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ORIGINAL ARTICLE

Does Meniere's Disease Really Increase Traveling Wave Velocity of the Basilar Membrane? Estimation with Frequency-Specific Electrocochleography*

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Objective: Aim of the study was to compare Action Potential latency difference (as a measure of traveling wave velocity-TWV) using electrocochleography (ECoG) in Meniere patients, non-Meniere cochlear hearing loss patients and control subjects.

Materials and Methods: This prospective study included three groups, 38 patients with Meniere's disease (41 ears), 30 patients with cochlear hearing loss other than Meniere's disease (40 ears) and 25 healthy subjects (40 ears). Tympanic membrane ECoG (click and tone-burst evoked) and caloric test were performed. Forty one ears with Meniere's disease were categorized into definite (28 ears) and probable Meniere patients (13 ears) based on recommendation of the Committee on Hearing and Equilibrium of the American Academy of Otolaryngology-Head and Neck Surgery. Summation potential (SP) / action potential (AP) ratio and AP latency difference for 0.5 kHz and 2 kHz were measured in click and tone-burst evoked ECoG. In caloric test, unilateral weakness was sought. The patients were then further stratified based on abnormal SP/AP ratio and presence of UW. AP latency difference was compared among the groups and subgroups (based on SP/AP ratio and presence of UW).

Results: Median AP latency differences were 0.14, 0.25, 0.19 and 0.23 ms in probable and definite Meniere groups, non-Meniere group and control subjects, respectively. There was significant differences in TWV between definite Meniere group and control group and also between probable Meniere and definite Meniere groups (p's<0.05). When including cases with abnormal SP/AP ratio, AP latency difference were 0.06, 0.25, 0.21 and 0.13 ms. AP latency difference were 0.12 and 0.23 ms in probable and definite Meniere groups with UW.

Conclusions: There was significant difference in TWV in probable and definite Meniere groups. This can be explained with adhesion or fibrosis involved with the basilar membrane hampering its appropriate movement in definite Meniere patients. Including cases with abnormal SP/AP ratio better distinguished probable and definite Meniere groups from control group. Presence of UW only helps better separation between probable Meniere group and control group.

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Introduction

Diagnosis of fully blown endolymphatic hydrops (ELH) is based on clinical symptoms and signs which is rarely problematic. However, very limited number of patients with vertigo only may need to wait a longer time to be diagnosed with. Problematic cases usually have slight hearing loss, especially at lower frequencies. Sometimes, it is difficult to identify causative or active ear in cases with bilateral slight hearing loss. Therefore, an objective test is still needed at least for such cases. Traveling Wave Velocity (TWV) or Traveling Wave Delay (TWD) is a noninvasive and objective test in diagnosis of endolymphatic hydrops (ELH) [1]. Several studies have been published with controversial results [2-14].

A recent study has already established methodology of traveling wave delay using frequency-specific electrocochleography (ECoG) [15]. Several advantages

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of this technique over classical methods were described. Early auditory evoked potentials other than ECoG do not seem to be completely free of error. One of the potential problems is their relatively long distance of origins to the cochlea. However, the spiral ganglion from which the Action Potential (AP) in ECoG does originate is very close to the basilar membrane. One may consider that this proximity might provide accuracy in TWD measurements [15]. Another possible problem with the "derived band auditory brainstem response (ABR) technique" is the difficulty in marking of real peak of the response that might be degraded resulting from low signal-to-noise ratio or the subtraction process [7]. If derived band ABR or auditory-evoked responses to tone-burst stimuli recorded by means of classic ABR montage is being used, most of the time, response quality or signal-to-noise ratio may allow correct threshold determination, but identifying the real peak may not be possible because of the few artifactual peaks along the trace. However, this is not the case in compound AP because of better response quality and wave resolution in frequency-specific ECoG. Moreover, for the derived band technique, commercially available evoked-potential equipment necessitates additional hardware for click stimulus to be mixed with high-pass filtered white noise [15]. For the mentioned reasons, we aimed to investigate utility of TWD using frequency-specific ECoG in diagnosis of ELH.

