ORIGINAL ARTICLE

Diagnosis of Ménière's disease using low-frequency masking

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Submited: 11 July, 2005
Revised: 03 September, 2005
Accepted: 19 September, 2005

Mediterr J Otol 2006; 2: 19-26

OBJECTIVE: To evaluate low-frequency masking in the early diagnosis of Ménière's disease.

PATIENTS AND METHODS: Thirty-five patients suffering from Ménière's disease were examined. The results were compared with that of 10 patients with normal hearing ears and 40 with noise-damaged ears. All examinations were made by the use of a special instrumentation capable of producing a low-frequency sound signal superimposed with a tone burst. We were able to independently change the amplitudes of the 2 applied signals, as well as the phase lag between them from 0° to 360°. Adjusting the 2 amplitudes and the phase lag, the examined individuals heard 2 sounds, 1 as the masking tone and 1 high-frequency tone superimposed on the low-frequency masking.

RESULTS: In normal individuals, the highest masking effect was evident at 250° and the lowest at 360°. A lower masking effect was also evident at 90°. Normal hearing participants had a modulation depth between 20 and 35 dB, while patients with noise damaged ears had an effect about 10 dB and patients with Ménière's disease had one closer to 0 dB. The instrumentation and method presented in this article show a relatively good clinical diagnostic accuracy because it is capable of diagnosing the basilar membrane stiffness on an almost null modulation depth. Test sensitivity is near 70%.

CONCLUSION: The developed instrumentation and method can distinguish and diagnose the early stages and acute recurrences of Ménière's disease. This may lead to a further study of the results of low-frequency masking on the anterior labyrinth in research centers worldwide, in order to reinforce the diagnostic accuracy and strength of this newly developed diagnostic method.

Ménière's disease is a chronic illness, generally characterized by attacks of vertigo, hearing loss, tinnitus, and a sensation of aural fullness. A major characteristic of Ménière's disease is the fluctuation of these symptoms. The diagnostic dilemmas that are raised during the first stages of this peculiar disease are difficult to answered. Although the full-blown clinical picture of the disease is diagnostic, this is not the rule at the initial stages, when the differential diagnoses from other inner ear disorders are not evident. The underlying pathophysiology of Ménière's disease is endolymphatic hydrops,[1] but a defined diagnosis can only be carried out postmortem. The acute hearing loss observed during the acute phases of Ménière's disease is explained by an elastic bias of the basilar membrane.[2]

The gold standard method for diagnosing Ménière's disease is electrocochleography (ECoG),[3] but this is invasive, difficult to perform, and highly specialized. The diagnostic accuracy of ECoG ranges from 60% to 80%,[4] and the electrical phenomena recorded reflects the pathology rather than exposes the mechanical status of the basilar membrane per se.

The radiographic measurements of the cochlear aqueduct5 or vestibular aqueduct[6] are difficult to perform, and they do not correlate well with endolymphatic pressure and, consequently, with Ménière's disease symptomatology.

The only known in vivo diagnostic methods that inform us about the mechanical properties of basilar membrane are the methods directly targeting the basilar membrane's mechanical properties. Marchbank's method, Tympanic Displacement Analysis[7]—small-volume variations in the external auditory canal measured as a consequence of tympanic membrane displacement after contraction of the stapedial muscle induced by an acoustic stimulus—cannot be considered, nowadays, as an additional tool to confirm or reject the diagnosis of Ménière's disease. This is because the method does not permit the detection of absolute pressure values and the results are characterized by large inter-individual differences in the measurement parameters.[8,9] Other methods targeting basilar membrane properties are based on the use of auditory masking.

