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Vestibular Evoked Myogenic Potentials (VEMP) Can Detect Asymptomatic Saccular Hydrops

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Abstract

Objective—The objective of this study was to explore the usefulness of vestibular evoked myogenic potential (VEMP) testing for detecting endolymphatic hydrops, especially in the second ear of patients with unilateral Ménière disease (MD).

Methods—This study was performed at a tertiary care academic medical center. Part I consisted of postmortem temporal bone specimens from the temporal bone collection of the Massachusetts Eye & Ear Infirmary; part II consisted of consecutive consenting adult patients (n = 82) with unilateral MD by American Academy of Otolaryngology–Head and Neck Surgery criteria case histories. Outcome measures consisted of VEMP thresholds in patients and histologic saccular endolymphatic hydrops in postmortem temporal bones.

Results—Saccular hydrops was observed in the asymptomatic ear in six of 17 (35%) of temporal bones from donors with unilateral MD. Clinic patients with unilateral MD showed elevated mean VEMP thresholds and altered VEMP tuning in their symptomatic ears and, to a lesser degree, in their asymptomatic ears. Specific VEMP frequency and tuning criteria were used to define a “Ménière-like” response. This “Ménière-like” response was seen in 27% of asymptomatic ears of our patients with unilateral MD.

Conclusions—Bilateral involvement is seen in approximately one third of MD cases. Saccular hydrops appears to precede symptoms in bilateral MD. Changes in VEMP threshold and tuning appear to be sensitive to these structural changes in the saccule. If so, then VEMP may be useful as a detector of asymptomatic saccular hydrops and as a predictor of evolving bilateral MD.

Keywords

Ménière disease; VEMP; vestibular evoked myogenic potentials; saccule; hydrops

INTRODUCTION

A significant minority of patients with Ménière disease (MD) eventually develop involvement of their second ear, a situation with a profound impact on patients' symptoms, quality of life, and management options. We have shown previously that ears affected by MD show elevated thresholds and altered frequency tuning of vestibular evoked myogenic potentials (VEMPs).³ Similar changes in mean VEMP threshold and tuning are also seen in the *unaffected* ears of patients with unilateral MD, although to a lesser degree than in the affected ears.³ One possible explanation for this observation is that the group of unaffected ears is really two groups, a group of true normal ears and a group of ears with *asymptomatic* or *presymptomatic* endolymphatic hydrops. The present study was undertaken to explore this possibility and to test the hypothesis that VEMP is sensitive to structural changes in the saccule indicative of asymptomatic or presymptomatic endolymphatic hydrops, a possible harbinger of evolving bilateral MD. The Ménière literature suggests that progression from unilateral to bilateral disease is seen in approximately one third of cases. Our first phase of the present study was to examine postmortem temporal bones from cases of unilateral MD to determine the prevalence of occult structural changes on the asymptomatic side. Then we analyzed VEMP results in the asymptomatic ears of our current patients with unilateral MD to determine the prevalence of "Ménière-like" VEMP responses. Results of these two different approaches are presented here, compared, and discussed.

METHODS AND MATERIALS

Temporal Bone Histopathology

Subjects—The temporal bone collection at the Massachusetts Eye & Ear Infirmary contains 1,771 specimens from 1,012 individuals. The vast majority of these specimens have been fixed in 10% formalin, decalcified in trichloroacetic acid or EDTA, embedded in celloidin, sectioned serially in the axial plane at a thickness of 20 μ m, and every tenth section stained with hematoxylin/ eosin and mounted for light microscopy.⁵ All cases include medical records documenting clinical histories, physical findings, and test results. Information about each case is stored in a computer database. Cases for the present study were selected in the following manner. First, the database clinical histories were searched for key words, including "vertigo," "vertiginous episode," "dizziness," "dizzy spell," "unsteady," "off-balance," and "dysequilibrium" to generate a case list likely to include all those clinically suspect for MD. A total of 230 cases were identified. Then, blinded to histopathologic information, each medical record was reviewed using Ménière diagnostic criteria of the American Academy of Otolaryngology–Head and Neck Surgery (AAO–HNS).⁴ Patients with chronic otitis media, positive VDRL test, vestibular schwannoma, congenital, genetic, autoimmune inner ear diseases, or any other known reasons for endolymphatic hydrops were excluded, thus limiting the list to idiopathic cases. Cases with other vestibular pathology, central lesions, head trauma, and other identifiable causes of vertigo attacks were excluded. Of 81 cases of episodic vertigo, we identified 43 with MD (23 definite, 10 probable, and 10 possible, according AAO–HNS scoring). Of the 43 cases identified with clinical MD, 17 (11 definite, 3 probable, and 3 possible) were clinically unilateral and the remainder were clinically bilateral. The 17 unilateral cases comprise our study subjects.

