Role of Clinical and Instrumental Evaluation in Patients with Vestibular Migraine: a Narrative review.

Augusto Pietro Casani[1], Nicola Ducci[1]

[1]Department of Surgical and Medical Pathology, ENT section, Pisa University Hospital, Pisa, Italy.
ORCID IDs of the authors: A.P.C.: 0000-0001-5261-9129.
Corresponding author: Augusto P. Casani, M.D., Associate Professor, Department of Medical and Surgical Pathology, ENT Section, Pisa University Hospital, Via Paradisa, 2, 56126 Pisa, Italy;
e-mail: augusto.casani@unipi.it

Abstract
Vestibular migraine (VM) is one of the most common causes of recurrent episodic vertigo, yet it remains a diagnostic challenge because of its marked clinical heterogeneity and the frequent dissociation between vestibular symptoms and headache. Patients may present with spontaneous, positional, visually induced, or motion-triggered vertigo, often in the absence of concurrent migraine headache, leading to under recognition or misdiagnosis in both otoneurological and neurological settings. VM pathophysiology is complex and multifactorial, involving abnormal sensory integration within vestibulo-thalamo-cortical networks, brainstem dysfunction, and altered ion channel and neurotransmitter activity shared with migraine mechanisms. Central vestibular hypersensitivity, impaired habituation, and transient asymmetries in vestibular processing appear to play a key role. Neuro-otological findings are often subtle and variable, ranging from normal examinations to transient central-type nystagmus, positional vertigo mimicking benign paroxysmal positional vertigo, and abnormal ocular motor responses observed during attacks or interictal periods. Diagnosis is currently based on clinical criteria, emphasizing the temporal association between vestibular symptoms and migraine features or history, while instrumental testing mainly serves to exclude alternative vestibular disorders. Triggering factors such as stress, sleep disturbances, hormonal fluctuations, dietary elements, and sensory overload frequently modulate attack occurrence and severity. In this review, we critically reappraise vestibular and neuro-otological instrumental findings and discuss their potential diagnostic value in VM, particularly in clinical scenarios that do not fully satisfy the 2012 diagnostic criteria. Selected instrumental patterns, when interpreted within a pathophysiological framework, may support a more accurate and confident diagnosis of vestibular migraine.

Keywords: *Vestibular migraine, vertigo, nystagmus, bedside evaluation, instrumental vestibular testing.*

Introduction

Vestibular migraine (VM) is a clinical entity for which diagnosis is grounded almost entirely in a detailed and methodical patient history, as recommended by international consensus documents (Lempert et al., 2012). A previous or current diagnosis of migraine is indispensable, yet a synchronous onset of headache and vestibular symptoms is *not* required for diagnostic purposes (Beh et al., 2019; Celebisoy et al., 2022). In practice, the disorder shows an extraordinary clinical variability, which complicates the application of strict diagnostic boundaries. Both the type and duration of vertigo or dizziness episodes may vary widely, as may the presence of imbalance or the degree and timing of migrainous accompaniments (Beh et al., 2019) (Celebisoy et al., 2022) (Teggi et al., 2018) (Huang et al., 2020). According to the present criteria, only half of all vestibular episodes must feature migraine-related phe-

nomena; however, these manifestations are frequently overlooked by patients, particularly when vertigo is of high intensity and overshadows other symptoms (Beh, 2020; Özçelik et al., 2022).

Several clinical situations reveal the limitations of the current diagnostic framework. The criteria cannot be easily applied when patients are unable to specify the duration of attacks, or when headache episodes have ceased for many years—a scenario consistent with *epigone VM*, considered a late-life equivalent of migraine (Pagnini et al., 2014). Furthermore, individuals with VM often report less severe headache than patients with migraine without vestibular involvement (Özçelik et al., 2022), which further contributes to underreporting. As no gold standard based on imaging or laboratory testing is available, clinicians must rely heavily on reconstructing symptom chronology and identifying potential triggers, despite the fact that many patients are unable to provide a precise description of their symptoms. For this reason, the diagnostic criteria (Lempert et al., 2012) risks being overly reductive for a condition whose recognition depends on integrating multiple anamnestic factors, such as hormonal fluctuation, stress, sleep deprivation, hydration status, diet, motion sickness, migraine premonitory symptoms, and familial predisposition.

From an instrumental standpoint, no specific audiovestibular pattern is uniquely associated with VM. Neuro-otological findings published in the literature present substantial heterogeneity, likely influenced by the interval from the most recent migraine or vertigo attack as well as by the dynamic pathophysiology of the disorder. Examination results may be normal or show minor alterations, and although these findings rarely confirm VM directly, they remain crucial for excluding conditions such as Menière's disease (MD), Benign Paroxysmal Positional Vertigo (BPPV), or other peripheral and central vestibular disorders (Beh, 2020).

The clinical spectrum of VM is exceptionally broad. Patients may present with spontaneous vertigo, positional vertigo of either short or prolonged duration, visually induced dizziness, head motion–triggered symptoms, or even forms of persistent dizziness (Teggi et al., 2018; Lempert et al., 2012; Young AS et al., 2021). Because several vestibular disorders share overlapping features, comprehensive testing is often required for differential diagnosis, particularly to distinguish VM from MD. Even though most VM patients show normal bedside and laboratory vestibular examinations, numerous studies highlight the presence of subtle vestibular abnormalities that may support the diagnosis in difficult or ambiguous cases. On the other hand, we must underscore that absence of abnormalities on standard vestibular clinical and instrumental tests does not exclude the diagnosis of VM.

Additionally, VM may coexist with other otovestibular conditions, or occur without typical migraine-associated findings such as motion sickness or a positive family history, further complicating the diagnostic process. The availability of a robust instrumental biomarker would therefore be invaluable, especially when the clinical criteria are not fully met. A careful analysis of the literature is needed to determine whether the variability seen in vestibular test results can be meaningfully attributed to VM.