Materials and Methods

The study consisted of three groups, 38 patients with Meniere's disease (Meniere group), 30 patients with cochlear hearing loss other than Meniere's disease (non-Meniere cochlear hearing loss group) and 25 healthy subjects with no hearing and balance disorders (control group).

Diagnosis of Meniere's disease was based on the criteria recommended by the Committee on Hearing and Equilibrium of the American Academy of Otolaryngology-Head and Neck Surgery (AAO-HNS) for Meniere patients [16]. There were 25 females and 13 males. Mean age was 42.2±10.13 years (ranging from 24 to 67 yrs). Duration of the disease was 54.3±30.48 months. There were 12 cases with bilateral involvement. However, 41 ears from 38 Meniere patients were included because of hearing thresholds exceeding 70 dB HL at which level ECoG recording was not possible in 9 ears. Forty-one ears with Meniere's disease were re-grouped into definite Meniere patients (28 ears) and probable Meniere patients (13 ears) accordingly.

In the group of non-Meniere cochlear hearing loss patients, there were 16 females and 14 males. Forty ears were selected from 30 patients. Mean age was 55.1±10.11 years. Causative disorders were noise-induced hearing loss, mumps-induced hearing loss (in childhood) and presbyacusis. Duration of the hearing loss was 7.5±2.34 years.

Forty ears were selected from 25 healthy subjects (all male) with no hearing and balance disorders. Mean age was 22.5±2.5 years. The criterion was 20 dB-hearing level at octave frequencies from 250 Hz to 6 kHz.

First diagnostic procedures included detailed Ear, Nose and Throat examination and neurotologic examination. Laboratory examination consisted of pure tone audiogram, tympanogram, stapedius reflex test, ECoG and caloric test. Pure tone audiogram across the frequencies from 0.125 Hz to 6 kHz and speech tests were performed using regularly calibrated audiometers (AC-5 and AC-30) (Interacoustics, Assens/Denmark) in sound-proof booths (Interacoustics, Assens/Denmark). Tympanogram and stapedius reflex were carried out using an empedancemeter (Amplaid model 775, Milano/Italy).

Audiograms, tympanic membrane electrocochleography and caloric tests were performed at the same day in order to provide consistency.

Before the TM-ECoG, the outer ear canal was cleaned and disinfected under otomicroscopic view. For topical anesthesia of the tympanic membrane, a combination of lidocaine 2.5% and prilocaine 2.5% (EMLA cream(tm), AstraZeneca Medical and Chemical Products Inc., İstanbul/ Turkey) was applied with a cotton ball to the tympanic membrane for a few minutes.

TM-ECoG test was performed when a subject or patient was in supine position in a quiet room (electromagnetically insulated aw well) designated for testing auditory-evoked potentials. ECoG potentials were recorded with a specially designed-tympanic membrane electrode (TM ECochGtrode electrode; Bio-logic Systems Inc., IL/USA) in the Smart-EP Multi-Channel Evoked Potential System (Intelligent Hearing Systems Inc., FL/USA). The positive tympanic membrane electrode was inserted at the posterior-inferior quadrant of the tympanic membrane under otomicroscopic view. The negative disc electrode was positioned on the mastoid skin, and served as a reference electrode. The ground electrode was placed on the forehead. Inter-electrode impedance difference was kept less than 7 kΩ. For delivering sound stimuli, an earphone (ER-3A(tm), Etymotic Research Inc., Elk Grove Village, Illinois/USA) was inserted into the ear canal. Fixation of the recording electrode was achieved by placing foam tip of the earphone into the ear canal.

