the ALHL and Meniere groups, a maximum -SP/AP ratio with minimum SD was obtained at the intensity of 85 dB (Fig. 10). In each group, the -SP/AP ratios were almost the same with both acoustic stimuli, except for 8 kHz tone-bursts, which apparently caused smaller -SP/AP ratios.

Both the ALHL and Meniere groups had significantly greater -SP/AP ratios than the normal using both acoustic stimuli, though the ALHL group showed a smaller ratio than the Meniere group (Table 5). Comparing the individual -SP/AP ratios, 15 of the 24 ALHL cases (63%) and 6 out of the 7 Meniere patients (86%) showed abnormally increased -SP/AP ratios using clicks. The detection rates were almost the same using tone-bursts of lower frequencies and decreased towards the higher frequencies (Table 3).

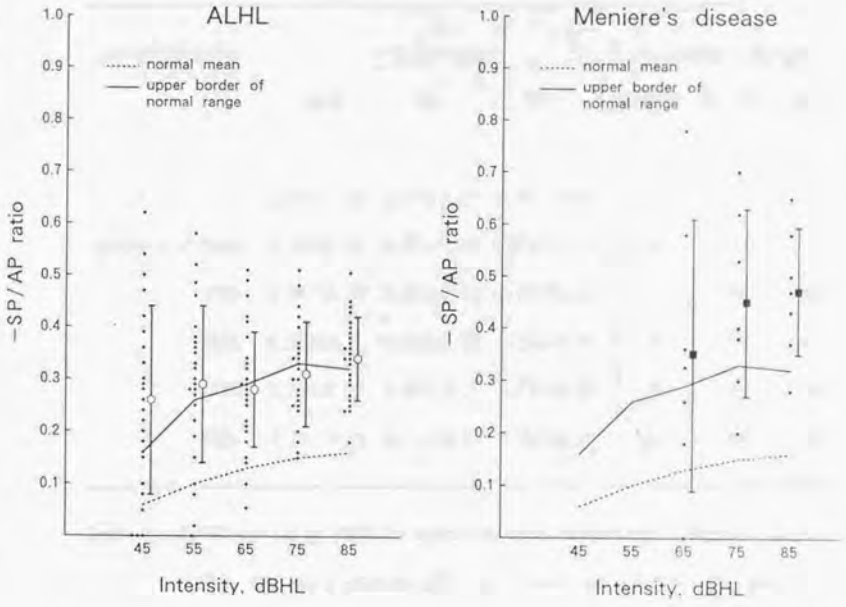


Figure 10. The -SP/AP ratio change in the ALHL group (left) and the Meniere group (right) as a function of stimulus intensity. Individual data are plotted as mean values ± 1 SD. The dotted line indicates the normal mean; solid line: upper border of the normal range (normal mean + 2 SD); abscissa: stimulus intensity, dBnHL; ordinate: -SP/AP ratio; stimulus: a click.

type of stimulus	-SP/AP ratio			statistical analysis		
	ALHL	MD	NH	ALHL:MD	ALHL:NH	MD:NH
click	0.34±0.08	0.47±0.12	0.16±0.08	**	**	**
toneburst 0.5kHz	0.35±0.11	0.47±0.16	0.15±0.10	ns	**	**
1kHz	0.34±0.13	0.46±0.15	0.13±0.08	ns	**	**
2kHz	0.36±0.11	0.49±0.16	0.16±0.08	*	**	**
4kHz	0.34±0.09	0.50±0.17	0.18±0.10	*	**	**
8kHz	0.19±0.11	0.31±0.16	0.09±0.12	ns	**	**

Table 5. -SP/AP ratios at 85dB in acute low tone sensorineural hearing loss (ALHL), Meniere's disease (MD) and normal hearing (NH). The mean values ± 1 SD are calculated. Two asterisks indicate $p<0.01$; one asterisk :$p<0.05$; ns: not significant.

DISCUSSION

This study reveals that both the -SP amplitude and the -SP/AP ratio of cases of ALHL, as well as those of Meniere's disease, are significantly greater than those of normal hearing controls.

Cochlear summating potentials recorded from the human promontory originate mainly from the outer hair cells (6) of the basal-most part of the cochlea (7) and represent a DC-shift during stimulation, which results from a nonlinear motion of the basilar membrane around its resting position or a nonlinearity in the mechano-electrical transduction process (8). Clinically, an abnormally increased -SP is widely known to be present in ears with Meniere's disease, whose histopathological finding is characterized as endolymphatic hydrops. In contrast, purely sensory damaged ears have a significantly smaller -SP than normal (9-13).