Evaluating patients during an ictal episode—an acute attack—is uncommon but highly informative. During such episodes, spontaneous nystagmus has been documented in approximately 22.58% to 70% of cases, appearing with peripheral, central, or mixed characteristics (von Brevern et al., 2005; Polensek & Tusa, 2010; Beh et al., 2019; Young AS et al., 2021; Batinovic et al., 2025). Vertical spontaneous nystagmus, when present, has shown very high specificity for VM (93.0%), whereas horizontal nystagmus appears more specific for MD (82.1%), even though nystagmus direction in both disorders may fluctuate considerably (Beh, 2020). Persistent positional nystagmus has been observed in almost all symptomatic patients (100%) (Po-

lensek & Tusa, 2010; El Badry et al., 2017). Recent work has confirmed these trends, reporting ictal spontaneous nystagmus in 71.3% of VM patients (49.5% horizontal, 21.8% vertical) and positional nystagmus in 97.3%, with significantly increased nystagmus velocity during symptomatic phases (Young AS et al., 2021). However, such elevated prevalence rates have not been consistently reproduced across all studies (Celebisoy et al., 2022). Table 1 summarizes the main clinical manifestations associated with Vestibular Migraine. In the following sections of the manuscript a detailed description of the results of the vestibular clinical and instrumental results in patients with VM is reported.

Clinical Domain	Manifestations
Spontaneous Vertigo	Brief or prolonged spells, rotational or non-rotational
Positional Vertigo	Triggered by head position changes; short or long-lasting
Visual Motion Sensitivity	Vertigo/dizziness induced by moving visual scenes
Head-Motion Intolerance	Vertigo, dizziness or nausea provoked by quick head turns
Persistent Dizziness	Interictal instability, “swaying” or “rocking” sensation
Migraine Features	Photophobia, phonophobia, headache (not always present)
Common Triggers	Stress, poor sleep, dehydration, hormonal changes, foods

Table 1. Overview of the main clinical manifestations observed in Vestibular Migraine.

Bedside oculomotor test

A wide range of oculomotor abnormalities may be identified in patients with vestibular migraine (VM) even during the interictal period. Alterations may involve horizontal or vertical saccades, positional nystagmus (PN), head-shaking nystagmus (HSN), gaze-evoked nystagmus (GEN), or spontaneous nystagmus (SN). Notably, the prevalence of these findings appears to increase over the course of long-term follow-up, suggesting a potential evolution of vestibular dysfunction in certain patients (Radtke et al., 2012; Neugebauer et al., 2013). Interictally, *spontaneous nystagmus*—with or without visual fixation—has been reported in 2–20% of VM patients, whereas GEN or downbeat nystagmus is much less frequent, ranging from 0 to 0.25% depending on the series (Dieterich & Brandt, 1999; Celebisoy et al., 2008; Casani et al., 2009; Jeong et al., 2010; Radtke et al., 2012; Boldingh et al., 2013; Beh et al., 2021; Young AS et al., 2021; Waissbluth et al., 2023; Zhang et al., 2024). Conversely, interictal *positional nystagmus* represents one of the most consistently observed abnormalities, with reported rates between 28% and 57.9%. The most common pattern is a downbeat nystagmus elicited during the Dix–Hallpike or head-hanging maneuvers, suggestive of subtle cerebellar involvement (Vitkovic et al., 2008; Radtke et al., 2012; Beh et al., 2019; Young AS et al., 2021); importantly, such findings often occur in the absence of subjective vertigo (Dieterich & Brandt, 1999). Interpretation, however, must be cautious: a non-negligible proportion of healthy individuals can exhibit spontaneous nystagmus without fixation (Geisler et al., 2000) or positional nystagmus during testing (Martens et al., 2016), underscoring the need for careful clinical correlation.

The *head-shaking test* (HST) is traditionally regarded as a sensitive indicator of

peripheral vestibular asymmetry. Nevertheless, VM may display atypical HSN patterns, including biphasic responses or *perverted* HSN—vertical nystagmus elicited by horizontal head shaking—typically considered a sign of central vestibular involvement. Reported rates of HSN in VM vary between 15% and 50%, predominantly horizontal, though central patterns have also been described (Jeong et al., 2010; Radtke et al., 2012; Boldingh et al., 2013; Shin et al., 2013; Beh et al., 2019;). While up to 78% of MD patients exhibit a positive HST—usually a paretic response beating toward the affected ear—approximately 50% of VM patients may also show positivity, often in the form of downbeat HSN, which again raises the possibility of central compensatory mechanisms (Kim CH et al., 2014). A particularly unusual response, HST-induced opsoclonus, has been documented in two VM cases, occurring in the absence of any structural cerebellar or brainstem deficits (Kim MK et al., 2024).

Hyperventilation-induced nystagmus (HVNy) has emerged as another frequent interictal finding. Beh et al. (2019) reported it in 22.5% of VM patients, slightly exceeding earlier estimates of 19.5% (Califano et al., 2011). Hyperventilation results in an acute rise in blood pH accompanied by reduced serum calcium, unmasking otherwise silent cerebellar or vestibular dysfunction (Califano et al., 2013). The predominance of downbeat HVNy in VM mirrors patterns typically associated with cerebellar pathology, indirectly supporting the hypothesis of cerebellar involvement in a subset of VM patients (Walker & Zee, 1999).

