Chapter

Hearing and Vestibular Testing in Menière's Disease

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Abstract

Endolymphatic hydrops (ELH) known as the main pathophysiology of Menière's disease (MD) changes both the cochlear and vestibular function of the inner ear. These physiological changes can occur simultaneously (cochleovestibular involvement) or separately (cochlear or vestibular involvement). They can also present unilaterally or bilaterally (simultaneously or sequentially). Moreover, ELH recurs periodically without any specific etiology and known rhythm. Therefore, the patient referred for audiological tests may be in attack phase (acute) or inter-attack phase (chronic). MD itself may be in early- or advanced stage. In addition, considering comorbidity (vestibular or non-vestibular) is vital for differential diagnosis. On the other hand, each audiological test (including PTA, ECochG, VNG, vHIT, SVV, VEMPs ...) has its specific diagnostic viewpoint and gives us a limited snapshot of MD's clinical picture. Consequently, in this chapter, we want to discuss these viewpoints and try to explain associations and dissociations of audiological test findings in MD patients.

Keywords: endolymphatic hydrops, pure-tone audiometry, videonystagmography, electrocochleography, video head impulse test, subjective visual vertical, vestibular evoked myogenic potential

1. Introduction

Menière's disease (MD) is an inner ear (or labyrinthine) disease [1]. It described by a French physician, Prosper Ménière, in 1861. Through observing a group of patients, he realized that symptoms such as spells of spontaneous vertigo attacks, occurrence of positional vertigo between attacks, tinnitus, and hearing fluctuations that do not necessarily occur together might be the clinical picture of a single disease. He was the first to emphasize that vertigo could be caused by a damage to the inner ear [2].

As an ear disease, with episodic vertigo and fluctuating aural symptoms (hearing, tinnitus, or fullness) [3], MD patients usually referred to audiology (or vertigo) clinics. However, we know that each audiological test has its specific diagnostic viewpoint. Therefore, each test shows us a snapshot of real disease.

MD can present in one or both ears (simultaneously or sequentially). Moreover, it is not a stable disease; it usually occurs in "attack-inter attack pattern" (i.e., it relapses) and progresses to different parts of inner ear (s) (i.e., cochlea, semicircular canals, saccule, or utricle). In each attack, new destruction or distortion can occur [4]. Hence,

the perspective of inner ear changes after each attack. This scene may return to previous state in inter-attack period.

Does destruction occur the same for all parts of inner ear(s)? Does recovery happen the same for all parts of inner ear(s)? What is the best time for testing the patient after the attack? What are the best tests to achieve the diagnosis? Do we have specific and sensitive tests for diagnosing the hydrops in each part of inner ear?

In the following, we discuss the viewpoint achieved by each audiological test and try to explain associations and dissociations of their findings in MD patients.

2. General auditory viewpoint

2.1 Introduction on pathophysiology and auditory tests

Endolymphatic hydrops (ELH) is the main pathophysiology of MD affects hydrodynamics of inner ear fluids [5]. As a result, it changes the mass and stiffness (as well as the resonance frequency) of labyrinthine compartment. We expect that cochlear hydrops resulted in auditory and aural symptoms such as hearing loss, tinnitus, fullness, and hyperacusis (vestibular hydrops discussed in the Section 3).

It seems that cochlear hydrops decreases the distance between stapes and endolymphatic space. The latter contains basilar membrane (BM), which holds outer hair cells (OHCs) and inner hair cells (IHCs). Thus, cochlear hydrops alter the activation of OHCs, IHCs and their afferents (and probably their efferents too). These alterations can explain auditory and aural symptoms of MD. Can we track the alterations (i.e., due to cochlear hydrops) using audiometry, otoacoustic emissions (OAEs), electrocochleography (ECochG), auditory brainstem response (ABR), or cochlear hydrops analysis masking procedure (CHAMP)?

On the other hand, cochlear hydrops increases the outward pressure on stapes and ossicular chain of middle ear. Accordingly, it changes the inner ear perspective concomitantly with changing the perspective of middle ear. Can we track these changed perspectives using a technique such as wide-band tympanometry (WBT)?

In the following sections, we want to sketch these auditory viewpoints about MD.

2.2 Audiometry and aural symptoms

We can consider general auditory profile (i.e., audiogram) as the main audiological test that aids in diagnosis and monitoring of MD. The Barany society (2015) introduces two categories for MD: definite and probable. Two or more episodes of spontaneous vertigo (lasting 20 min to 12–24 h) and fluctuating aural symptoms (hearing, tinnitus, or fullness) are common between two categories. Nevertheless, in definite MD patients, sensorineural hearing loss (SNHL) in low to mid audiometric frequencies, at least in one ear and on one occasion, is an additional diagnostic criterion [3]. Consequently, it seems vital to acquire periodic audiograms to diagnose the MD and its progression behavior.

Is there a specific pattern for hearing loss in this disease? What is the pattern of progress? Do we have a classification for different stages of the disease?

Several audiometric configurations are proposed in MD: peak audiogram (normal hearing in 2000 or 3000 Hz), rising (low frequency SNHL), falling (descending, or high frequency SNHL), trough, atypical notch (in 1 or 2 KHz, based on our experience; see **Figure 1**), and flat SNHL. In early stage of MD, peak and low-frequency

audiograms are more common. However, in the advanced stage, flat audiogram (with or without more high-frequency loss) arises [6]. In AAO-HNS guidelines (1995), four stages of MD classified based on pure-tone audiometry average (PTA): stage I, <26 dB; stage II, 26–40 dB; stage III, 41–70 dB; stage IV, >70 dB [7].