The abnormal increase in the -SP suggests that an asymmetrical activity occurs. Whether this nonlinearity is related to the initial hydromechanical processes, or to subsequent mechanoelectric transduction within the hair cells, has yet to be elucidated. However, the mode of the vibration of the basilar membrane is modified by alteration of the intra-cochlear hydrostatic pressure: When pressure in the scala media is increased, vibration toward the scala

tympani is limited and the normal asymmetric movement toward the scala media is enhanced, resulting in an electrocochleographic recording of an increased -SP (14). A similar modification of the DIF SP has been experimentally demonstrated using low frequency stimulus biasing, which causes displacement of the basilar membrane (15). Further, a greater -SP/AP ratio to condensation clicks than to rarefaction clicks is found in patients with electrophysiologic evidence of endolymphatic hydrops (16). These findings support the hypothesis that an increased -SP in endolymphatic hydrops is mainly due to a mechanical asymmetry of the basilar membrane.

In addition, the -SP amplitude in many patients with Meniere's disease is known to reduce by dehydration with glycerol (17,18), which decreases the endolymphatic pressure (19). It seems likely that the effect of glycerol is to reduce endolymphatic hydrops, resulting in a better symmetry of the basilar membrane vibration.

This increase in the -SP has been discussed mainly in terms of the -SP amplitude and the -SP/AP ratio. Although Gibson et al. (20) considered the duration of the -SP to be more reliable than the -SP amplitude, the tail of the SP-AP complex waveform evoked by a click is composed primarily of the AP rather than the SP, even at a high stimulus rate (9), and the duration of the -SP using a tone-burst is mainly

determined by stimulus duration. Therefore, the duration of the -SP was not analyzed in this study.

The amplitude of the -SP varies across subjects probably because of the randomly distributed values of the shunt resistances from the middle ears or the randomly distributed differences in the electroanatomy of the various cochlea (7). Considering the reports of test-retest variability in transtympanic ECoG by Schmidt et al. (21) and Bergholtz et al. (22), it is unlikely that this large variation in the -SP amplitude is caused by differences in the position of the transtympanic electrode.

Because of this variability in the -SP amplitude and because of the linear relationship between amplitudes of -SP and AP in normal hearing (11,24), the individual -SP amplitude has been considered less useful for detection of an increased -SP than the individual -SP/AP ratio (7,10). In fact, the detection rates for the abnormally increased -SPs are smaller using the -SP amplitude than using the -SP/AP ratio (Table 6). This tendency was also found in both the AIHL and Meniere subjects in this study, which had detection rates with a click and tone-bursts below 4 kHz similar to previous studies on Meniere's disease (7,9-13). These rates decrease toward the higher frequencies; Eggermont (7) has pointed out that the -SP amplitude in Meniere's disease shows a loss of sensitivity and maximum

Authors	-SP amplitude(%)	-SP/AP ratio(%)
Eggermont (7)	13	42
Coats (9)	45	64
Ohashi & Takeyama (10)	25	75
Goin et al. (11)	38	62
Mori et al. (12)	23	80
Gibson et al. (13)	36	94

Table 6. Detection rates for abnormally increased -SP in Meniere's ears with the use of the -SP amplitude and the -SP/AP ratio. Eggermont's data (7) are quoted from Coats (9) because of no direct description of the incidence in Eggermont's original. Detection of abnormality has generally been made at 2 SD above normal mean; Ohashi and Takeyama (10) decided on 1 SD above normal mean as a normal range for the -SP amplitude, and Gibson et al. (13) used purely sensory damaged ears as controls.

output toward higher frequencies compared to normal hearing ears.

Regarding the mean values of -SP amplitudes, most investigators (9,11,12,23) have found that Meniere's disease shows significantly greater values than normal. In this study, the mean values of the -SP amplitude in the ALHL group were significantly greater than those of the normal hearing controls with both acoustic stimuli. Because the tone-bursts used in this study were composed of a relatively long plateau, thereby increasing frequency specificity, the increased -SP amplitudes found even at lower frequencies suggest that a pathological condition causing an increased -SP might exist in not only the basal-most part of the cochlea but also in the more apical part.