More recently, the *skull vibration-induced nystagmus test* (SVINT) has been proposed as a rapid and sensitive tool to detect vestibular asymmetry (Dumas et al., 2017). In VM, SVINT positivity is relatively uncommon, occurring in only 6% of patients, in stark contrast with the 59.2% positivity reported in Menière's disease (MD) (Teggi et al., 2021). Because SVIN is strongly associated with partial or complete unilateral vestibular loss, its low prevalence in VM suggests distinct underlying mechanisms—peripheral in MD and more likely central in VM, potentially relating to brainstem or cerebellar processing abnormalities.

Eye Movement

During symptom-free intervals, the neurological examination of patients with vestibular migraine (VM) is typically unremarkable. Nevertheless, several studies have documented the presence of subtle and non-specific vestibulo-ocular abnormalities, which may emerge intermittently in association with the disorder. Among these, smooth pursuit deficits—most often taking the form of saccadic intrusions—have shown extremely heterogeneous prevalence rates, ranging from 0% to 90.3% across studies (Celebisoy et al., 2022; Radtke et al., 2012; Waissbluth et al., 2023; Casani et al., 2009; Boldingh et al., 2013; El Sherif et al., 2020; Fu et al., 2021; Li Y et al., 2023; Batinovic et al., 2025). Saccadic smooth pursuit was observed in 52% of patients both in VM and MD patients: this high number likely reflects non-specific central susceptibility, age-related factors, or examiner variability, and its diagnostic value appears limited when considered in isolation (Huppert et al., 2025). Abnormalities in saccadic performance, including prolonged latencies and hypometric responses, have likewise been reported with highly variable frequencies, generally between 0% and 32% (Radtke et al., 2012; Boldingh et al., 2013; ElSherif et al., 2020; Fu et al., 2021; Celebisoy et al., 2022). A more recent contribution, however, described saccadic alterations in as many as *70%* of VM patients (Waissbluth et al., 2023), further illustrating the inconsistency of the available evidence. Indeed, findings in the literature are often contradictory and, in several reports, show little distinction between VM patients and those with migraine without vestibular symptoms (Casani et al., 2009; Boldingh et al., 2013; Beh, 2020).

An additional layer of complexity derives from the temporal variability of these ocular motor findings. The presence and magnitude of abnormalities may fluctuate according to the number of days elapsed since the most recent vertigo attack (Li Y et al., 2023), suggesting that these signs may be dynamically modulated by the disease state. Longitudinal observations further indicate that such abnormalities may increase in prevalence over extended follow-up, rising from 16% to 41% over periods ranging from 5 to 11 years (Radtke et al., 2012). This progression may reflect either subtle cumulative alterations in central vestibular processing or improved detectability over time.

A concise overview of the most common bedside oculomotor and eye movements abnormalities observed during interictal evaluation is provided in Table 2.

Oculomotor Sign	Prevalence in VM	Typical Pattern	Clinical Interpretation
Spontaneous Nystagmus	2–20%	Horizontal or vertical	Variable central/peripheral involvement
Gaze-Evoked Nystagmus	0–0.25%	Horizontal	Low diagnostic value
Positional Nystagmus	28–57.9%	Mostly downbeat	Suggestive of cerebellar involvement
Head-Shaking Nystagmus	15–50%	Horizontal or downbeat	Possible central involvement
Saccadic Abnormalities	Up to 70%	Long latency, hypometria	Frequent but not specific

Table 2. Most frequent bedside oculomotor abnormalities described in the interictal phase of Vestibular Migraine.

Pupil Test

Even under rigorously constant environmental conditions—unchanged luminance, stable fixation, and neutral emotional state—the pupil is never static. Instead, it exhibits continuous micro-oscillations commonly known as pupillary hippus, characterized by rhythmic fluctuations between 0.04 and 2 Hz, generally with amplitudes below 0.5 mm (McLaren et al., 1992). These oscillations are interpreted as the expression of autonomic nervous system (ANS) modulation, given that pharmacological suppression of parasympathetic tone reduces or abolishes the phenomenon (Turnbull et al., 2017).

Recent work has highlighted distinct pupillary cycle characteristics in migraineurs. In particular, the pupillary cycle period is significantly prolonged in patients with migraine—even in the interictal phase—suggesting persistent alterations in central autonomic processing and sensory integration (Cortez et al., 2019), and potentially reflecting the known cortical hyperexcitability associated with migraine. The phenomenon known as *pupillary unrest* or *hippus* consists of low-frequency, spontaneous oscillations of the pupil under constant lighting. In vestibular migraine (VM), however, clinicians have described a distinct and more irregular pattern termed pupillary nystagmus.

Unlike physiologic hippus, pupillary nystagmus features:

- irregular, sometimes higher-frequency oscillations,
- abrupt changes reminiscent of nystagmus fast/slow phases,
- behavior suggestive of involvement of intrinsic ocular muscles,
- a dynamic pattern far less smooth and predictable than typical autonomic hippus.

This finding appears frequent among VM patients in the interictal phase, while

remaining rare in individuals with non-migraine dizziness or other vestibular conditions (Gufoni & Casani, 2023). Such selectivity has led to the hypothesis that pupillary nystagmus may reflect instability within the network linking vestibular nuclei, brainstem autonomic centers, and midbrain pupillary control circuits.

Several observations support this interpretation:

1. Brainstem autonomic–vestibular coupling
   VM may produce transient dysregulation in structures such as the Edinger–Westphal nucleus, generating oscillatory instability in pupillary control.
2. Sensory integration conflict
   Discrepancies between vestibular/otolithic signals and visual input could provoke oscillatory "pupil instability," analogous to visually induced vertigo.
3. Laterality phenomena
   In some patients with strictly unilateral headache, pupillary nystagmus has been observed only on the symptomatic side, suggesting hemispheric or brainstem asymmetry in autonomic regulation.