In the course of disease, it seems that the hearing loss increases from about slight-to-mild (low-frequency SNHL) in early stage to moderate-to-severe (flat SNHL) in advanced stage. Some studies showed a significant correlation between the grade of hydrops and severity of hearing thresholds in low and mid audiometric frequencies. Moreover, the audiogram fluctuates with the average of 20–30 dB. The fluctuation

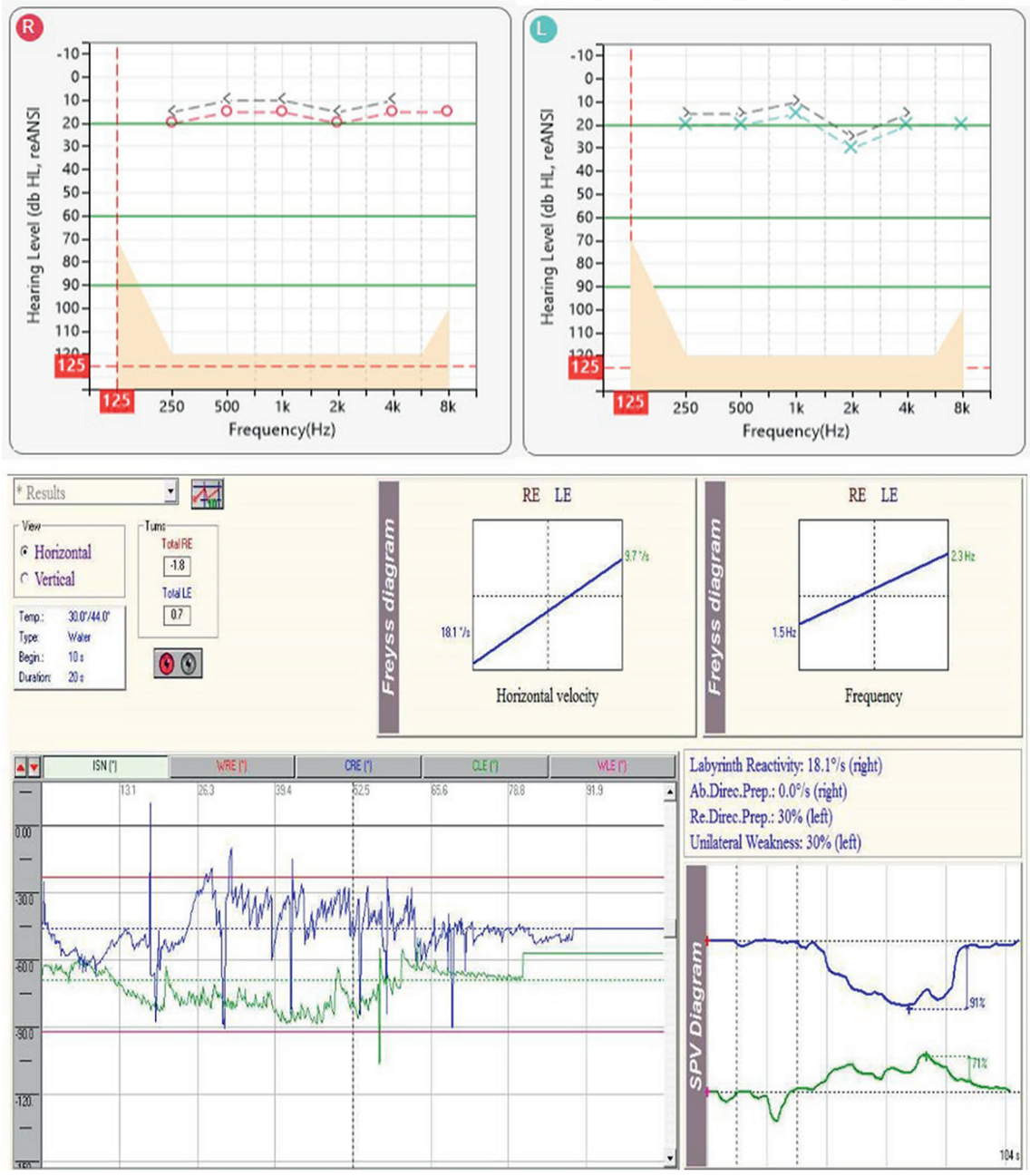


Figure 1.
Upper part: Audiogram of a female patient (52y) with 7 years' history of episodic vertigo attacks that occurred in every 2 months. As you can see, we have only a 2 KHz notch in the left ear (i.e., the ear with aural fullness and tinnitus). Lower part: The picture shows the caloric test results (SYNAPSYS VNG software by Inventis srl.). Caloric result of the same patient with significant vestibular weakness in the left ear (left-UW). Note. The patient had normal middle-ear function and normal vHIT results.

is more during the early stage (perhaps the first year following MD onset) and then subsides [1, 5–6, 8]. The frequency of bilateral MD is about 2–47% [9].

2.3 Mechano-acoustic viewpoint

The ear is composed of three main parts: outer ear, middle ear, and inner ear. In these three parts, exactly before mechanoelectrical transduction by hair cells, we are facing with mechanical energy of incident sound power. Part of this power reflected back and part of that absorbed in the ear. We can track changes in power reflectance and power absorbance of the middle ear by wide-band tympanometry (WBT).

2.3.1 Wide-band tympanometry

WBT is a method for evaluating middle-ear diseases such as otitis media with effusion (OME), otosclerosis, and ossicular chain discontinuity. Compared to the traditional 226-Hz tympanometry, WBT uses multiple frequencies (ranging from to 8000 Hz) in 1/24 octave intervals through a descending pressure sweep (between +250 and − 350 daPa). Therefore, it is more sensitive to middle-ear transfer function [10–11].

By increasing perilymphatic pressure, ELH can push the stapes footplate toward the middle ear. Thus, it restricts the movement of the ossicular chain and decreases the compliance of the middle ear [11]. This condition is almost similar to the OME (among other middle-ear pathologies). From another viewpoint, ELH decreases the impedance of cochlear part, just like large vestibular aqueduct syndrome (LVAS) (among other inner ear pathologies). Consequently, studies showed that resonance frequency of the middle ear decreased in MD [11], LVAS [12], and OME [13]. Moreover, power reflectance in high frequency range (2–4 KHz) increased in MD patients [14]. Therefore, WBT has opened a new window on inner-ear pathologies.

2.4 Electro-acoustic viewpoint

The cochlea emits some sounds spontaneously or in response to an external auditory stimulus. These sounds are called spontaneous otoacoustic emissions (SOAEs) or evoked OAEs (EOAEs), respectively.