Inspection of the -SP/AP waveform suggests that my baseline-to-peak AP amplitude measurement, which used the same method as most previous reports(9-13,23,25), is actually the sum of the -SP and AP amplitudes. Hence, the strong covariability between the -SP and AP amplitudes may be at least partly an artifact of our measurement procedure. In this regard, it might be better to measure the AP amplitude from the SP-produced inflection to the N1 peak. However, using a click, the -SP peak is at, or close to, the leading edge of the N1 wave and, by the time the N1 peak

occurs, the SP has undergone a significant positive swing. Thus, when the AP is measured from the -SP inflection to the N1 peak, the effect of the SP waveform may be to artificially decrease the AP amplitude. Indeed, if the -SP has returned to the baseline by the time the N1 peak occurs, then the baseline-peak AP criterion would better approximate the "true" AP amplitude than would the inflection-peak criterion (9). A similar effect also occurs using a tone-burst, though such positive swing of the -SP may be smaller than that obtained using a click. When the -SP/AP ratio is compared between Meniere patients and normal hearing persons, the results obtained with the use of SP inflection-peak AP measurement are almost identical to those using baseline-peak AP amplitude measurement (9). Therefore, my standard baseline-to-peak measurement is considered the most useful for evaluating the -SP/AP ratio.

When comparing the -SP/AP ratio between groups, a significantly greater ratio has been found in Meniere cases than in normals by most laboratories (11,12,23); only Gibson et al. (13) failed to find a significant difference between cases of normal hearing and those with Meniere's disease with hearing better than 40 dB HL. This study reveals a significantly greater ratio in the ALHL patients than in normals, though the ALHL patients showed an apparently smaller ratio than did our Meniere patients.

The increase in the -SP/AP ratio may result either from an abnormal increase in the SP amplitude or from a relatively unchanged SP amplitude with a marked decrease in the AP amplitude (10). In Meniere's patients, the -SP amplitude is almost the same without regard to hearing thresholds, whereas the AP amplitude decreases as hearing loss worsens (13). As a result, the -SP/AP ratio increases in some proportion to the elevated hearing threshold (10,13). In this study, the AP amplitudes differed significantly between the ALHL and the Meniere patients because of their different hearing thresholds. This could explain the smaller ratio in the ALHL group than in the Meniere group showing moderate hearing loss.

The possibility still remains that an increased -SP may occur predominantly in low tone deafness with no relationship to cochlear pathology. However, familial low tone deafness showed decreased -SP (25). In addition, apparent differences in the -SP amplitude have been noted between ears with matched audiogram shapes but with a different clinical diagnosis (Meniere vs non-Meniere cases) (9). Therefore, a significant relationship between low tone deafness and an increased -SP seems unlikely.

In conclusion, this study has revealed an abnormally increased -SP in ALHL, as well as in Meniere's disease, which suggests that ALHL may be due to a pathophysiological

process similar to Meniere's disease and probably due to endolymphatic hydrops. Low tone deafness in endolymphatic hydrops could be explained by an elastic bias of the basilar membrane and/or a mass loading of the cochlear duct (26). Considering the clinical course of ALHL, it could be speculated that transient endolymphatic hydrops in the cochlea might cause ALHL, and that when hydrops becomes more severe and broader, involving also the vestibulum, ALHL might eventually develop into Meniere's disease.

REFERENCES

1. Kobayashi T, Kobayashi R. Low-tone sensorineural deafness of sudden onset. Otologia (Fukuoka) 1978;24:656-659
2. Abe T, Kon Y, Murai K, et al. Clinical pictures of low tone sudden deafness. J Otolaryngol Jpn 1988;91:667-676
3. Yamasoba T, Kikuchi S, Yagi M, et al. Prognosis of acute low-tone sensorineural hearing loss. J Otolaryngol Jpn 1992;95:41-50
4. Osawa H, Kumagami H, Kunimura M, et al. Electrocochleographic findings in low tone sensorineural deafness. Audiology Jpn 1985;28:749-757
5. Takemori S, Hirota T, Matsubara H. Endolymphatic hydrops, its diagnosis and treatment. Pract Otol (Kyoto) 1984;77:723-728
6. Dallos P, Cheatham MA. Production of cochlear potentials by inner and outer haircells. J Acoust Soc Am 1976;60:510-512
7. Eggermont JJ. Summating potential in Meniere's disease. Arch Oto-Rhino-Laryng 1979;222:63-75
8. Durrant JD, Gans D. Biasing of the summating potentials. Acta Otolaryngol (Stockh) 1975;80:13-18
9. Coats AC. The summating potential and Meniere's disease. I. summating potential amplitude in Meniere and non-