The first published report on subjective-phase–dependent threshold using low-frequency masking is from Zwicker, who discovered that the basilar membrane moves in phase with a low-frequency–masker tone in the first and second turns of the human cochlea.[10]

In recording cochlear potentials, Morizono and Sikora found that in experimentally induced endolymphatic hydrops in animal models, the masking of a short-tone stimulus does not depend on the phase of a low-frequency–masking tone, contrary to the normal hearing models.[11] Using the aforementioned method, Hoehmann and colleagues studied other types of cochlear disorders.[12]

The use of low-frequency masking in humans was also investigated by recording brainstem wave V.13 The same research group concluded that the simplest and most rapid method consisted of recording the subjective-phase–dependent threshold of a short-test stimulus that is superimposed on a low-frequency masking tone. The results they published in 1996 confirm that it is possible to investigate cochlear micromechanics by the use of this method.[14]

We developed a simpler and easier method based on the concept developed by this pioneering research group. We present the results of this method on normal hearing participants, on patients with noise-damaged hearing loss, and on patients with Ménière's disease at different stages of their illnesses.

MATERIALS AND METHODS

Methodology. The methodology followed involved the application of a low-frequency (30 Hz)–sound signal by a pair of headphones to the examined ear of the patient. This low-frequency sound was used as a masking signal, which periodically extended the basilar membrane to a level analogous to its amplitude. A second periodic signal (burst tone) of short duration (ª1 msec) and high frequency (ª 10 kHz) was also applied, superimposed on low-frequency signals at a certain point of its cycle period. We suppose that during the application of a low-frequency–masker tone, the basilar membrane moves synchronously along its total

length. During this movement, the sensitivity of the organ of Corti varies periodically. This change can be measured by applying a short stimulus at different phases of the masker tone. With the help of proper instrumentation, we were able to independently change the amplitudes of the 2 applied signals, as well the phase lag between them from 0° to 360°. Thus, we were able to apply the burst tone at any point, scanning the entire low-frequency cycle period. The low-frequency sound applied was measured at the patient's ear and regulated at 115 dB sound pressure level (SPL). By properly adjusting the 2 amplitudes, the patient heard the 2 sounds as a low-frequency sound interrupted by an instantaneous high-frequency–stimulated noise.

Instrumentation, The instrumentation used resulted in a combination of 2 audio signals produced by 2 function generators, presented schematically in Figures 1 and 2. We were using 2 generators (HM 8030-5 [A] and HM 8131-2 [B]; Hameg Instruments GmbH, Mainhausen, Germany). Generator A produced a pure sinusoidal signal of 30-Hz frequency. Generator B is a synthesized-function generator with a built-in ability to control the phase lag of the produced signal with respect to a triggering external-referring signal. The signal produced in B was a synthesized burst tone of 10-kHz frequency and 1-msec duration. Signals from A and B were mixed and amplified via a custom-made triggering-output amplifier (Figures 3 and 4). This device was triggered in parallel generator B by a referring square-wave signal, consistent with the signal produced by A. By this method, the starting point of the burst tone from B was synchronized with the starting point of the 30-Hz signal from A when the phase lag of B with respect to A is set at 0°. By adjusting the phase lag with respect to the low-frequency signal of A from 0° to 360°, the starting point of the burst can be set at any point of the cycle of the 30-Hz signal. The output mixed 30-Hz and burst signals leading to a patient's headphones. It was also monitored on the screen of a digital oscilloscope (4020; Gould Instruments, Oxnard, Calif, USA). The low-frequency level was measured with the help of a sound level meter (Nor118; Norsonic, Lierskogen, Norway), connected with a quarter-inch free-field, prepolarized, high-level–measuring microphone system (Type 40BE; GRAS Sound and Vibration, Vedbæk, Denmark) properly attached to the cavity of the stimulated headphone (Figure 2).

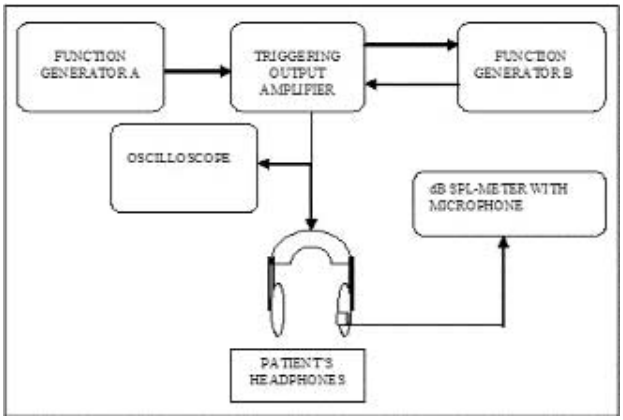

Figure 1: Schematic diagram of the apparatus used in the diagnosis of Ménière's disease.