2.4.1 Otoacoustic emissions

OAEs provide a tool for evaluating the function and integrity of outer hair cells (OHCs). Presence or absence of OAEs, their amplitudes in different frequencies, and suppression of them through contralateral noise are valuable cues for delineating OHC loss or dysfunction, estimation of audiogram, and testing auditory efferent system, respectively. Do these cues change in Ménière disease?

In MD patients, OAE features are usually compatible with the level of hearing loss. In other words, it does not differ from other patients with the same level of sensorineural hearing loss [15]. However, in some of MD patients with hearing level between 30 and 60 dB, OAEs are normally present. Based on these cases, it is possible that IHCs become involved sooner due to ELH [16]. On the other hand, a feature of OAE (or OHC function) that we have not yet evaluated or do not know may have changed in MD patients. A study by Murdin and cowerkers (2010) showed that DOPAE suppression is lower in patients with vestibular migraine [17].

Suppression of OAEs needs acetylcholine (Ach). Ach is inhibited by calcitonin gene-related peptide (CGRP) activity. Increased activity of CGRP in the migraineurs' inner ear could result in an abnormal OAE suppression [18]. Future studies will show whether we can use OAE suppression to differentiate between MD and vestibular migraine patients.

A case study by Chun and colleagues (2009) revealed that OAE recovery is a tool for monitoring treatment in patients with luetic endolymphatic hydrops (due to syphilis) [19]. Consequently, OAE features, OAE suppression, and OAE recovery provide important toolbox for evaluating and monitoring the diseases that involve inner ear.

2.5 Auditory electro-physiologic viewpoint

There are several electro-physiologic tests, which analyze auditory responses and contain good information for diagnosis of ELH. In this section, we discuss about electrocochleography (ECochG) and cochlear hydrops analysis masking procedure (CHAMP).

2.5.1 Electrocochleography (ECochG)

In ECochG test, we present an acoustic stimulus (usually click or tone burst) via a headphone (or insert phone) and record a two-component response through several leads (electrodes) around the ear (i.e., extratympanic method). One component originates from cochlear part, mainly inner hair cells (named summating potential or SP), and the other component evokes by auditory nerve fibers (named compound action potential or AP). The hypothesis is that ELH can deform basilar membrane and affect the latency and amplitude of these two components [20].

There are two ratio methods for analyzing ECochG results: 1. SP/AP amplitude ratio; and 2. SP/AP area ratio [21].

By defining latency and amplitude of SP and AP components, we can measure SP/AP amplitude ratio. For latency measurement, we need the stimulus onset (zero time); and for amplitude measurement, we need a baseline (BL). Enhancement of SP/AP amplitude ratio is a positive indicator for ELH [21] (**Figure 2**). Baba et al. (2009) reported 0.314 as upper limits of normal for SP/AP amplitude ratio [22]. Based on previous studies, it seems that the ratio of more than 0.37 (or less than 0.5 as stated by Gibson [23]) has a low sensitivity (less than 60%) but an acceptable specificity (around 90%) for diagnosing MD [20, 23]. Using 1-KHz tone burst and through transtympanic recording, which can make the response four times bigger, the audiologist can reach a higher sensitivity for diagnosing MD (around 79%) [23].

Several factors related to the subject (i.e., MD patient) affect the usefulness of this ratio that include being in attack or inter-attack phase, in early or advanced stage of disease, and the degree of hearing loss. Therefore, this criterion may only work in half of the MD patients [20].

Considering SP/AP area ratio can improve the sensitivity and specificity of the diagnosis. Baba et al. (2009) reported 1.56 as upper limits of normal for this ratio. They reported area ratio of 1.97 ± 0.55 in definite MD patients [22].

Finally, it should be noted that although the specificity of the ECochG results (the amplitude and area ratios) is high, but sometimes the test is positive in similar diseases such as superior semicircular dehiscence (SSCD) [24] and vestibular migraine [25].

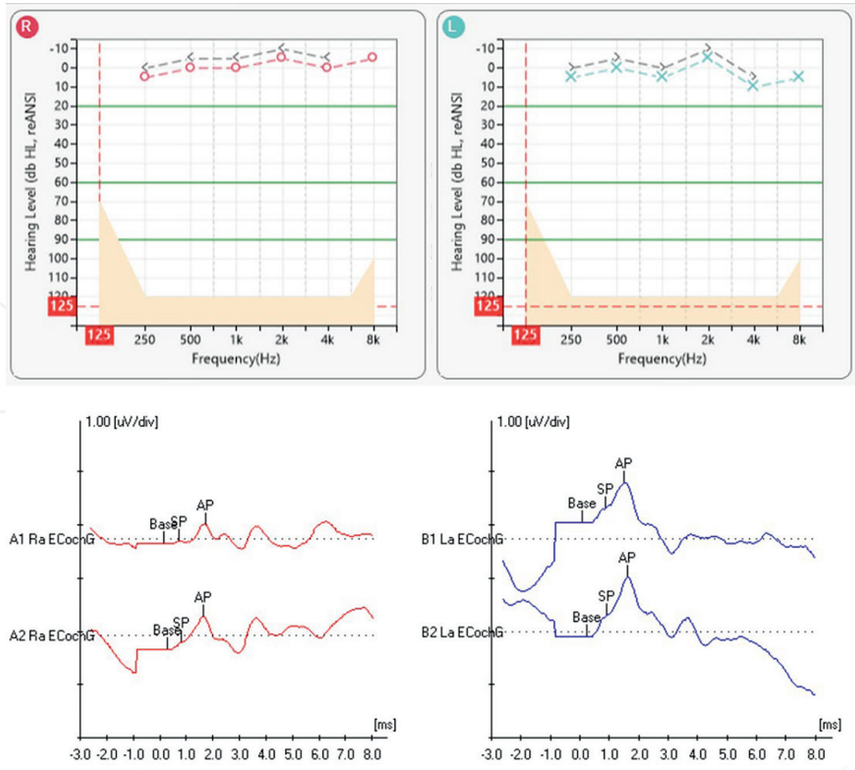


Figure 2.
Upper part: Audiogram of a female patient (39y) with 3 years' history of episodic vertigo attacks (in every 4–5 months) associated with nausea and left-ear tinnitus. She had normal audiogram and normal middle-ear function. Lower part: ECochG results. The amplitude ratio of SP/AP was 29% for A2-trace (normal ear) and 44% for B1-trace (disordered ear).