There were two types of stimuli used. First stimulus type was click stimuli in alternating polarity. Stimulus intensity was 90 dB nHL. Stimulus repetition rate was 9.7/sec. Low- and high-pass filters were set at 10 to 3,000 Hz. Summation potential (SP) and action potential (AP) were obtained using this type of stimuli. The summation potential (SP) and action potential (AP) were identified on each trace. SP/AP ratio was automatically calculated following the identification the peaks of the potentials.

Second type of stimuli were a series of 1-0.5-1 ms-tone stimuli (at the rate of 19.7/sec) delivered with a Blackman window at 0.5 and 2 kHz at 90 dB nHL. Power spectrums of 0.5- and 2 kHz- tone burst stimuli are presented in Figures 1a and 1b. The figures show frequency specificity of the stimuli. Low- and high-pass filters were set at 10 to 1,500 Hz. Only AP was identified on these traces. AP latency was measured on traces obtained in response to tonal stimuli at mentioned frequencies. Then, AP latency difference was compared among the groups in terms of SP/AP ratio positivity. For this comparison, upper cut off value of normal SP/AP ratio was measured based on ±2 standard deviation of the mean SP/AP ratio (0.19 + 2 X 0.05 = 0.29) which was obtained from the control subjects. Those patients with SP/AP ratio exceeding 0.29 were considered abnormal.

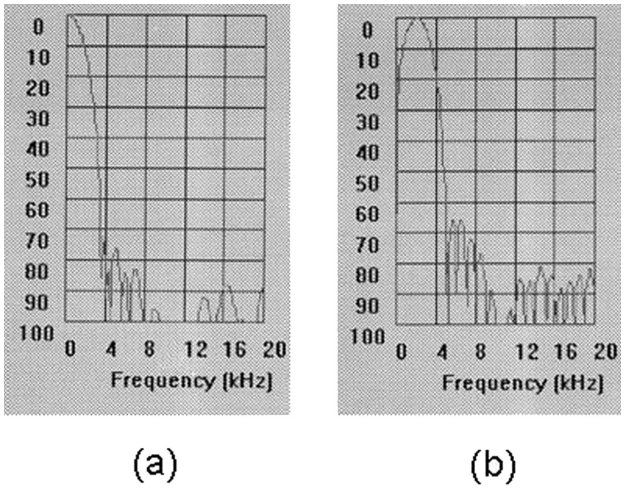

Figure 1. Power spectrums of 0.5 kHz- (a) and 2 kHz-tone burst stimuli (b).

For both types of stimuli, a total of 128 or 256 stimuli were used depending on response quality. Response quality was always considered to stop acquisition. Time base was set at 5 msec. Potentials were amplified with a gain of 105.

For measuring Traveling Wave Delay (TWD), AP latency difference was used. AP latency difference was calculated by subtracting AP latency measured for 0.5 kHz from another AP latency measured for 2 kHz.

Caloric test was performed in all patients, but not in control subjects and non-Meniere HL patients. For caloric testing, a video goggles (Model VG-30, ICS Medical, Schaumburg/IL, USA), videonystagmography (CHARTR VNG Diagnostic Systems MCU-90, ICS Medical Inc., Schaumburg/IL, USA) software and its equipment with a water irrigation system (CHARTR Water Caloric Stimulator, Model NCI-480E, ICS Medical Inc., Schaumburg/IL, USA) were utilized. Details of caloric test may be found elsewhere [17]. Briefly, peak slow phase velocity (pSPV) was averaged over a 10-sec period at the time of maximal

intensity of the nystagmus. A difference in pSPV between the sides >20% was considered presence of unilateral weakness (UW).

Grouping

Overall AP latency difference was investigated in all groups, probable Meniere group, definite Meniere group, non-Meniere HL group and control group. AP latency difference was also investigated in subgroups based on normal and abnormal SP/AP ratio. Thus, each group was split into two subgroups, those with normal SP/AP ratio and those with abnormal SP/AP ratio. Further stratifying the groups (except for control subjects and non-Meniere HL patients) was based on presence of UW.