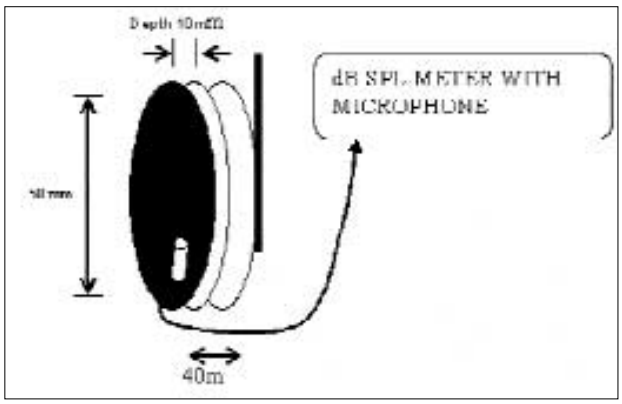

Figure 2: Detailed schematic description of the headphone equipped with the measuring microphone.

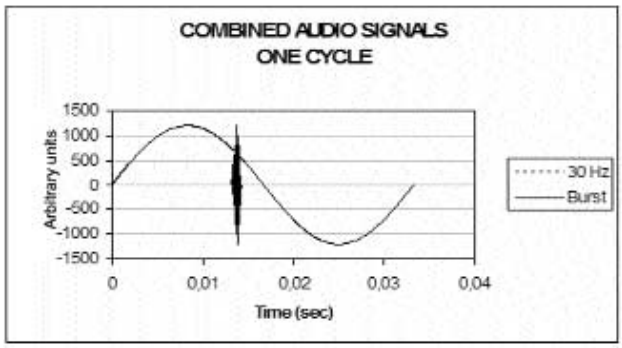

Figure 3: One cycle of the combined low-frequency and burst audio signals. The curve marked 30 Hz represents the masking-tone signal.

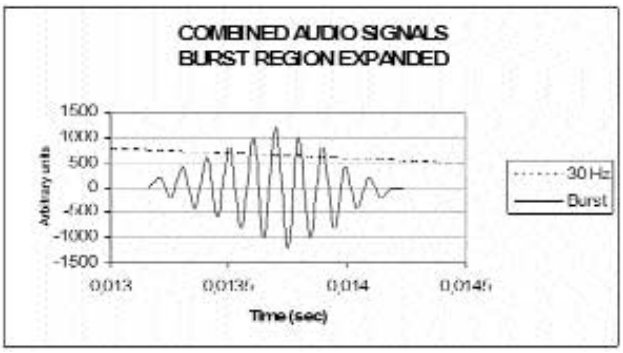

Figure 4: Expanded portion of diagram presented in Figure 3, showing details in the region of the applied burst signal. The curve marked 30 Hz represents the masking-tone signal.

Procedure. The phase-dependent recording of the masking threshold of short-time stimuli has to be in discrete steps. The stimulus to be masked has to be short compared with the period of the masking tone. The click stimulus fulfills this condition optimally. Tone bursts (short-tone stimuli) are better suited to the subjective measurement, as they are more audible at low levels within the low-frequency tone.[15]

The patient under examination was supplied with headphones. Initially, we applied the 30-Hz and then the burst-tone to the patient's ear, controlling the sound level at near 115 dB. After that, the phase lag was set to 0, and the combined 30-Hz and burst tone were applied. The examined participant heard a low-tone sound interrupted with the high-tone burst sound. By gradually increasing the phase lag from 0° to 360°, the participant heard both the low and high sounds in different phase lag positions. The measurement was controlled by a computer that also accepts the data of the patients and controls, the measurement parameter settings, and the results. The measurement was performed in steps of 30°. The reported investigations have been performed in accordance with the principles of the Declaration of Helsinki.