Clinical relevance and differential diagnosis

Clinically, pupillary nystagmus in VM shows several advantageous features:

- high prevalence in VM,
- rare occurrence in dizziness not related to migraine,
- absence or extreme rarity in Menière's disease and most peripheral vestibular disorders,
- simplicity of bedside detection, especially with Frenzel goggles or a lit lens.

For patients who do not fully meet VM diagnostic criteria, detecting pupillary nystagmus during interictal evaluation may serve as an important supportive sign.

The role of pupillometry as an emerging tool

The use of modern infrared pupillometry adds a new dimension to clinical assessment by enabling highly precise, quantitative analysis of pupillary behavior. Based on your clinical experience, a pupillometer allows:

- high-frequency sampling, capturing subtle oscillations invisible to the naked eye;
- accurate measurement of oscillation amplitude, frequency, and asymmetry;
- detection of complex, irregular patterns characteristic of pupillary nystagmus;
- evaluation of dynamic changes during specific visual tasks (e.g., fixation vs. motion stimuli);
- identification of eye-to-eye asymmetries, potentially related to unilateral migraine phenomena.

Pupillometry may also reveal:

- increased oscillation amplitude during visual motion tasks,
- greater instability during sensory conflict (e.g., with optokinetic patterns),
- altered pupil dynamics correlating with visual dependence and visually induced dizziness.

Its quantitative nature may make it an excellent adjunct to standard vestibular testing, offering objective support for VM diagnosis in uncertain cases and providing a tool to monitor changes over time or in response to therapy.

An intriguing observation is that, in some individuals with strictly unilateral headache, pupillary nystagmus appeared exclusively in the eye ipsilateral to the painful side, suggesting a lateralized dysfunction of autonomic or central regulatory mechanisms. Although these findings require validation in larger clinical cohorts, the detection of pupillary nystagmus may represent a simple, inexpensive, and highly informative bedside sign for supporting the diagnosis of VM. The oscillations can be easily documented using standard video devices or even a smartphone, making this sign accessible to clinicians in various settings (Gufoni e Casani, 2023). Table 3 compares physiological pupillary hippus with the pupillary nystagmus pattern typically observed in VM while Table 4 summarizes the principal pupillometric parameters that may show alterations in VM.

Feature	Physiological Hippus	Pupillary Nystagmus (VM)
Oscillation Pattern	Smooth, rhythmic	Irregular, nystagmoid (“fast/slow phases”)
Frequency	0.04–2 Hz	Higher or variable
Amplitude	<0.5 mm	Often greater / asymmetric
Origin	Autonomic oscillation	Central vestibular–autonomic dysregulation
Laterality	Usually, symmetric	Often asymmetric, sometimes unilateral
Clinical Value	Low	High specificity for VM

Table 3. Comparative features distinguishing physiological hippus from pupillary nystagmus typically observed in Vestibular Migraine.

Pupillometric Parameter	Description	Expected Pattern in VM
Oscillation Frequency	Cycles per second	Increased or irregular
Oscillation Amplitude	Peak-to-peak variation	Increased compared to controls
Cycle Regularity	Stability of waveform	Reduced (chaotic/unstable pattern)
Eye Asymmetry Index	Difference between eyes	Possible unilateral abnormality
Response to Visual Motion	Pupil behavior during OKN	Exaggerated instability
Sensory Conflict Response	Pupillary change during conflicting cues	Abnormal amplification

Table 4. Quantitative pupillometric parameters potentially altered in patients with Vestibular Migraine.

In our clinical practice, we employed a modern infrared digital pupillometer (Figure 1), capable of capturing high-frequency, sub-millimetric fluctuations of the pupil. This device allowed a precise and reproducible quantification of spontaneous pupillary oscillations under controlled illumination, providing objective metrics on diameter variability, waveform morphology, and inter-ocular asymmetry. Among the parameters generated by this technology, a particularly informative one is the PUAL (Pupillary Unrest in Ambient Light) index, which quantifies the amplitude and instability of spontaneous pupillary micro-oscillations recorded under constant lighting conditions. PUAL reflects the degree of intrinsic pupillary unrest and is considered a sensitive marker of altered autonomic–central regulation. This form of digital pupillometry represents a novel technological approach in the assessment of vestibular and migraine-related disorders. In contrast with traditional bedside examination—which relies solely on qualitative visual inspection—the pupillometer provides an operator-independent, quantitative analysis capable of detecting subtle oscillatory patterns such as pupillary nystagmus, which are typically invisible to the naked eye. The ability to objectively document these oscillations even during the interictal phase offers a unique window into the disturbed vestibular–autonomic integration that characterizes Vestibular Migraine. As such, infrared pupillometry, and specifically metrics such as PUAL, may emerge as promising biomarkers for improving diagnostic accuracy, supporting differential diagnosis, and monitoring treatment response in VM. Representative pupillometric recordings are shown in Figures 2 and 3, comparing spontaneous pupillary oscillations in a Vestibular Migraine patient versus a healthy subject under identical ambient light conditions.



Figure 1. Infrared digital pupillometer used for high-frequency recording of spontaneous pupillary oscillations.