2.5.2 Cochlear hydrops analysis masking procedure (CHAMP)

ELH can affect the traveling wave velocity (TWV). Previous studies showed that TWV increases in MD patients [20]. By comparing wave V latency of auditory brainstem response (ABR) in response to click-only stimulus, and click-plus-high pass

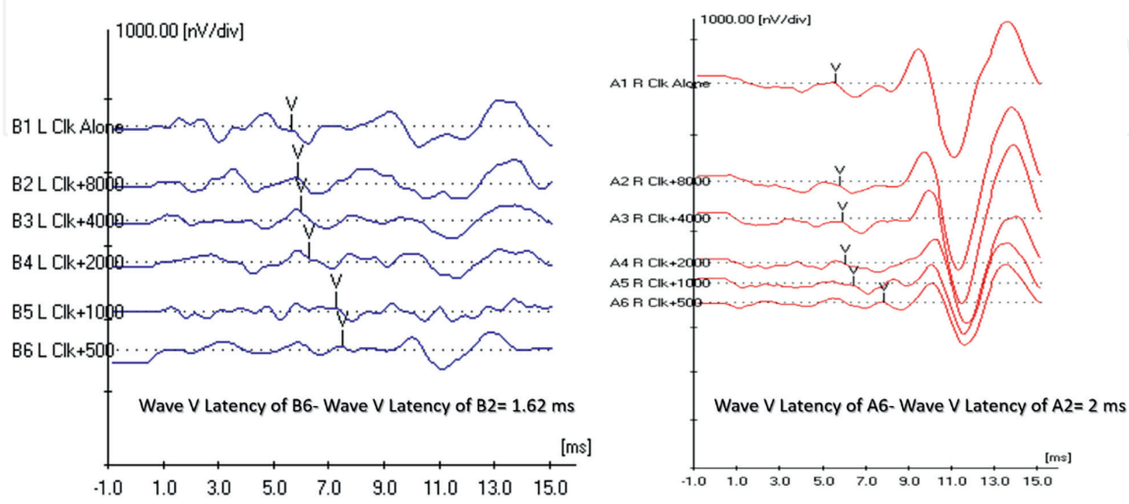


Figure 3.
CHAMP results of the patient presented in the **Figure 2.** *Note the decreased difference between the wave V latency of basal part and that of apical part in the left ear with a history of MD.*

noises, we can measure the effect of increased TWV in MD patients. It means that different noises mask different parts of basilar membrane and permit us to differentiate the response of basal part of cochlea from its apical part.

Increased TWV resulted in decreased latency difference between the wave V latency of basal part and that of apical part (**Figure 3**). This is really occurs in MD patients. However, the CHAMP test has only high sensitivity and specificity in definite MD patients [26].

3. General vestibular viewpoint

ELH as the main pathophysiology of MD affects hydrodynamics of vestibular system (semicircular canals and two otolithic organs including utricle and saccule) too. We expect that vestibular hydrops resulted in vestibular symptoms such as dizziness, vertigo, nausea, vomiting, motion sensitivity, and imbalance.

It seems that vestibular hydrops changes the distance between cochlear and vestibular organs, as well as between inner ear and middle ear. Like as cochlear part, vestibular end organs have also specialized sensory cells including type I and type II hair cells. Thus, vestibular hydrops alters the activation of type I, type II and their afferents (and probably their efferents too). These alterations can explain vestibular symptoms of MD. We can categorize them as vestibulo-ocular and vestibulo-spinal symptoms mediated by neural pathways of vestibulo-ocular reflex (VOR) and vestibulo-spinal reflex (VSR), respectively. It is worthy to note that vestibulo-collic and vestibulo-sympathetic reflexes have important roles in balance system, too.

The vestibular system differs from auditory system in that the right and left vestibular systems have functional pairing. It means that they function as two interconnected scale pans. This intra-system connection (or functional pairing) exists in both peripheral vestibular system (from vestibules up to vestibular nuclei in brainstem) and central vestibular system (from vestibular nuclei toward cortical vestibular regions). Therefore, vestibular hydrops can affect this functional pairing. The pairing deficiency can track using cervical vestibular evoked myogenic potentials (cVEMP), ocular vestibular evoked myogenic potentials (oVEMP), videonystagmography (VNG), video head impulse test (vHIT), and subjective visual vertical/horizontal (SVV/AVH) tests.

Another unique feature of vestibular system is that it is a sensory input for our global balance system. Other sensory inputs for balance include visual and somatosensory ones. These three main inputs work as a whole-interconnected system to keep us balanced (i.e., we have inter-system functional pairing, too). Thus, vestibular hydrops can affect the global balance of the patient. Using posturography test, we can evaluate the balance of the MD patient.

In the following sections, we want to sketch these vestibular viewpoints about MD.

3.1 Vestibular electro-physiologic viewpoint

Interestingly, we can record the sound-evoked responses of the vestibular system by placing electrodes on the neck muscle (i.e., sternocleidomastoid muscle or SCM) or below the eyes (i.e., inferior oblique muscle). The former mode records cervical vestibular evoked myogenic potentials (cVEMP), and the latter mode records ocular vestibular evoked myogenic potentials (oVEMP).

3.1.1 Cervical vestibular evoked myogenic potentials (cVEMP)

cVEMP is a saccular response. Therefore, it evaluates the function of inferior vestibular nerve and sacculocollic reflex. cVEMP response is a biphasic electrophysiological response named p13-n23, based on the occurrence latency of each component [27–28]. Recent study by Shahnaz and David (2021) on cVEMP that performed in supine position with raised and turned head showed normal latency range of 16 ± 1.08 ms for p1 and 24.6 ± 1.98 ms for n1 [29].

cVEMP is an ipsilateral inhibitory response. It means that presentation of each acoustic stimulus (usually click or 500 Hz tone burst) for example, to the right ear instantaneously decreases the tonus of contracted right SCM. This change induces a potential in the recording electrode placed on the SCM. The average of them in response to several stimuli (usually about 50–200 stimulus) creates p1-n1 response. Using BC stimulus (via a bone oscillator) can provoke a bilateral response. The exact placement of oscillator determines the latency and amplitude of each response [20, 30].