Laterality of Unilateral Ménière Disease—In these 17 cases, the side of disease was assigned based on three criteria: 1) medical history with explicit statement identifying side of unilateral symptoms of tinnitus, aural fullness, and/or hearing loss (HL) accompanying vertigo attacks; 2) asymmetric sensorineural HL with audiometric documentation of fluctuation and/or progression in the affected ear by audiograms acquired after onset of vestibular symptoms; and 3) when available, caloric asymmetry that corroborated side of disease in cases with

asymmetric hearing or identified side of disease if hearing was symmetric but caloric asymmetry was $\geq 40\%$.

Histopathologic Grading System—A four-level grading system was established to score the severity of saccular hydrops in sections examined by light microscopy (Fig. 1).

1. No hydrops: the membranous wall of the saccule was in the normal position.
2. Mild hydrops: dilatation of the saccule, but its wall did not reach the undersurface of the stapes footplate in any section.
3. Moderate hydrops: dilatation of the saccular wall such that its wall made contact with part but not all of the stapes footplate.
4. Severe hydrops: severe dilatation of the saccular wall such that it made contact with the entire stapes footplate and the surrounding bony wall of the vestibule.

Complete sets of temporal bones from both ears of all 17 unilateral MD cases and three normal control temporal bones were provided to one of the authors (SNM), who was blinded to any information from the case histories. Each bone was scored for saccular hydrops according to the scheme described previously.

Subjects—We tabulated records of 82 consecutive adult patients with definite unilateral MD by AAO–HNS criteria⁴ that were seen at the Massachusetts Eye & Ear Infirmary. VEMP results of 34 of the 82 subjects were reported previously.³ All subjects were evaluated by otologic examination, audiometry, and VEMP testing. Subjects were diagnosed as unilateral Ménière cases based on their subjective report of episodic vertigo occurring in conjunction with hearing fluctuation in the same ear objectively documented as sensorineural HL. Subjects had no subjective or objective evidence of inner ear instability in the unaffected ear such as low frequency or fluctuating sensorineural HL, episodic tinnitus, or aural fullness of the unaffected ear during vertigo attacks. Preexistent age-related or acoustic trauma HL in the contralateral ear was permissible. Subject enrollment and record review was performed in compliance with Institutional Review Board-approved protocol.

Vestibular Evoked Myogenic Potential—VEMP results in unilateral Ménière subjects were compared with VEMP in normal subjects ($n = 12$). Testing was performed as previously described.³ Briefly, VEMPs were recorded from both the right and left sternocleidomastoid muscle (SCM) for 250-, 500-, and 1,000-Hz tonebursts. All patients had sensorineural HL in the Ménière ear.

Stimuli—Tonebursts were generated by custom-programmed evoked potential software. Tonebursts were generated using a Blackman gating function with a two-cycle rise and fall and no plateau for 250, 500, and 1,000 Hz. Stimuli were presented at 13/second to circumaural headphones (Telephonics TDH-49). The starting phase of each stimulus was alternated during recording. The software originally used a decibel HL calibration for the tonebursts (ANSI 1969). This calibration corresponded to peak sound pressures at 90-dB HL of 133 dB peak-equivalent sound pressure level (PSP) at 250 Hz and 123 dB PSP for 500 and 1,000 Hz. Using these calibrations, stimulus intensity levels were converted to PSP before analysis.