Meniere ears. Arch Otolaryngol 1981;107:199-208

10. Ohashi T, Takeyama I. Clinical significance of SP/AP ratio in inner ear diseases. ORL 1989;51:235-245

11. Goin DW, Staller SJ, Asher DL, et al. Summating potential in Meniere's disease. Laryngoscope 1982;92: 1383-1389

12. Mori N, Asai H, Doi K, et al. Diagnostic value of extratympanic electrocochleography in Meniere's disease. Audiology 1987;26:103-110

13. Gibson WPR, Prusher DK, Kilheng GPS. Diagnostic significance of transtympanic electrocochleography in Meniere's disease. Ann Otol 1983;92:155-159

14. Bulter R, Honrubia V: Responses of cochlear potentials to changes in hydrostatic pressure. J Acoust Soc Amer 35:1188-1192,1963

15. Durrant JD, Dallos P: Modification of DIF summating potential components by stimulus biasing. J Acoust Soc Amer 56:562-570,1974

16. Gatland DJ, Billings RJ, Youngs RP, Johnson NP: Investigation of the physiological basis of summating potential changes in endolymphatic hydrops. Acta Otolaryngol (Stockh) 105:218-222,1988

17. Moffat DA, Gibson WPR, Ramdsen RT, Morrison AW, Booth JB: Transtympanic electrocochleography during glycerol dehydration. Acta Otolaryngol (Stockh) 85:158-166,1978

18. Gibbin KP, Mason SM, Singh CB: Glycerol dehydrayion tests in Meniere's disorder using extratympanic electrocochleography. Clin Otolaryngol 6:395-400,1981

19. Bosher SK, Warran RL: A study of the electrochemistry and osmotic relationship of the cochlear fluids in the neonatal rat at the time of the development of the endocochlear potential. J Physiol 212:739-761,1971

20. Gibson WPR, Moffat DA, Ramsden RT. Clinical electrocochleography in the diagnosis and management of Meniere's disorder. Audiology 1977;16:389-401

21. Schmidt PH, Odenthal DW, Eggermont JJ, et al. Electrocochleographic study of a case of Lermoyez's syndrome. Acta Otolaryngol (Stockh) 1975;79:287-291

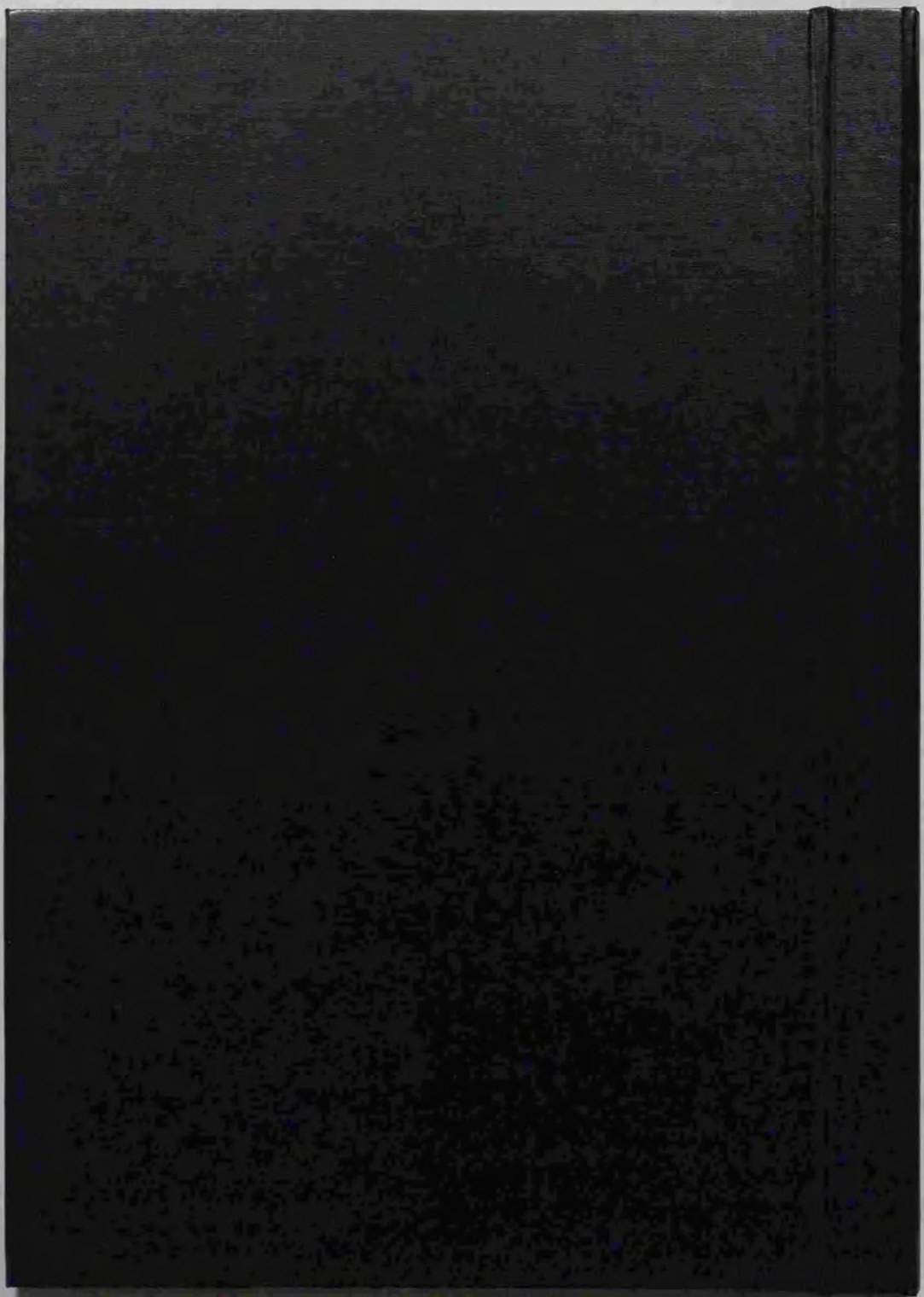
22. Bergholtz LM, Hooper RE, Mehta DC. Test-retest reliability in clinical electrocochleography. Ann Otol 1976;85:679-686