Usually, we record cVEMP unilaterally and compare the amplitude of responses (i.e., from the peak of p13 to trough of n23) recorded from each side (**Figure 4**). This analysis is called interaural asymmetry ratio (IAR). The upper limit of normal for IAR is about 0.4 [31]. In some reports, abnormal IAR is considered as more than 0.33 [32]. Based on IAR and the symptomatic ear (as well as historical background), we can define the augmented side (i.e., an increased VEMP response that indicated a hyperactive or hypersensitive saccule) or the reduced side (i.e., a decreased or absent VEMP response that indicated a hypoactive or hyposensitive saccule).

An increased and decreased/absent VEMP responses reported in the early and late stages of MD, respectively. Saccular hydrops and saccular degeneration proposed for explaining these results, respectively [27]. Moreover, interaural differences such as IAR and threshold difference between left and right VEMPs, usually affected by the fact that the asymptomatic (or unaffected) ear might have some abnormalities (i.e., subclinical MD). For pointing this problem, the tuning curve method suggested. In this method, amplitude of 500 Hz VEMP compared with that of 1000 Hz in the same ear [33]. The ratio decreased in MD patients (i.e., flattening of the tuning curve occurs because in hydropic

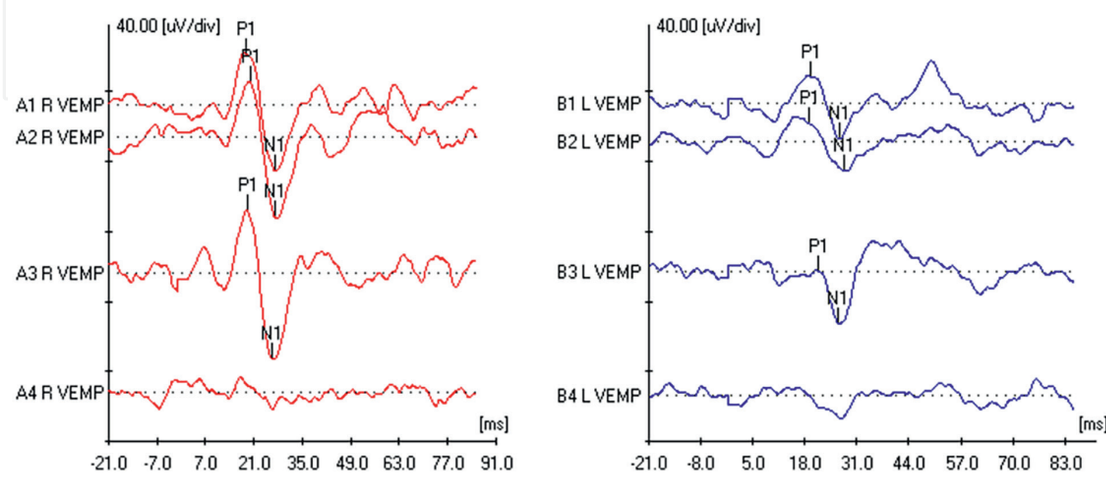


Figure 4.
cVEMP results of the patient presented in the **Figures 2** *and* **3**. *Note the interaural asymmetry ratio (IAR) of −48% (i.e., smaller VEMP response in the left ear with a history of MD).*

ear, endolymphatic tuning shifts to 100 Hz). They showed that this ratio could be a marker for MD progression because the ratio is high during acute phase and is normal during intermediate stable phase [33]. Of course, the ambiguity is whether we are going to use the test to determine the disorder or to use the test to grade the disorder we already know.

In any case, the point of specificity that mentioned above about ECochG raised again. cVEMP threshold is a suitable parameter for distinguishing SSCD from MD, but overlaps with vestibular migraine remains a clinical challenge [20].

3.1.2 Ocular vestibular evoked myogenic potentials (oVEMP)

oVEMP is an utricular response. Therefore, it evaluates the function of superior vestibular nerve and the utriculo-ocular reflex. oVEMP response is a biphasic electrophysiological response (n1-p1; n10-p15) [27, 34]. Recent study by Shahnaz and David (2021) with a new electrode montage named nasal alar montage found a shorter latency for n1 (12 ± 1.67 ms) and p1 (16 ± 1.72 ms) in comparison with traditional infraorbital montage [29].

oVEMP is a contralateral excitatory response provoked by excitation of contralateral inferior oblique muscle (re. stimulus ear). It should be noted that almost everything that was said about cVEMP in MD is true here for oVEMP as well [20, 30]. However, the different origins of these two VEMP responses can help us to differentiate vestibular disorders or to estimate the extent of the disease. In fact, it is better to record both responses, even with both AC and BC stimuli, to achieve the best clinical picture of the patient.

3.2 Vestibulo-ocular viewpoint

We know that the involuntary eye movements, which can occur due to head movements, follow vestibular stimulation (i.e., excitation and inhibition of both peripheral and central vestibular systems in the frames of intra-system and inter-system functional pairing). Therefore, unilateral vestibular disorder, or asymmetric bilateral vestibular disorder, can cause involuntary eye movements when the head is at rest. These eye movements are vestibular-evoked nystagmus.

Accordingly, by examining eye movements during head rest, head movements, or positioning the head, or while stimulating the vestibular system, for example, by thermal (caloric), vibrational, or electrical stimulation, the function of VOR reflex and the occurrence of abnormal eye movements (such as compensatory saccades or different types of nystagmi) can be examined. For this purpose, we use videonystagmography (VNG) test battery (SYNAPSYS VNG by Inventis srl) and video head impulse test (vHIT) (SYNAPSYS VHIT by Inventis srl).