Comparisons and Statistical Analyses

Data were analyzed using SPSS 10.0 software (SPSS Inc. Illinois/USA). Equality of medians among groups and subgroups (based on either SP/AP ratio or presence of UW) was investigated using Kruskal-Wallis test in terms of overall AP latency difference, AP latency difference in patients with abnormal and normal SP/AP ratio and also with/without UW. Inter-group comparisons of overall AP latency difference were made using Mann-Whitney U test.

Results

Average air conduction (AC) thresholds was 13±5 dB, 39±18 dB, 33±9 dB, and 9±2 dB for probable Meniere group, definite Meniere group, non-Meniere cochlear hearing loss group and control group, respectively.

Traces at 0.5 and 2 kHz in response to 90 dB nHL tone-burst stimuli in a control subject, probable and definite Meniere patients are presented in Figures 2a, 2b and 2c.

AP latency difference [median (25th and 75th percentiles)] is shown in Table 1. When taking overall AP latency difference values in the table into account, Kruskal-Wallis test confirmed the significant difference among all 4 groups ($p<0.05$). Among the all groups, lowest AP latency difference was obtained from probable Meniere group (Figure 3). Albeit an obvious trend of lower AP latency difference in probable Meniere patients compared to control subjects, the difference was not significant ($p>0.05$, Mann-Whitney U test) (Tables 1 and 2). Highest AP latency

Table 1. AP latency difference [median (25th and 75th percentiles)] and its subgroup (based on SP/AP ratio) comparisons

Groups		median (25th and 75th percentiles)
Probable Meniere Group	**Overall (n=13)**	0.14 (0.04,0.27)
	SP/AP normal (n=5)	0.23 (0.14,0.29)
	SP/AP abnormal n=(8)	0.06 (0.03,0.14)
	p*	**0.044**
Definite Meniere Group	**Overall (n=28)**	0.25 (0.20,0.33)
	SP/AP normal (n=11)	0.26 (0.20,0.34)
	SP/AP abnormal (n=17)	0.25 (0.18,0.36)
	p*	0.940
Non-Meniere Cochlear HL Group	**Overall (n=40)**	0.19 (0.13,0.25)
	SP/AP normal (n=32)	0.18 (0.12,0.25)
	SP/AP abnormal (n=8)	0.21 (0.14,0.22)
	p*	0.865
Control Group	**Overall (n=40)**	0.23 (0.15,0.25)
	SP/AP normal (n=38)	0.23 (0.15,0.25)
	SP/AP abnormal (n=2)	0.13 (0.02,0.25)
	p*	0.413
	p**	**0.011**
	p***	0.147
	p****	**0.023**