Electromyographic Recording—VEMPs were recorded ipsilaterally while subjects were seated upright with their chin turned over the contralateral shoulder to tense the SCM. Electromyographic (EMG) activity was recorded from surface electrodes placed on the SCM; the positive electrode was placed on the upper third of the muscle belly and the reference electrode was placed on the muscle tendon just above the clavicle. A ground electrode was placed on the forehead. Subjects were instructed to tense the SCM during acoustic stimulation

and relax between recordings. Ongoing EMG was visually monitored on an oscilloscope to ensure tension of the muscle. EMG activity was amplified, bandpass-filtered (10–2,000 Hz), and sampled for 30 msec after the stimulus using a digital analog converter and custom evoked potential averaging software on a PC. Separate averages were stored for initial rarefaction phase stimuli and initial condensation phase stimuli. These averages were combined into one average for alternating phase stimuli.

The VEMP was visually judged to be present when the response to alternating phase stimuli was one to two times the residual noise in the alternating average and the response was replicated in the separate rarefaction phase and condensation phase averages. The VEMP was judged to be absent when no replicable response was observed for a residual noise of $\geq 3 \mu V$. Measurements made on VEMP responses were the latency of the first positive peak (P1), latency of the next negative peak (N1), and the amplitude difference between the P1 and N1 amplitudes.

Subclassification of Asymptomatic Ears—Asymptomatic ears were subclassified according to two VEMP characteristics: 1) VEMP threshold-based method and 2) VEMP tuning-based method. In the threshold-based classification, the mean VEMP threshold at each frequency (250, 500, and 1,000 Hz) was calculated for the unaffected ears of the entire study population, and all subjects with VEMP thresholds below the mean at two or more frequencies were assigned to the threshold “normal-like” group. All other subjects were assigned to the threshold “Ménière-like” group. In the tuning-based classification, all subjects whose unaffected ear showed a VEMP threshold pattern of $500 \text{ Hz} \leq 1,000 \text{ Hz}$ were assigned to the tuning “normal-like” group. All others were assigned to the tuning “Ménière-like” group.

This study was approved by the Human Studies Committee of the Massachusetts Eye & Ear Infirmary and all practices were HIPAA-compliant.

RESULTS

Temporal Bone Histopathology Demonstrated Asymptomatic Saccular Hydrops

Conventional clinical wisdom and the MD literature suggest that approximately one third of patients with MD develop bilateral disease. We sought to test this notion using the temporal bone collection of the Massachusetts Eye & Ear Infirmary. We hypothesized that if endolymphatic hydrops precedes clinical MD, then a subset of cases with clinical unilateral MD will have asymptomatic endolymphatic hydrops in the contralateral ear. Table I gives the summary of results. Overall, 17 of 17 (100%) cases showed saccular hydrops in the symptomatic MD ear, of which six of 17 (35%) were mild saccular hydrops, one of 17 (6%) was moderate saccular hydrops, and 10 of 17 (59%) were severe hydrops. In six of 17 (35%) cases, there was saccular hydrops in the asymptomatic ear. Half of these six cases were mild and half were severe. All three control ears were correctly scored as having normal saccules. Because approximately one third of patients with unilateral MD are destined to develop bilateral disease, we interpret our finding of occult saccular endolymphatic hydrops in temporal bones from one third of cases of clinical unilateral MD as support for the hypothesis that hydrops precedes clinical MD.

Vestibular Evoked Myogenic Potential Thresholds Show Elevation and Loss of Frequency Tuning in Both Ears of Patients With Unilateral Ménière Disease

Comparison of normal ($n = 12$), MD-affected, and MD-unaffected ears ($n = 82$) showed that the MD subjects had mean threshold shift and loss of 500-Hz frequency tuning in *both* ears despite having only unilateral clinical MD (see Fig. 2). The unaffected ears showed less mean threshold shift than the affected ears. These results in 82 patients confirm our earlier findings

in a smaller group of 34 patients with unilateral MD.³ The two VEMP parameters that showed change in symptomatic Ménière ears were threshold and frequency tuning. If a subset of patients with unilateral MD is destined to develop active disease in their second ear, then we would predict that their asymptomatic or presymptomatic endolymphatic hydrops could cause “Ménière-like” changes in VEMP in the asymptomatic ear. Such presymptomatic changes in a subset of patients could account for the observed shift in mean VEMP thresholds of the entire group. We tested this hypothesis by dividing the 82 patients with unilateral MD into two groups, “normal-like” and “Ménière-like,” first by threshold criteria, then by tuning criteria, and then by the combination of the threshold and tuning criteria.