The otolith system detects and provides us the real vertical axis of gravity. Based on this, and with the help of our real-world experiences from childhood, and the benefit of intra-system and inter-system pairings, we can understand other degrees of tilt in space; i.e., the tilt of ourselves, which can also be extended to the tilt of objects in our surrounding world. Our perception of the real vertical and horizontal axes is a manifestation of the balance in this complicated system. For testing this perception in general (and not precisely!), we can use subjective visual vertical/horizontal (SVV/SVH) tests.

3.2.1 Videonystagmography (VNG)

For performing VNG test battery, a goggle with infrared camera(s) placed over the patient's eyes. VNG test battery consists of two categories: 1. Central oculomotor

tests that have a visual target and 2. Peripheral vestibular tests that do not have any target. The first category includes saccade, smooth pursuit, optokinetic, and gaze tests (here, we will not discuss their results for MD diagnosis). The second category includes spontaneous nystagmus test (SNT), head-shaking test (HST), Dix-Hallpike test (DHT), Positional test (PT), and Caloric test (CT). Instead of head shaking, we can use vestibular vibrator to assess vibration-induced nystagmus (VIN).

In peripheral tests, we are looking for abnormal nystagmus. Unilateral MD (or bilateral asymmetric MD), especially in the acute (or active) phase, can make an imbalance in vestibular system. This imbalance can manifest as spontaneous nystagmus (SN) (**Figure 5**). Bery et al. visited an interesting MD patient during vertigo attack and recorded initial "irritative" nystagmus that beats toward affected ear and after about 2 minutes of onset reverses its direction toward the unaffected ear ("paralytic" nystagmus) [35]. Following the latter nystagmus, and about 3 days after onset (or within the acute phase, a recovery nystagmus toward the affected ear can occur in some MD patients [35]. For that reason, in interpreting the results of SN, the audiologist should pay attention to the disease phase.

Moreover, the imbalance may exhibit itself as head-shaking nystagmus (HSN) (**Figure 6**). HSN is reported in about 68% of MD patients. However, HSN is more recordable in the acute phase and has a diverse direction. Its direction may be toward or away from the affected side or may be biphasic [36–37]. Therefore, for its correct interpretation, the patient's symptoms and the results of other tests should be considered.

On the other hand, the imbalance may exhibit itself as VIN (**Figure 7**). VIN is reported in 28–71% of MD patients. It is often recordable in the acute phase and mostly as the irritative type (i.e., beating toward the affected ear) [38]. In our study on 29 patients with unilateral chronic MD tested in inter-attack phase, we found that vibrational stimulation of the affected ear with 100 Hz stimulus (mastoidal stimulation)

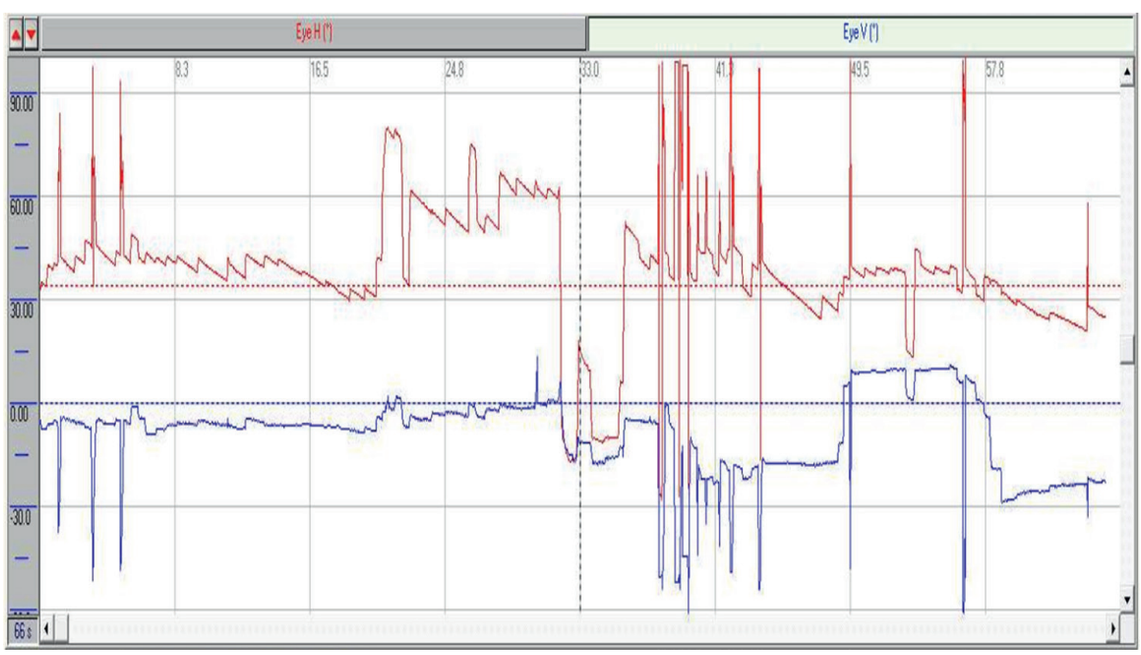


Figure 5.
The picture shows spontaneous nystagmus (SN) test results (SYNAPSYS VNG software by Inventis srl.) of a male patient (29y) with 5 years of MD symptoms in his right ear. Note the right-beating SN (with SPV of about 5 degree/seconds). His last attack occurred 2 days before recording this VNG test. This type of SN can be considered as recovery nystagmus. Please see the text for more discussion.