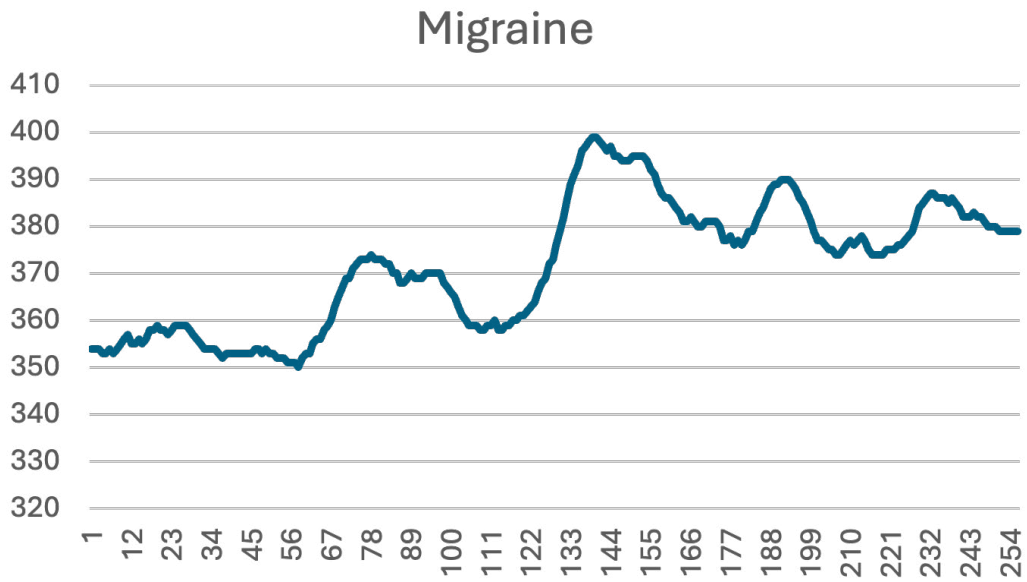


Figure 2. Pupillometric recording in a patient with Vestibular Migraine.

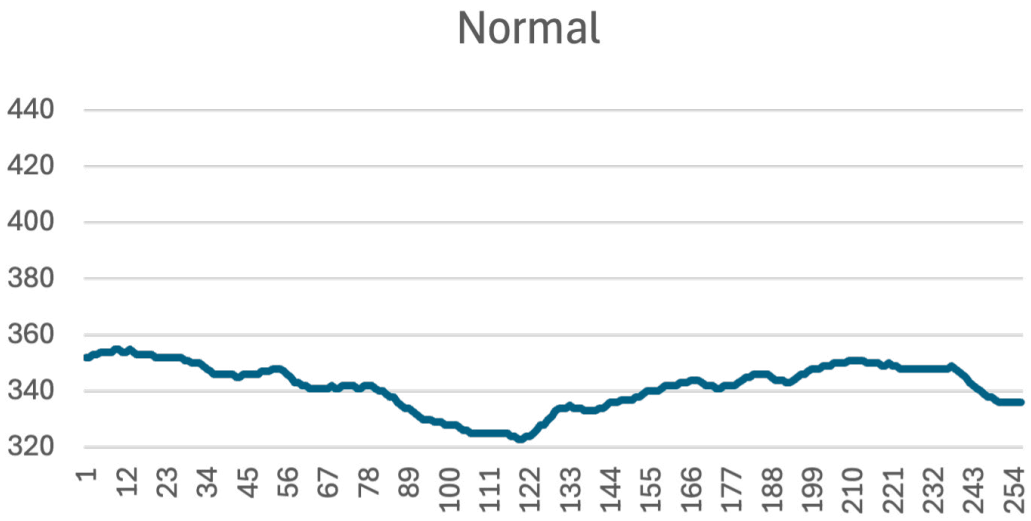


Figure 3. Pupillometric recording in a healthy control.

The Caloric Test

Among laboratory vestibular assessments, the caloric test has historically provided some of the most reproducible findings in patients with vestibular migraine. A unilateral reduction of caloric responsiveness, typically mild in magnitude (with asymmetries rarely exceeding 50%), has been reported in 10–30% of VM patients across several studies (Cutrer & Baloh, 1992; Casani et al., 2009; Teggi et al., 2009; Jeong et al., 2010; Radtke et al., 2012; Kang et al., 2016; Yilmaz et al., 2021; Waissbluth et al., 2023). Conversely, earlier investigations described bilateral caloric response in up to 15% of VM cases (Kayan & Hood, 1984; Cass et al., 1997; Bayazit et al., 2001; Casani et al., 2009). These patterns could indicate a higher-than-expected frequency of peripheral vestibular involvement in VM. However, alternative explanations must be considered, including the coexistence of other vestibular conditions presenting with migraine symptoms, or migraine episodes secondary to a pre-existing uncompensated vestibular deficit (Huang et al., 2020).

Importantly, caloric irrigation itself is recognized as a potential trigger for migraine, reinforcing the possibility that disorders such as benign paroxysmal positional vertigo or Menière's disease may precipitate migraine attacks in susceptible individuals (Murdin et al., 2009). Consequently, persistent caloric asymmetry is likely the exception rather than the rule in VM and should be interpreted cautiously (Calic et al., 2020).

Another consistent finding across studies is a high rate of caloric hyperresponsiveness, observed in up to 34% of VM patients, often accompanied by pronounced nausea or vomiting during stimulation. This pattern has been reported both in VM and in migraine without vestibular symptoms (Vitkovic et al., 2008; Casani et al., 2009; Teggi et al., 2009; Gedik-

Soyuyuce et al., 2022; Hannigan et al., 2024). Hyperresponsiveness is typically considered indicative of central vestibular dysfunction, particularly when accompanied by abnormalities in the visual suppression test. In such cases, the exaggerated caloric response may reflect increased neurotransmitter activity or heightened excitability of vestibular pathways (Cutrer & Baloh, 1992). This phenomenon aligns with the well-documented motion hypersensitivity prevalent among migraine patients (Teggi et al., 2018; Lempert et al., 2019). Indeed, caloric hyperresponsiveness may be considered the most characteristic caloric finding in VM, surpassing the diagnostic value of caloric hypofunction.