**:Comparison between those with normal and abnormal SP/AP ratio (Mann-Whitney U test)*

***: Comparison among the all overall values (Kruskal-Wallis test)*

****: Comparison among the subgroups with normal SP/AP ratio (Kruskal-Wallis test)*

*****: Comparison among the subgroups with abnormal SP/AP ratio (Kruskal- Wallis test)*

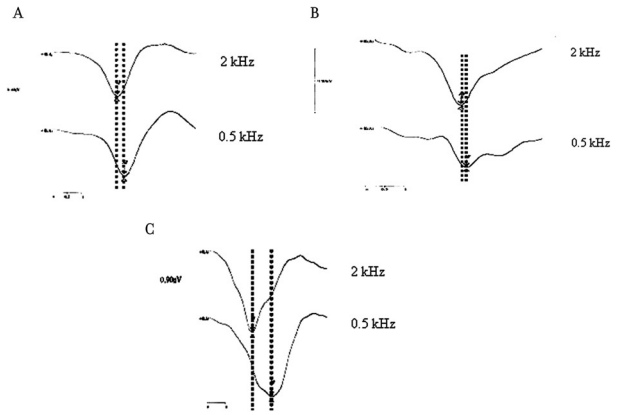

Figure 2. ECoG traces obtained from a control subject (2A), probable (2B) and definite Meniere patients (2C). Time difference between the broken lines corresponds to the AP latency difference for frequency pair of 0.5-2 kHz. Note that AP latency differences in (A), (B) and (C).
2A: a control subject; AP latencies at 0.5 and 2 kHz :2.55 and 2.30 ms. AP latency difference:0.25 ms.
2B: a probable Meniere patient; AP latencies at 0.5 and 2 kHz : 2.50 and 2.35 ms. AP latency difference:0.15 ms.
2C: a definite Meniere patient; AP latencies at 0.5 and 2 kHz : 2.45 and 3.50 ms. AP latency difference:1.05 ms.

differences were observed in definite Meniere patients (Figure 3). Difference in AP latency difference between control subjects and definite Meniere patients was marginally significant (p= 0.05, Mann-Whitney U test) (Table 2). AP latency difference was lower in probable Meniere patients compared to definite Meniere patients, and the difference between the two was significant (p<0.05, Mann-Whitney U test) (Figure 3, Table 2). Since very similar AP latency difference values were measured in control subjects and patients with non-Meniere cochlear hearing loss, the difference between the two was not significant (p>0.05, Mann-Whitney U test) (Table 2). p values of all inter-group comparisons was in Table 2.

When investigating those cases including normal SP/AP ratio only in groups, Kruskal-Wallis test failed to show any significant difference among the groups in terms of AP latency difference (p>0.05) (Table 1). However, when including only cases with abnormal SP/AP ratio in groups, the same test yielded a significant difference in AP latency difference (p<0.05) (Table 1). In those cases with abnormal SP/AP ratio, while the highest AP latency difference was obtained from definite Meniere patients, the lowest AP latency difference was observed in probable Meniere patients (Figure 4). AP latency difference in each group with reference to the SP/AP abnormality is presented in Table 1 and Figure 4.

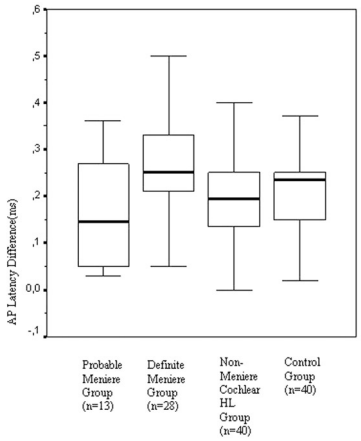

Figure 3. Box plot of AP latency difference in groups (based on overall data)

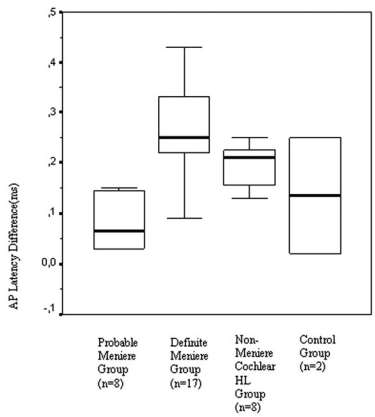

Figure 4. Box plot of AP latency difference in patient groups and subjects with abnormal SP/AP ratio only (based on participants with abnormal SP/AP ratio only). Note that increased gap between definite and probable Meniere groups, and also between definite Meniere group and control subjects, compared to the gaps among the mentioned groups in Figure 2.

Within the probable Meniere group, those with abnormal SP/AP ratio had a lower AP latency difference compared to those with normal SP/AP ratio (p<0.05, Mann-Whitney U test) (Table 1, Figure 5). This trend was almost not observed in the definite Meniere patients, and the difference was not significant (p>0.05, Mann-Whitney U test). Control subjects showed big difference in fact there were only 2 subjects with UW. Therefore, the difference was not significant (p>0.05, Mann-Whitney U test) (Table 1, Figure 5).