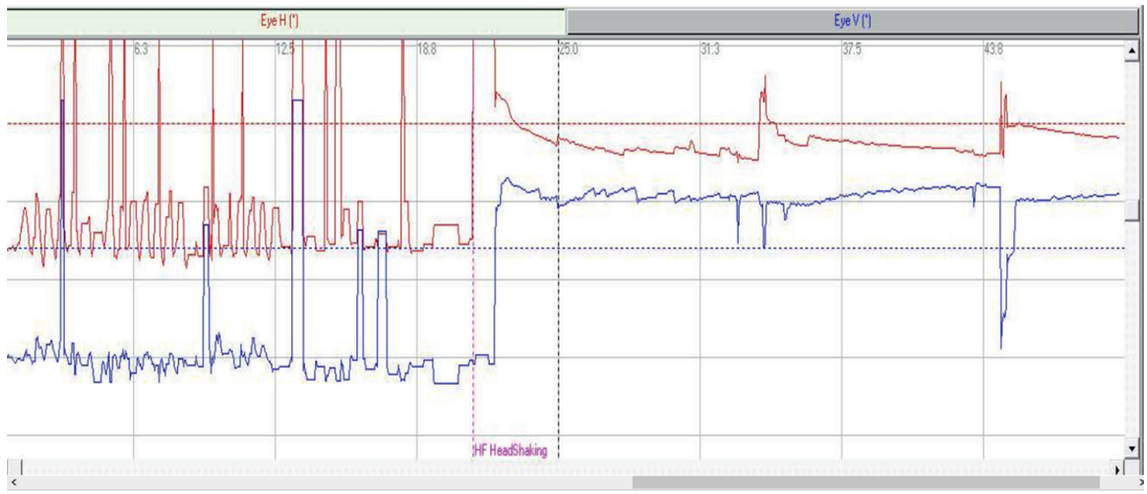


Figure 6.
The picture shows head-shaking nystagmus (HSN) test results (SYNAPSYS VNG software by Inventis srl.) of the patient presented in the **Figure 1***. Note the right-down-beating HSN (with SPV of about 0.7 degree/seconds in both directions).*

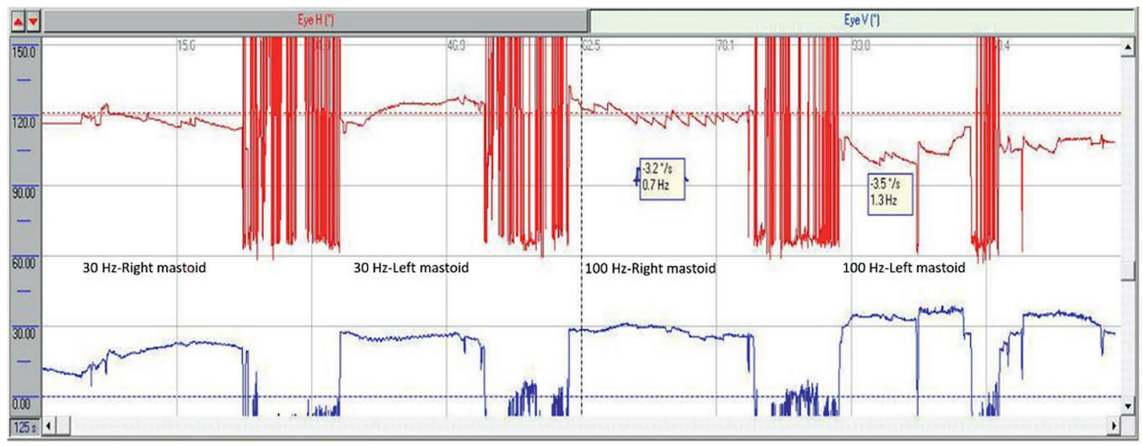


Figure 7.
The picture shows vibration-induced nystagmus (VIN) test results (SYNAPSYS VNG software by Inventis srl.) of a 45y female patient referred for her 5 years' history of vertigo attacks (about four attacks) associated with nausea and left-ear tinnitus. She had moderately severe SNHL in the left-ear. As you can see, the right-beating VIN provoked by performing 100 Hz vibration on both mastoids.

provokes more reliable VIN (average of 3.46°/s) compared with 30 Hz stimulus or stimulation of the unaffected ear [39]. Thus, this test can also help in MD diagnosis.

Additionally, the imbalance may present as unilateral weakness (UW) in the caloric test (**Figure 8**). Previous studies showed abnormal caloric results in 42–76% of MD patients [40].

Sometimes, the SN can also be seen (or even provoked) in Dix-Hallpike and/or positional tests. It is worthy to note that in some patients in attack phase, a direction-changing nystagmus can occur [41]. Consequently, we must be careful to distinguish these patients from others with benign paroxysmal positional vertigo (BPPV) or central type of positional vertigo.

The interesting thing to note is that the vestibular system also has a certain operating frequency. Some tests, such as caloric, cover a very low frequency range of the system (0.002–0.004 Hz), and some tests, such as vHIT, cover its high frequency range (5–7 Hz) [42]. Head-shaking test stimulates the vestibular system in the range