More recently, it has been proposed that the relationship between subjective and objective responses to caloric stimulation may offer additional diagnostic insight. Patients with migraine tend to report stronger subjective vertigo during caloric activation than individuals without migraine; notably, patients with vestibular migraine report the highest perceptual sensitivity among all comparison groups, including those with other vestibular disorders accompanied by migraine (Hannigan et al., 2024). This discrepancy between measured nystagmus intensity and patient-reported vertigo may represent a useful clinical discriminator between VM and other vestibular pathologies. The main caloric features associated with Vestibular Migraine are summarized in Table 5.

Finding	Prevalence	Interpretation
Unilateral Weakness	10–30%	Mild peripheral involvement or migraine overlay
Bilateral Weakness	≤15%	Rare; unclear significance
Hyperresponsiveness	Up to 34%	Central hyperexcitability; motion sensitivity
Strong Nausea Response	Frequent	Migraine-linked vestibular hypersensitivity

Table 5. Summary of characteristic caloric test findings in Vestibular Migraine.

The Video Head Impulse Test

Assessment of the high-frequency vestibulo-ocular reflex (VOR) using the video Head Impulse Test (vHIT) generally reveals fewer abnormalities in vestibular migraine (VM) compared with caloric irrigation, confirming a comparatively lower sensitivity of vHIT in this population (Blödow et al., 2014; Özçelik et al., 2022). Nevertheless, the literature reports considerable variability in vHIT findings among VM patients. In most studies, the predominant abnormality is a reduction in VOR gain, observed in approximately 9–11% of cases, which has led several authors to conclude that vHIT underestimates vestibular dysfunction in VM relative to caloric testing (Blödow et al., 2014; Kang et al., 2016; Yilmaz et al., 2021). Conversely, other investigations have documented higher rates of vHIT abnormalities, including reduced gain in at least one semicircular canal and/or the presence of corrective saccades across any canal (ElSherif et al., 2018; Fu et al., 2021; Du et al., 2022; Waissbluth et al., 2023). While VOR gain reductions remain infrequent, covert or overt saccades appear to be relatively common in VM and may therefore constitute a more sensitive indicator of subtle VOR instability (Calic et al., 2020; Huang et al., 2020; Du et al., 2022). On the other hand, Bernetti et al. (2018) reported an increase of vHIT gain hypothetically related to a cerebello-vestibular dysfunction causing hyperactivity of the vestibular system. Fu et al. (2021) reported that 15% of VM patients exhibited abnormal lateral canal vHIT responses, whereas 27% showed abnormalities involving the vertical canals. In contrast, some authors have described completely normal vHIT findings during the interictal phase (Özçelik et al., 2023), although a separate study noted that VM patients may display elevated vertical canal VOR gains compared with healthy controls, suggesting a distinct pattern of vestibular behavior in this population (Salmito et al., 2021). Because VM and Menière's disease (MD) may share overlapping cochleo-vestibular symptoms, several comparative studies have sought to differentiate the two disorders using instrumental vestibular testing. Caloric–vHIT dissociation is now considered an hallmark of MD (Cerchiai et al., 2017; Hannigan et al., 2019): this result is significantly more common in MD than in VM, consistent with previous findings suggesting that a mismatch between low- and high-frequency canal function is characteristic of MD (Mavrodied et al 2024) highlighting the added diagnostic value of combining caloric testing with vHIT.

These analyses consistently demonstrate that caloric weakness and pathological (reduced) vHIT responses are significantly more prevalent in MD than in VM (Shin et al., 2013; Blödow et al., 2014; Yilmaz et al., 2021), supporting the use of combined caloric and vHIT data in complex diagnostic cases. In this context Vosbeeck et al. (2025) found that the optimal sensitivity and specificity to differentiate between MD and VM was a caloric unilateral weakness threshold of 34%.

Table 6 highlights the instrumental differences between Vestibular Migraine and Menière's Disease.

Feature	Vestibular Migraine	Menière's Disease
Unilateral Caloric Weakness	Less common	More frequent
Hyperactive Caloric response	More common	Rare
vHIT Gain Reduction	Uncommon	Frequent
Corrective Saccades	Common	Very common
Vertical Canal Findings	Sometimes enhanced gain	Usually normal/low
Evolution Over Time	Fluctuating	Progressive deterioration

Table 6. Instrumental differences between Vestibular Migraine and Menière's Disease based on caloric testing and vHIT.

Cervical and Ocular VEMPs

A subset of patients with vestibular migraine (VM) report brief, isolated paroxysms of vestibular sensations even during interictal periods. These transient episodes—often lasting only a few seconds—may be described as sudden dropping, falling, or ground-shifting sensations. Such phenomena, along with tilt illusions, have been attributed to a possible dysfunction of the otolithic organs, prompting the use of cervical and ocular vestibular evoked myogenic potentials (c-Vemps and o-Vemps) to evaluate saccular and utricular integrity, respectively. However, findings derived from Vemps testing in VM have been highly inconsistent across studies. Some authors have reported absent (Boldingh et al., 2011) or reduced (Özdemiret al., 2020; Zuniga et al., 2012) cVemps amplitudes in one or both ears, occurring more frequently in VM patients compared with healthy controls (Boldingh et al., 2011; Zuniga et al., 2012; Zaleski et al., 2015; Özdemiret al., 2020;). Yet, a recent systematic review concluded that c-Vemps alone are insufficient to support diagnosis or guide management in VM (Fife et al., 2017), reflecting their limited diagnostic reliability. Indeed, several investigations have found no significant differences in c-Vemps parameters between VM patients, migraineurs without vestibular symptoms (Roceanu et al., 2008), and healthy individuals (Taylor et al., 2012; Salviz et al., 2016; Young AS et al., 2021).