Patients with Unilateral Ménière Disease Were Divided into Two Groups, ‘Normal-Like’ or ‘Ménière-Like,’ Based on the Vestibular Evoked Myogenic Potential Thresholds of Their Asymptomatic Ear

We calculated the mean VEMP threshold at each stimulus frequency (250, 500, and 1,000 Hz) for the asymptomatic ears of our 82 patients with unilateral MD. All asymptomatic ears with VEMP threshold below the group mean at ≥ 2 frequencies were assigned to “normal-like” and all others to “Ménière-like” groups. Of 82 cases, 43 (52%) were classified as “normal-like” and 39 (48%) were classified as “Ménière-like.” The “normal-like” group showed thresholds and tuning indistinguishable from true normals. As demonstrated in Figure 3, the “Ménière-like” group showed elevated thresholds and loss of tuning similar to the *affected* ears of these unilateral Ménière subjects.

Patients with Unilateral Ménière Disease Were Divided into Two Groups, ‘Normal-Like’ or ‘Ménière-Like,’ Based on the Vestibular Evoked Myogenic Potential Tuning of Their Asymptomatic Ear

VEMP thresholds in normal subjects show frequency tuning such that the VEMP threshold at 500 Hz is less than at 250 Hz and VEMP threshold at 500 Hz is less than or equal to the threshold at 1,000 Hz. In our 82 patients with unilateral MD, we classified all whose asymptomatic ear met these two threshold criteria as “normal-like” (45 of 82 [55%]) and all others as “Ménière-like” (37 of 82 [45%]). The mean VEMP thresholds and tuning in the “normal-like” group were indistinguishable from normal. As seen in Figure 3, the “Ménière-like” group showed elevated mean thresholds and loss of tuning typical of the *affected* ears of these unilateral Ménière subjects.

When we combined the group assignments by the two classification methods, we found that 22 of 82 patients (27%) were “Ménière-like” by both (see Fig. 3). Our finding that 27% of asymptomatic ears of patients with unilateral MD have “Ménière-like” VEMP changes of both threshold and tuning is highly suggestive that the VEMP is sensitive to detecting asymptomatic or presymptomatic endolymphatic hydrops.

DISCUSSION

Clinical MD is associated with cochleosaccular endolymphatic hydrops. Although there is wide variation across studies, a significant minority of patients with MD eventually develop involvement of both ears. In clinical studies with large samples and long follow up, second ear involvement was seen in 31% to 37% of cases.^{6–8} Similarly, temporal bone studies suggest a 25% to 30% incidence of bilaterality.^{9,10} On average, the literature suggests that symptomatic involvement of the second ear occurs in approximately 30% to 35% of Ménière cases. We have shown previously that the hydropic changes in the affected ears of patients with unilateral MD result in elevated threshold and altered frequency tuning of their VEMP reflex.³ In this study, we looked at the VEMP reflex in asymptomatic ears of patients with unilateral MD. We found that 27% of these patients had VEMP reflexes with threshold elevation and altered tuning

characteristic of symptomatic Ménière ears. In addition, we looked at the histopathology of temporal bones from donors with unilateral MD and found that 35% of asymptomatic ears had saccular endolymphatic hydrops. The similarity of these percentages leads us to draw two important, although tentative, conclusions from this study: 1) endolymphatic hydrops precedes symptoms in MD, and 2) VEMP appears sensitive to the hydropic structural changes in the saccule of patients with MD and is therefore a potential detector of asymptomatic endolymphatic hydrops. It is possible that many (if not all) cases of *asymptomatic* saccular hydrops are actually *presymptomatic* saccular hydrops. If so, VEMP changes may predict eventual bilateral MD.