The modulation depth (or modulation index) is defined as the maximum excursion of the trailing edge from one extreme to the other.[16]

The SPSS software package (Version 12; Chicago, Ill, USA) was used for all statistical analyses.

Patients. Thirty-five patients suffering from Ménière's disease were examined repeatedly in different phases of their illnesses. Their results were compared with that of 10 patients with normal hearing ears and 40 with noise-damaged ears. The individuals under examination were supplied with the headphones described above and instructed to respond to the tone burst (the examined acoustic stimulus) as it is just audible. They were also instructed not to respond to the masking tone, because it is factitious and to be ignored.

The masking tone was presented at 115 dB SPL, a level sufficient to stretch the basilar membrane but not sufficient to invoke the stapedial reflex.

RESULTS

The results of the participants with normal hearing ears (n=10) and a schematic explanation of the modulation depth are shown in Figure 5. The highest masking effect (least audible level) was evident at 250° with a mean value of 37 dB and the lowest at 360° or 0° (most audible level) with a mean value of 17 dB. A smaller masking effect was found at 90°. The results from the patients with noise-damaged hearing loss and the patients suffering from Ménière's disease are shown in Figure 5.

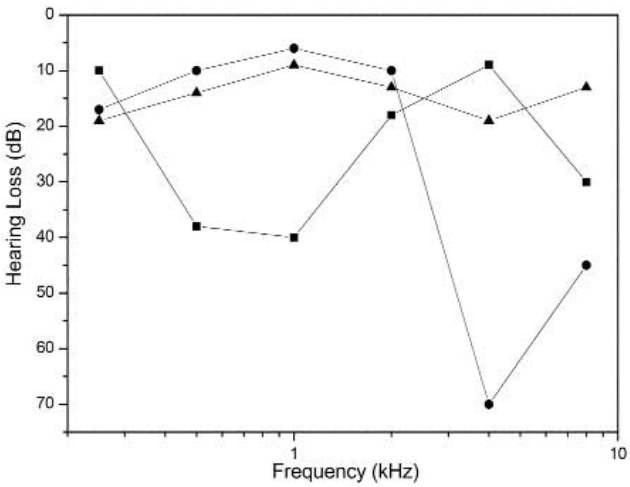

Figure 5: Comparative Tone Audiogram: Ménière's disease subjects (■), noise damaged subjects(●), normal hearing subjects (▲).

The distribution of modulation depth for subjects with normal hearing, noise damage, and Ménière's disease are summarized in Figures 6A, 6B, and 6C, which also show a shift of the modulation depth with the seriousness of the condition. While normal hearing subjects had a modulation depth between 20 and 35 dB, subjects with noise damage appeared with a modulation depth about 10 dB lower. and subjects with Ménière's disease had a modulation depth closer to 0 dB.

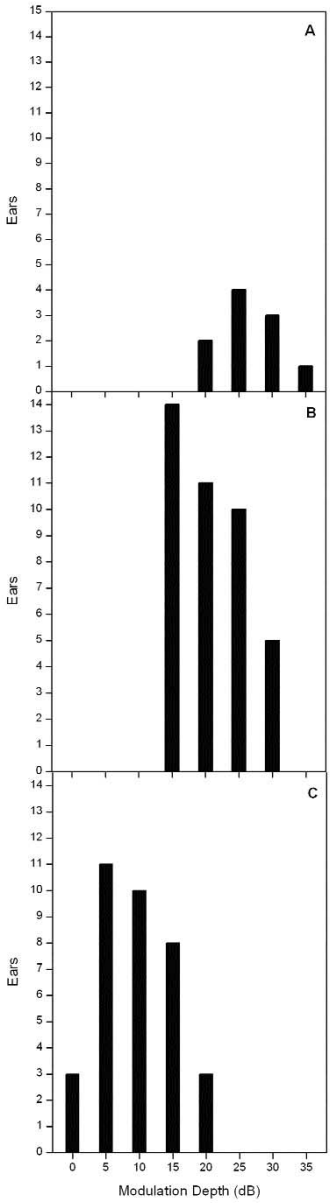

Figure 6: Modulation depth of subjects with normal hearing (n=10) (A), noise-damaged (n=40) (B), and Ménière's disease (n=35) (C).