The literature on o-Vemps mirrors this variability. While some studies have described reduced o-Vemps amplitudes in VM (Zaleski et al., 2015; Salviz et al., 2016; Oh et al., 2021), many more have found no clear deviation from control values (Taylor et al., 2012; Makowiec et al., 2018). Nevertheless, one pattern has emerged with potential diagnostic value: VM patients appear more likely than individuals with other vestibular disorders to show normal c-Vemps accompanied by abnormal o-Vemps, a dissociation proposed as a possible VM biomarker (Oh et al., 2021).

An elevated frequency of Vemps abnormalities has also been associated with worse clinical prognosis, particularly in patients experiencing recurrent attacks for more than six months (Young YH, 2013). This suggests that, although individually inconsistent, Vemps results may carry some prognostic significance.

Several studies have attempted to exploit c-Vemps and o-Vemps to differentiate VM from Menière's disease (MD). Reports indicate:

- Higher c-Vemps asymmetry ratios (AR) in unilateral MD compared with VM (Taylor et al., 2012; Salviz et al., 2016).
- Reduced o-Vemps amplitudes in unilateral MD relative to VM (Zuniga et al., 2012).
- Greater o-Vemps asymmetry ratio in unilateral MD (77%) than in VM (57%), although bone conducted o-Vemps asymmetry ratios (27% vs. 34%) and air conducted c-Vemps asymmetry ratios (51% vs. 44%) did not differentiate the two groups (Inoue et al., 2016).

Longitudinal data further suggest that Vemps characteristics tend to fluctuate significantly in VM but show a progressive symmetry ratio increase in MD, consistent with the evolving hydropic injury affecting the inner ear neuroepithelium (Dlugaiczyk et al, 2020).

Despite these observations, current evidence indicates that no vestibular laboratory test—whether caloric stimulation, vHIT, or Vemps—can reliably distinguish VM from MD in isolation. Ultimately, the most decisive audiovestibular feature supporting the diagnosis of MD remains the presence of fluctuating, progressive, low-frequency unilateral sensorineural hearing loss, a pattern not typical of VM. Hearing loss in VM, when present, is usually mild, largely symmetrical, and demonstrates limited progression over time (Beh, 2020; Chen et al., 2023). Table 7 provides a comparative overview of cervical and ocular VEMPs in VM and Menière's Disease.

Parameter	Vestibular Migraine	Menière's Disease
c-Vemps Amplitude	Normal or mildly reduced	Often markedly reduced
c-Vemps Asymmetry Ratio	Normal or variable	Frequently elevated
o-Vemps Amplitude	Normal or reduced	Commonly reduced
o-Vemps Asymmetry Ratio	Mildly increased	Strongly increased
c-Vemps–o-Vemps Dissociation	Common	Rare
Longitudinal Pattern	Fluctuating	Progressive worsening

Table 7. Comparative cervical and ocular VEMPs features in Vestibular Migraine and Menière's Disease.

Vertical Subjective Visual (SVV)

The subjective visual vertical (SVV) has been proposed as a potentially valuable indicator of disturbed spatial orientation—an alteration frequently reported by patients with migraine and, particularly, vestibular migraine (VM). Such abnormalities may contribute to the persistent dizziness often described during interictal periods (Beh et al., 2019). However, current evidence remains inconsistent. Some studies have documented larger SVV deviations in VM, especially during head-tilt conditions, suggesting an overestimation of tilt magnitude and pointing toward abnormal multisensory integration for spatial orientation (Ashish et al., 2017; Winnick et al., 2018; Bogle et al., 2023). Other investigators, however, have argued that SVV lacks adequate diagnostic sensitivity for VM, emphasizing that its utility remains uncertain and requires further systematic evaluation (Li F et al., 2012). Recently, significant alterations in spatial orientation perception was documented during the ictal phase of VM , as evidenced by higher SVV deviation, more frequently than in the interictal phase (Batinovic et al., 2025) It has been proposed that incorporating head-roll tilt positions, both static and dynamic, rather than relying solely on the traditional upright-head paradigm, may reveal more robust and clinically meaningful differences in VM and migraine patients (Mallampalli et al., 2022).

Within this broader context, the *vestibular autorotation test* (VAT) (O'Leary & Davis, 1990) has emerged as another tool that may assist in characterizing vestibular function in VM. In VAT, patients actively rotate the head at high frequencies (2–6 Hz) while horizontal and vertical vestibulo-ocular reflex (VOR) responses are recorded. Unlike many conventional vestibular tests, VAT examines VOR behavior at frequencies closer to those encountered in everyday activities. Studies have shown that VM patients may exhibit abnormally elevated VOR gain in both horizontal and vertical planes, accompanied by a reduced phase shift compared with healthy controls (Thungavelu et al, 2017). Similar findings have been replicated by other authors, who also noted that the combination of low horizontal phase and high horizontal gain can help differentiate VM from Menière's disease (MD), potentially excluding the latter when this VOR pattern is present (Yao et al., 2022).

The Functional Head Impulse Test

A recently introduced assessment, the functional Head Impulse Test (fHIT), enables clinicians to obtain quantitative information regarding a patient's ability to maintain clear visual perception during rapid, passive head rotations. Unlike the conventional vHIT, which evaluates the mechanical integrity of the vestibulo-ocular reflex (VOR), the fHIT employs a dynamic, performance-based paradigm designed to measure the *functional outcome* of gaze stabilization, specifically the percentage of correctly identified visual targets during head impulses (Corallo et al., 2018). The test can also be administered in the presence of optokinetic background stimulation, generat-