Table 2. p values for inter-group comparisons of AP latency difference

Frequency	Inter-group comparisons					
Pair	**1-2**	**1-3**	**1-4**	**2-3**	**2-4**	**3-4**
0.5-2 kHz	**0.008**	0.189	0.119	**0.008**	**0.050**	0.270

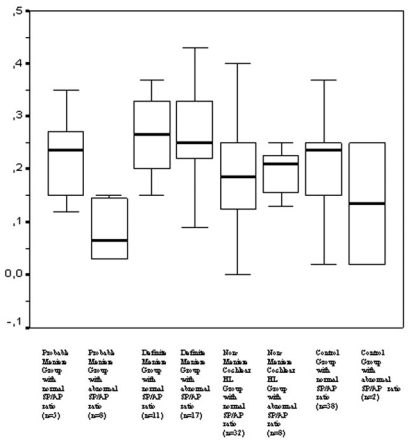

Figure 5. Box plot of AP latency difference in patients and subjects w/wo abnormal SP/AP ratio. Note that the increased gap in probable Meniere group with normal and abnormal SP/AP ratio.

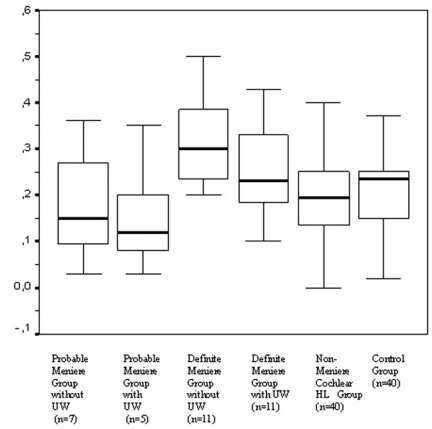

Figure 6. Box plot of AP latency difference in patients w/wo unilateral weakness, non-Meniere cochlear HL group and control subjects. While presence of UW provides a better separation between the probable Meniere group and control group, it brings the definite Meniere group closer to the control group.

As for effect of presence of UW on AP latency difference, Kruskal-Wallis test showed a significant difference in groups (p=0.013). The lowest AP latency difference was seen in probable Meniere patients with UW. Those probable Meniere patients with UW had a lower AP latency difference compared to those without UW (Table 3, Figure 6). However, the difference was not significant (Tables 3 and 4) (p>0.05, Mann-Whitney U test). This trend was also present in definite Meniere patients. However, comparison did not yield a significant difference (p>0.05, Mann-Whitney U test). Significant difference was present only between probable Meniere patients without UW and definite Meniere patients without UW and also between definite Meniere patients without UW and control subjects (p's<0.05, Mann-Whitney U test)

Discussion

In this study, we aimed to investigate TWD using ECoG in Meniere patients who were categorized according to the Committee on Hearing and Equilibrium of the AAO-HNS. The reason behind the categorization is the fact that TWD, if any, should change based on pathologic stage of the disease. The AAO-HNS categorization, at least, reflects severity of the disease. One may assume that TWD should not be the same in different categories.

The reason for using TWD in diagnosing ELH is the fact that high pressure in endolymph is considered to increase stiffness of the basilar membrane leading to augmented movement of the traveling wave [1]. However, if Reissner's membrane ruptures, collapse of membranes occurs and severe adhesions and fibrosis develops, all of which are the signs of somewhat advanced disease, one may assume that the traveling wave velocity would slow down instead of speeding up. Therefore, decreased velocity should be taken into account for diagnosis of endolymphatic hydrops as well.