The mean values for the modulation depth and hearing loss at 500 Hz for the above-mentioned subjects are shown in Figure 7.

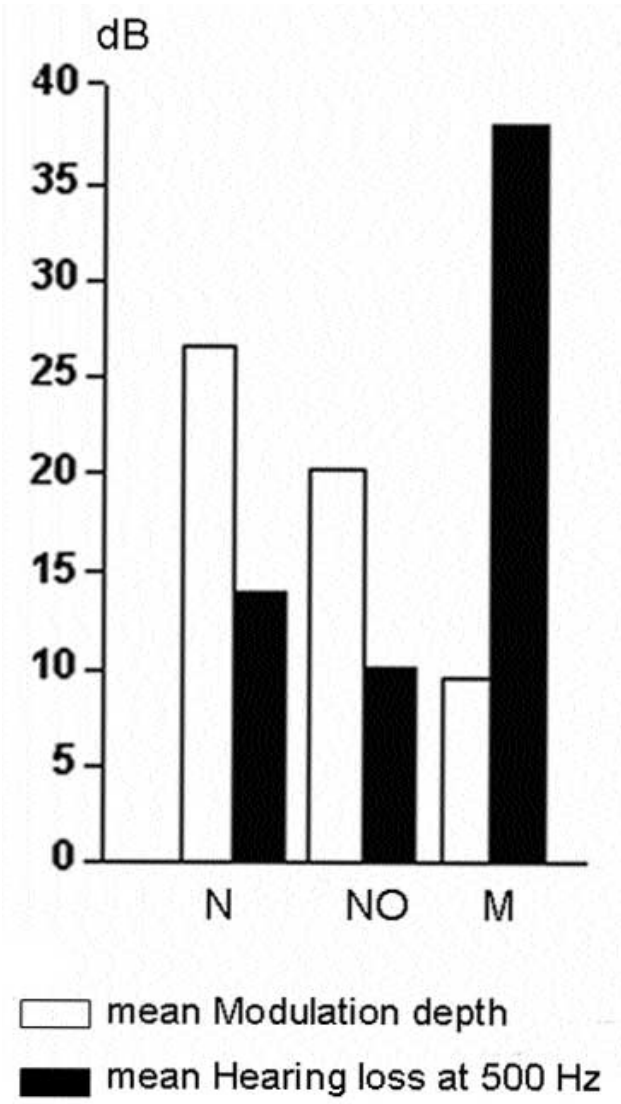

Figure 7: Modulation depth and hearing loss at 500 Hz (mean values) for 10 normal hearing subjects (N), 40 noise-damaged subjects (NO), and 35 Ménière's disease–subjects (M).

The comparative tone and phase audiograms are presented in Figures 5 and 8, respectively, in some typical forms of hearing loss. The hearing loss for frequencies up to 1 kHz varies between 10 and 20 dB.

Comparing each group with the other (normal hearing subjects to subjects with Ménière's disease, nor-

mal hearing subjects to noise-damaged subjects, and Ménière's disease subjects to noise-damaged subjects) using the independent-sample t-test, we find significant differences (P<.05) among the mean values of the compared groups.

When comparing normal hearing subjects with subjects suffering from Ménière's disease, we found significant difference for all angles, except for 90°, 210°, and 300°. Comparing normal hearing subjects with noise damaged subjects, we found significant difference for all angles.

The sensitivity of our method is 68.6%.