ing a variant referred to as optokinetic-fHIT (o-fHIT). This configuration introduces a moving visual environment on the display, allowing investigation of the visual contribution to balance control and the degree of dynamic visual dependence, a common feature in vestibular migraine (VM) and likely related to impaired multisensory integration (Beh et al., 2019). Comparing fHIT performance with and without the moving background provides valuable insight into the relative influence of visual flow on perceptual stability. A substantial deterioration in performance—typically expressed as a reduction in the percentage of correct answers (%CA)—is expected when a mismatch arises between visual motion cues and vestibulo-proprioceptive information (Versino et al., 2020). In fact, approximately 58% of VM patients demonstrate a significant decline in %CA during o-fHIT compared with the baseline condition, a finding not commonly observed in healthy subjects (Casani et al., 2021). This reduction appears to correlate with clinical complaints such as visually induced vertigo, head-motion–triggered vertigo, and head-motion–evoked dizziness accompanied by nausea (Bednarczuk et al., 2019). These observations carry important clinical implications. Firstly, VM patients who exhibit a pronounced drop in %CA during optokinetic stimulation may benefit from rehabilitation strategies specifically targeting visual motion desensitization, a therapeutic approach aimed at reducing excessive visual dependence (Pavlou, 2010). Secondly, the fHIT—particularly its optokinetic variant—may serve as a useful instrumental marker in cases where VM is suspected but the international diagnostic criteria are only partially fulfilled. In such situations, an otherwise normal or inconclusive clinical/vestibular examination combined with an abnormal fHIT exclusively under optokinetic stimulation can provide supportive evidence toward a VM diagnosis. Recently these results were confirmed in a group of VM patients showing lower fHIT %CA than patients suffering from Persistent Postural-Perceptual Dizziness and subjects with migraine without vertigo symptomatology. In VM patients after the use of optokinetic background stimulation, a significant drop in %CA was noted in comparison with the other group of patients (Kirazli et al., 2025). Further research is needed to determine the differential diagnostic value of the fHIT in distinguishing VM from Menière's disease (MD), although preliminary findings suggest promising potential in this direction (Mallampalli et al., 2022). Table 8 illustrates the functional VOR performance in standard and optokinetic fHIT conditions in VM.

Test Condition	VM Findings	Clinical Implication
Standard fHIT	Normal or mildly reduced %CA	Functional VOR preserved
Optokinetic-fHIT	Strong reduction in %CA	High visual dependence
Difference (%CA drop)	Significant in VM	Suggests visual-vestibular mismatch
Symptoms Correlated	motion-induced nausea, head-motion vertigo	Supports VM diagnosis
Rehabilitation Response	Good response to visual-motion desensitization	Useful therapeutic target

Table 8. Functional VOR (Vestibulo-Ocular Reflex) performance in standard fHIT and optokinetic-fHIT conditions in patients with Vestibular Migraine (VM). %CA: percentage of correct answer.

Final Remarks

Vestibular migraine (VM) remains a diagnosis grounded primarily in a meticulous reconstruction of the clinical history. Patients may experience an exceptionally broad spectrum of vestibular manifestations, and this variability—combined with

the often-inconsistent temporal relationship between headache and vestibular symptoms and the wide range of headache intensity, sometimes perceived only as cranial pressure or fullness—frequently generates confusion for both patients and clinicians (Lempert & von Brevern, 2019; Beh, 2020). Because individuals with VM are not always able to articulate their symptoms accurately, obtaining a detailed and reliable history requires time and careful questioning, yet omissions of diagnostically relevant details remain common.

Instrumental vestibular testing in VM typically yields normal or inconsistent findings, serving mainly to exclude alternative diagnoses rather than to confirm VM. Nonetheless, the availability of a specific instrumental hallmark would be of great clinical value, particularly when patients exhibit symptoms suggestive of VM but fail to meet all established diagnostic criteria (Casani & Gufoni, 2025). During the interictal period, individuals with VM may show a variety of neuro-otological abnormalities—central, combined central-peripheral, or purely peripheral—but these findings are generally mild, fluctuating, and non-specific. Among the most frequently reported abnormalities are saccadic pursuit deficits (up to 50%) and positional nystagmus (up to 28%), whereas spontaneous nystagmus, gaze-evoked nystagmus, and pathological vHIT results are uncommon. Long-term studies have shown that these non-specific ocular motor signs may increase over time, rising from 16% to 41% over follow-up periods of 5.5 to 11 years (Radtke et al., 2012). Nevertheless, between attacks most patients exhibit no nystagmus or only very low-velocity positional nystagmus and otherwise normal vestibular test results (Young AS et al., 2021). Taken together, current evidence confirms that no pathognomonic sign and no gold-standard diagnostic test is available for the diagnosis of VM. The absence of a definitive biomarker has significant implications in clinical practice. A large subset of patients presents with spontaneous episodic dizziness which, after exclusion of other causes, does not satisfy the criteria for VM (Dominguez-Duran et al., 2021). Many of these cases fall under the historical category of benign recurrent vertigo (BRV), now better conceptualized as recurrent vestibular symptoms not-otherwise-specified (RVS-NOS). This condition is defined by spontaneous and/or positional vertigo—either spinning or non-spinning—without hearing loss, tinnitus, aural fullness, or non-vestibular migrainous features. In a long-term follow-up of more than 5 years, Dlugaiczyk et al. (2021) reported that patients with RVS-NOS show no female predominance (unlike VM), exhibit milder neurovegetative symptoms, and present a clinical profile more reminiscent of VM than of Ménière's disease (MD). Moreover, the shared temporal pattern and the presence of migraine precursors (such as childhood motion sickness) in both RVS-NOS and VM suggest a possible common pathophysiological substrate (Teggi et al., 2023).

These observations highlight the pressing need for multicenter studies involving large patient cohorts, with the aim of integrating current diagnostic tools with emerging tests—including the functional Head Impulse Test (fHIT) and the assessment of pupillary nystagmus—alongside potential biomarkers (Flook et al., 2019). Such an approach may ultimately support the development of a robust diagnostic algorithm capable of identifying VM with greater accuracy, complementing and reinforcing the information obtained through thorough clinical history-taking.

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