This is one of the few studies in which ECoG has been used in estimating TWD in categorized Meniere patients, non-Meniere cochlear hearing loss patients

Table 3. AP latency difference [median (25^{th} and 75^{th} percentiles)] and its subgroup (based on presence of unilateral weakness) comparisons

Groups	Subgroups	median (25^{th} and 75^{th} percentiles)
Probable Meniere Group	Unilateral Weakness (-) (n=7)	0.15 (0.05,0.27)
	Unilateral Weakness (+) (n=5)	0.12 (0.05,0.27)
Definite Meniere Group	Unilateral Weakness (-) (n=11)	0.30 (0.22,0.40)
	Unilateral Weakness (+) (n=11)	0.23 (0.15,0.33)
Non-Meniere Cochlear HL Group	Overall (n=40)	0.19 (0.13,0.25)
Control Group	Overall (n=40)	0.23 (0.15,0.25)
	p*	**0.013**

**: Comparison among the all 6 subgroups (Kruskal-Wallis test)*

Table 4. p values for inter-group comparisons of AP latency difference (based on presence of UW)Inter-group comparisons

Inter-group comparisons	p
1-2	0.625
1-3	**0.033**
1-6	0.673
2-4	0.100
2-6	0.183
3-4	0.236
3-6	**0.004**
4-6	0.380

and control subjects. As for interpreting our results, probable Meniere group showed an insignificantly increased TWV compared to control group and non-Meniere cochlear HL group. In contrast, definite Meniere group had a significantly decreased TWV with reference to the control group and non-Meniere cochlear HL group. Analysis also yielded a significant difference between definite Meniere group and probable Meniere group. There was no significant difference between control group and non-Meniere cochlear HL group. Then, one may propose that the results establish a clear separation between definite Meniere patients and control subjects, between definite Meniere patients and non-Meniere cochlear hearing loss group, and also between probable Meniere patients and definite Meniere patients.

Of interest, while probable Meniere group had significantly the highest TWV, definite Meniere group the lowest. The reason behind this contrasted result might have been adhesion or fibrosis involved with the basilar membrane hampering the appropriate propagation of the traveling wave in definite Meniere group. On the contrary, higher TWV in probable Meniere group may be explained with the mentioned classic hypothesis that increased stiffness of the basilar membrane due to hydrops leads to propagation of the traveling wave with higher velocity. This probable Meniere group had no hearing loss (pure tone average was 13 dB HL) and one vertigo episode only as defined by the Committee on Hearing and Equilibrium of the AAO-HNS [16].

Investigation of TWV within the groups with reference to the SP/AP ratio yielded interesting findings. There was no significant difference among the groups with normal SP/AP ratio, while significant difference was observed among the groups with abnormal SP/AP ratio. Probable Meniere patients with abnormal SP/AP ratio had a higher TWV than those probable Meniere patients with normal SP/AP ratio. This trend was also observed insignificantly in definite Meniere group. As for inter-group comparisons with reference to the SP/AP ratio, TWV in probable Meniere patients with abnormal SP/AP ratio was shorter than control group (0.06 ms vs 0.13 ms). One may consider that these results bring out two further elucidations on clinical use of the test and also cochlear mechanics. First, including SP/AP ratio as a parameter increased the TWV test sensitivity. Second, an increment in hydrops (as evidenced by abnormal SP/AP ratio) renders increase in TWV. This second effect was rather significantly observed in probable Meniere group. Under the circumstance of abnormal SP/AP ratio, less

increment in TWV in definite Meniere group may be explained with adhesion or fibrosis involved with the basilar membrane hampering an increase in TWV. In histopathologic examinations, fibrosis or new bone formation in Meniere's disease was shown in scala tympani and vestibuli [18].

Including unilateral weakness as a parameter did not produce a beneficial effect in distinguishing disease categories. It seems that presence of UW causes an increase in TWV in probable and definite Meniere groups. However, this phenomenon had an opposite effect in discriminating the disease categories as such increased the gap between probable Meniere group and control group (better separation between the two), and decreased the gap between definite Meniere group and control group (less separation between the two). Even though it seems that presence of UW resulted in higher TWV in probable and definite Meniere groups compared to absence of UW, the analysis could not differentiate probable or definite Meniere groups from control group.