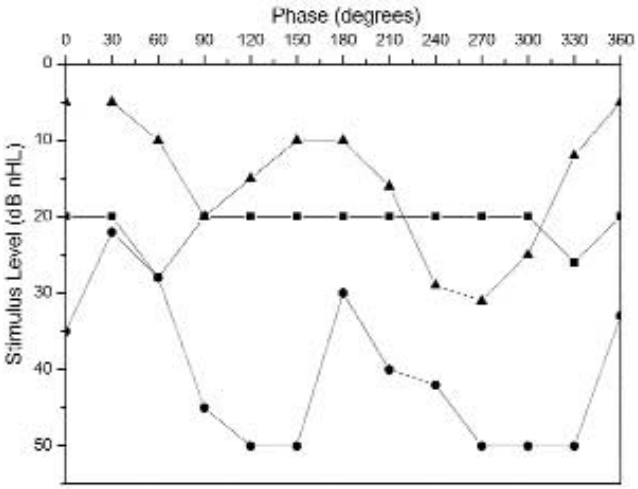

Figure 8: Comparative Phase Audiogram: Ménière's disease subjects (■), noise-damaged subjects (●), normal hearing subjects (▲).

DISCUSSION

The idea behind masking lies in the most important psychoacoustic effect known as "auditory masking." in which faint elements of a sound are simply not heard if similar but louder elements are present in the sound. Low-frequency masking makes it possible to investigate the effect of the displacement of the basilar membrane on the sensitivity of the human cochlear partition, and it is used as a clinical procedure for the differential diagnosis of sensory hearing loss. From experiments in animals and investigations in humans, it is know that the normally phase-dependent masking of a short stimulus by a low-frequency continuous tone does not occur in the case of endolymphatic hydrops.[15]

Since Ménière's disease was first described, its causes remain more or less unknown.[17] The illness runs a course characterized by attacks of vertigo, hearing loss, tinnitus, and sensation of aural fullness, mainly with a relapsing mode. The diagnosis of Ménière's disease at the early stages of its course or the prediction of an acute recurrence of this disease has been always desirable. The early diagnosis of Ménière's disease should influence the therapeutic decisions, and, of course, the early detection of a probable bilateralism of this affliction should influence the decision for a destructive surgical approach.

Many methods have already been proposed to achieve this goal but, to this day, the only well-documented method is ECoG. ECoG is a rather invasive method, with a sensitivity of 60% to 80% in patients with Ménière's disease.[12] Whether you choose invasive-type electrodes, the method is difficult to perform, it needs highly trained personnel, and, sometimes, it can be poorly tolerated by patients. According to our results, a rough estimate of the sensitivity of our method is about 68.6%, and this is consistent with that of ECoG. The advantage of our method is that it is noninvasive, easier to perform, and is very well tolerated by patients.

In the present study. the masked threshold shows a maximum at a phase of 250° and a second maximum at 90°. At 250°, the basilar membrane shows the largest displacement toward the scala tympani, and at 90°, it shows the largest displacement toward the scala vestibule.

The results obtained by this method are in agreement with that of Mrowinski and colleagues, 14 except in the phase where the maximum masking effect is exerted (250° contrary to 270°). This phenomenon may reflect the interference of the 2 waves (masking and tone burst) in the same space, which is much bigger than that of the Mrowinski dedicated earplug. In addition, the emission of the 2 sounds is simultaneous from the same transmitter. Our method, carried out in vivo, does not fall short of clinical diagnostic accuracy, as it is capable of diagnose the basilar-membrane

"stiffness" reflected at an almost-null modulation depth.

As regards clinical accuracy of the measurements based on the modulation depth, it is possible to say they are capable of diagnosing the majority of Ménière's disease cases. A smaller percentage of diagnoses may be confused with that of patients with noise-damage hearing loss, probably due to enhanced recruitment. Of course, the combination of this method and pure-tone audiometry enhances the diagnostic accuracy.

Our method has a relatively good sensitivity in diagnosing the early stages of Ménière's disease. Some additional work has to be done in order to enhance the sensitivity of the method.

In conclusion, the instrumentation and method we developed distinguishes and diagnoses the early stages and acute recurrences of Ménière's disease. The instrumentation needed is easy accessible and it can found in any medical physics or engineering laboratory. This may lead to further study of the results of low-frequency masking on the anterior labyrinth from research centers worldwide, in order to reinforce the diagnostic accuracy and strength of our newly developed diagnostic method.

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