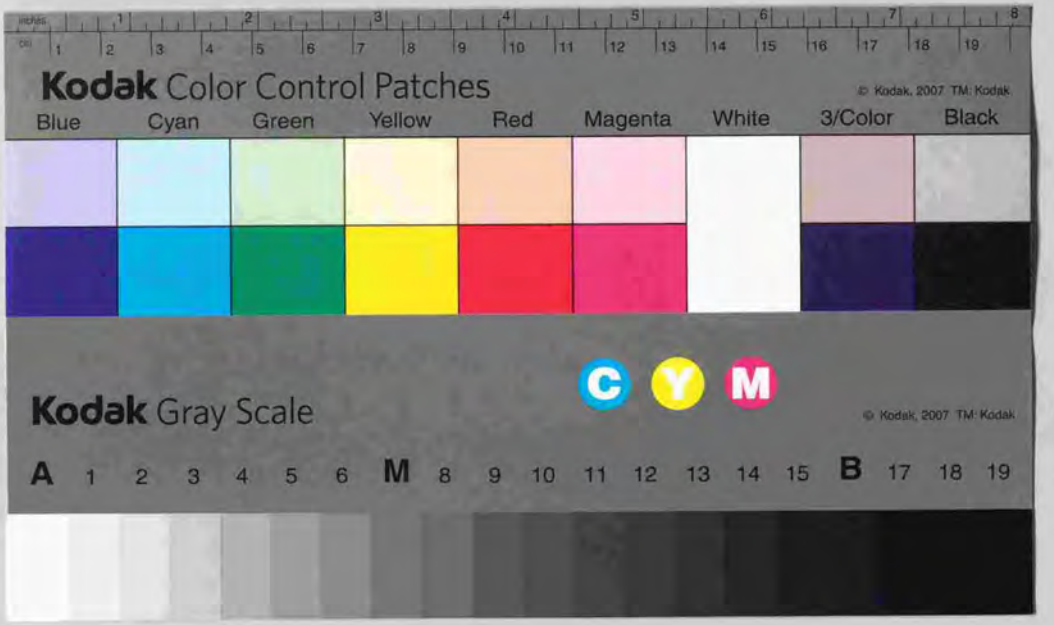
23. Aso S. Clinical electrocochleography in Meniere's disease. J Otolaryngol Jpn 1990;93:1093-1105

24. Coats AC. The normal summating potential recorded from external ear canal. Arch Otolaryngol Head Neck Surg 1986;112:759-768

25. Sasano T. Familial deafness showing hearing pattern of low-tone losses. J Otolaryngol Jpn 1991;94:667-677

26. Tonndorf J. Endolymphatic hydrops, mechanical causes of hearing loss. Arch Oto-Rhino-Laryng 1976;212:293-299

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