In terms of understanding the pathophysiology of MD, it is interesting to note that endolymphatic hydrops appears to precede symptoms temporally. This does not necessarily assure a cause-and-effect relationship. For example, many senior citizens get gray hair before they die but gray hair is not the cause of death. If there was a causal progression from mild hydrops to more severe hydrops to Ménière symptoms, one might expect the majority of asymptomatic saccular hydrops to be mild and symptomatic hydrops to be severe. However, this was not our observation. Our six cases of asymptomatic saccular hydrops were evenly split, with three mild cases and three severe cases. Even the symptomatic MD ears of our 17 temporal bone cases showed a range of saccular hydrops severity, with 35% mild and 59% severe. As in the example of gray hair and death in the elderly, endolymphatic hydrops in MD may be an epiphenomenon of the true pathophysiological mechanism that produces the symptoms of fluctuating HL and vertigo. The notion of hydrops as an epiphenomenon has been discussed in detail elsewhere.^{2,11}

Clinically, the prospect of a test for presymptomatic hydrops is exciting. It is conceivable that VEMP could be used to differentiate “pre-Ménière” patients at high risk of developing full-blown MD from non-Ménière patients such as migraine-related vertigo. Initiation of Ménière treatments such as dietary restriction or diuretics in presymptomatic patients may be more effective at preventing progression to overt symptoms than at mitigating symptoms in an ear with full-blown disease. Similarly, “Ménière-like” VEMP changes seen in the asymptomatic ear of a patient with unilateral MD would indicate occult bilateral disease. Such information would be of great value in considering treatment options for intractable vertigo from the first ear. We used both VEMP threshold and VEMP tuning parameters to define “Ménière-like” responses. Either parameter alone showed too much variability to be informative. Use of multivariate models to optimize use of a test or test battery has been shown to be an effective approach in other applications of vestibular function test interpretation.^{12–15}

There is striking similarity between the prevalence of bilateral MD reported in the literature (31–37%), the prevalence of occult saccular hydrops in the asymptomatic temporal bones of unilateral Ménière donors in this study (35%), and the prevalence of “Ménière-like” VEMP changes in asymptomatic ears of patients with unilateral MD in this study (27%). However, we have not proven that patients with “Ménière-like” VEMP are assured of developing symptomatic MD. This can only be done by prospective clinical trials, which are underway at the present time. As we follow our 82 Ménière subjects over time, we will see which develop involvement of their second ear. Only then will be able to assess the positive and negative predictive value of the VEMP test.

VEMP testing is an evolving field. There are a number of different methods that have been described and are in early clinical use. To date, the majority of VEMP publications use toneburst stimuli at a single frequency, usually 500 Hz, and report amplitude and latency of the response. We believe that multifrequency testing is essential to assay necessary frequency tuning characteristics of the saccule. Many aspects of the sensitivity and specificity of VEMP have not yet been adequately explored. Thus, there is much work to be done before VEMP

testing becomes routine. However, the findings in this study and others suggest that VEMP will become a valuable tool in assessment of dizzy patients.

CONCLUSIONS

In MD, endolymphatic hydrops involving the saccule leads to elevated threshold and altered frequency tuning of the VEMP reflex. In this study of patients with unilateral MD, 27% showed “Ménière-like” VEMP responses in their asymptomatic ears, very similar to the 31% to 37% rate of second ear involvement reported in the literature and to the 35% prevalence of occult saccular hydrops seen in our present study of *postmortem* temporal bones from unilateral MD cases. These findings indicate that 1) endolymphatic hydrops of the saccule precedes clinical symptoms in MD and 2) that VEMP testing can detect these changes. VEMP may prove clinically useful for detection of asymptomatic or presymptomatic saccular hydrops.

Acknowledgments

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Fig. 1. Grading scale for saccular hydrops: upper left panel, normal; lower left panel, mild hydrops; upper right panel, moderate hydrops; and lower right panel, severe hydrops.

Fig. 2.

Mean ($\pm$ standard error of mean) vestibular evoked myogenic potential thresholds as a function of stimulus frequency for 12 normal subjects and both ears (affected and unaffected) of 82 patients with unilateral Ménière disease.

Fig. 3.

Normal mean ($\pm$ standard error of mean) vestibular evoked myogenic potential thresholds compared with those from the unaffected ears of 82 patients with unilateral Ménière disease classified as “Ménière-like” by tuning criteria, by threshold criteria, or by both.

TABLE I

Saccular Hydrops in Asymptomatic Ears of Unilateral Ménière Disease Temporal Bone Donors.

AAO–HNS Class	N	Asymptomatic Ear Normal	Asymptomatic Ear Hydropic
Definite	11	8	3
Probable	3	2	1
Possible	3	1	2
Total (%)	17 (100)	11 (65)	6 (35)