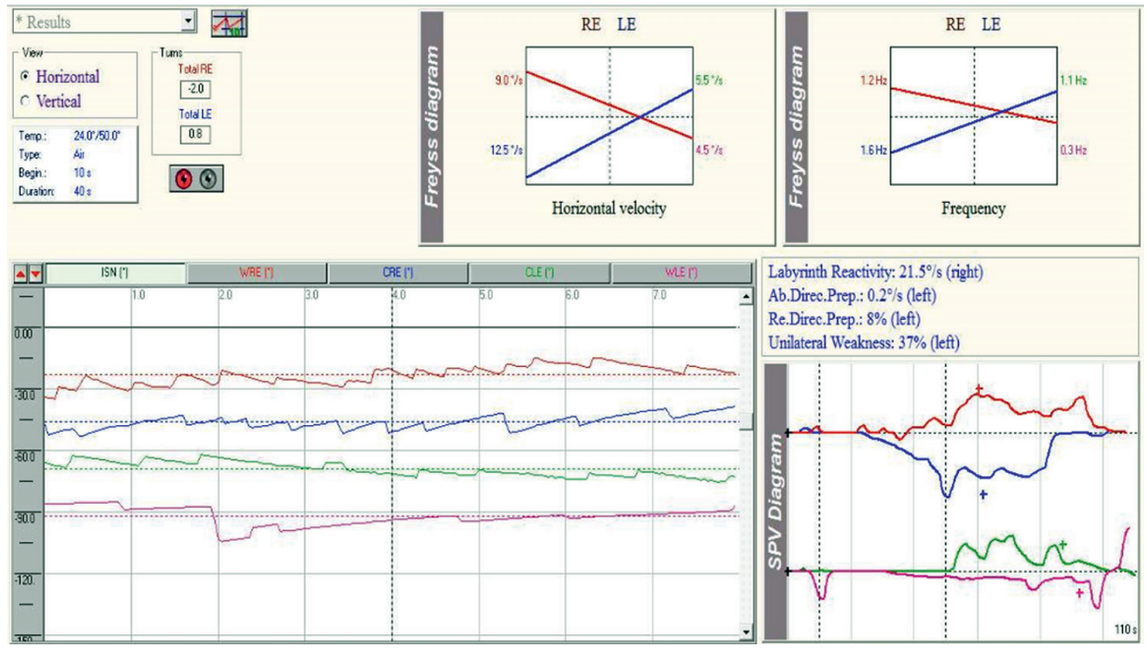


Figure 8.
The picture shows bi-thermal caloric test results (SYNAPSYS VNG software by Inventis srl.) of the patient presented in the **Figure 7**. *Note the significant vestibular weakness in the ear (disordered side).*

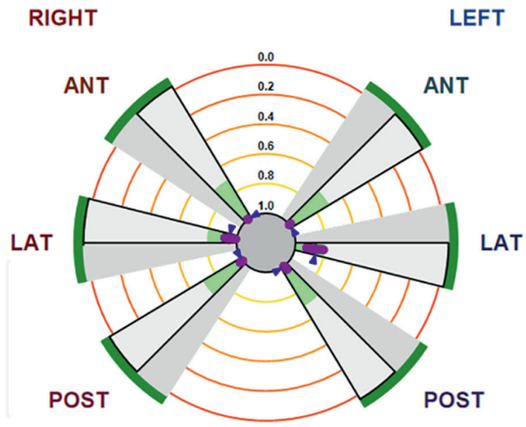
of 1–2 Hz, and vibrational stimulus provokes the system in the range of 10–100 Hz [38]. Therefore, we can consider them as complementary tests that incite different population of vestibular hair cells and reinforce the diagnosis process.

3.2.2 Video head impulse test (vHIT)

During vHIT (or VHIT), a goggle with infrared camera placed on the patient's eyes or an infrared camera placed in front of the patient. The audiologist moves the patient's head quickly and unexpectedly in the horizontal and vertical planes to test horizontal and vertical semicircular canals (SCCs) and their neural pathways. In fact, six different movements are performed to test each of the six semicircular canals. The patient must keep his/her gaze on a front visual target. If the specific SCC works correctly, proper VOR will occur and the gaze will remain on the visual target. If the specific SCC does not work correctly, proper VOR will not occur and instead, corrective saccades will occur. VOR gain (eye velocity/head velocity) of >0.8 for horizontal SCCs and of >0.7 for vertical SCCs considered as normal response. Corrective saccades that occurred during head impulse (about 150 ms) or after head impulse called covert and overt saccades, respectively [40, 43–44].

Several studies showed that vHIT results are usually normal in MD patients (**Figure 9**). On the other hand, a study by Lee et al. that analyzed HIT responses by magnetic search coil during MD attack showed that there is variation in the HIT results in terms of both the involved SCCs and the involved ear. Alongside, caloric results usually show some weakness on the affected side [45]. This dissociation may be due to how the vestibular system is stimulated during each test and cell types that involved by Meniere's disease [43, 45].

Anyway, as mentioned above, vHIT and caloric test are complementary for MD diagnosis.



Impulses			VOR			Early saccades		
Canal		n	Mean gain	σ	Asymmetry	Ratio	Mean latency	Mean apparent gain
Ant.	R	10	1.11	0.03	2 %	0 %		
	L	10	1.07	0.04		0 %		
Lat.	R	12	0.95	0.02	5 %	0 %		
	L	15	0.86	0.03		0 %		
Post.	R	10	1.04	0.04	1 %	0 %		
	L	10	1.03	0.03		0 %		

Figure 9.
The picture shows video head impulse test (VHIT) results (SYNAPSYS VHIT software by Inventis srl.) of the patient presented in the **Figures 7** *and* **8***. Note the normal VOR function in the plane of all six semicircular canals. However, sometimes a slight asymmetry can be seen between the results of the two sides, as presented in this case (VOR gain of 0.95 versus 0.86 for the right- and left-lateral SCCs, respectively).*

3.2.3 Subjective visual vertical/horizontal test (SVV/SVH)

During SVV/SVH, a luminous line projected on a screen and the patient must perceptually adjust it in vertical/horizontal plane. An error of more than ±2 degrees from the actual vertical or horizontal determined by the software is considered abnormal. This test can be performed in the upright or tilted head position; the latter is more sensitive to otolithic function [46].

In Meniere's disease, the results usually show tilt toward the affected ear, particularly in the acute phase. The results can be used to monitor the effects of surgery, treatment, or rehabilitation on the otolith function recovery [47–48].

3.3 Vestibulo-spinal viewpoint

3.3.1 Posturography

Vestibulo-spinal function can be assessed by sensory organization test (SOT). SOT is a functional balance evaluation performed by posturography device. Posturography measures the postural sway and its parameters such as the direction, amplitude, velocity, and frequency [49]. The use of SOT may be helpful in the differential diagnosis of vestibular migraine from Meniere's disease [50].

SOT, caloric, and VHIT have a complementary role in evaluating VOR and VSR functions.

4. Conclusions

We have no specific test as "Meniere test," and its classification remains symptom-based. However, we often face with under-diagnosis or over-diagnosis. On the other hand, each audiological test has its own sensitivity and specificity in diagnosis of MD. Therefore, in addition to the symptoms, we must use a suitable and complementary set of hearing and vestibular tests to achieve the best diagnosis. We can look at MD from different perspectives that include: audiometry and aural symptoms,

mechano-acoustic viewpoint, electro-acoustic viewpoint, auditory electro-physiologic viewpoint, vestibular electro-physiologic viewpoint, vestibulo-ocular viewpoint, and vestibulo-spinal viewpoint.

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