Previous studies on TWV or TWD were performed using derived band ABR technique or high-pass noise masking of ABR, tone burst ABR or tone burst-evoked otoacoustic emissions [1,8,19]. Some controversial results have been obtained in diagnosing endolymphatic hydrops [1,2,8-14]. Zerlin (1969) recorded a latency delay in response to tonal stimulus pairs of different frequency generated through a dual channel pulse generator in three normal listeners, and found out that TWV decreased from 30 m/s at high frequencies to 1 m/s at low frequencies [20]. Thornton et al. (1989) successfully observed changes in TWV in response to glycerol dehydration test in 5 of 6 patients [1]. Thornton and Farrell (1991) measured 0.6 ms as a lower 95% confidence limit for wave V-latency difference between 1.42 kHz and 5.68 kHz. They stated that differentiation between noise induced hearing loss and Meniere's disease was good enough at high frequencies. TWV was higher in Meniere group [2]. As shown in a study by Gould and Sobhy (1992), mean traveling time from 4 to 1 kHz was 0.69 ms in 18 normal hearing subjects [4]. Donaldson and Ruth (1993) measured TWV in 24 normal-hearing subjects and found out high inter-subject variability at higher frequencies as opposed to smaller variability at the lower frequencies [5]. Kim et al. (1994) reported higher TWV at 8 kHz in 8 Meniere patients compared to control subjects and patients with sensorineural hearing loss [6]. On the contrary, Murray et al. (1998) measured AP latency difference using tone-burst ABR and found out that the test failed to differentiate 12 Meniere patients from 10 normal subjects and 10 patients with cochlear HL [8]. Don et al. (2005) reported 100% sensitivity and 100% specificity with the high-pass noise masking of auditory brainstem responses in distinguishing Meniere patients from control subjects [9]. De Valck et al. (2007) examined latency delay in 28 Meniere patients and 17 normal subjects using a technique called "Cochlear Hydrops Analysis Masking Procedure" similar to high pass noise masking ABR. Almost half the results was not interpretable. Sensitivity and specificity were as low as 31% and 28%. They came to conclusion that the test failed to discriminate Meniere patients from control subjects [10]. This unfavorable result opposite to results of the study by Don et al. (2005) initiated a debate on patient selection, analysis of high-pass responses and selection of stimulus intensity [11,12]. Claes GM et al. (2008) measured TWV in 28 normal subjects and 9 Meniere patients, and found that TWV correlated to ECoG. Other conclusion of the study was the fact that TWV did not correlate to symptoms of Meniere disease [13]. While Elberling (1974) used AP of ECoG, Teas et al. (1962) preferred cochlear microphonics to measure TWV [21,22]. Eggermont and Odenthal (1974) measured TWV using band-reject filtered masking of the AP [23].

Analysis of this study concluded with some clinical and physiopathological remarks:

1. TWD could be successfully estimated using TM-ECoG. Thus, armamentarium to measure TWV is expanded.

2. Our probable Meniere patients (one vertigo attack only and almost no hearing loss- pure tone threshold of 13 dB HL) had a higher traveling wave velocity compared to definite Meniere patients (vertigo attacks more than 2 and pure tone threshold of 39 dB HL) for whom traveling wave velocity was lower.
3. The results displayed that TWD estimated by TM-ECoG was able to distinguish definite Meniere patients from control subjects, non-Meniere cochlear HL patients and probable Meniere patients.
4. Adding SP/AP abnormality provided a better separation between probable and definite Meniere groups and also between definite Meniere patients and control subjects.
5. While an increment in SP/AP ratio caused a higher TWV in probable Meniere patients, the same effect was almost not observed for the definite Meniere group which implies that hydrops could have increased TWV if appropriate movement of the basilar membrane had not been impeded due to fibrosis.
6. Presence of UW also caused higher TWV in both probable and definite Meniere groups. However, adding UW as a parameter did not provide a better separation between definite Meniere group and control subjects as opposed to clear separation between the probable Meniere patients and control subjects.

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