Cervical and Ocular VEMPs: from Otolith Physiology to Clinical Algorithms — A Narrative Review

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Abstract
Vestibular-evoked myogenic potentials (VEMPs)—cervical (cVEMP) and ocular (oVEMP)—are short-latency reflexes to air-conducted sound (ACS) or bone-conducted vibration (BCV) that probe otolith–brainstem–muscle pathways. To synthesize the physiological foundations (sustained vs transient otolith systems) and the clinical signatures of cVEMP/oVEMP across common vestibulopathies, and highlight high-frequency paradigms relevant to third-window disorders. Narrative synthesis of foundational physiology, translational studies, and clinical series for vestibular neuritis, Ménière's disease (MD), and superior semicircular canal dehiscence (SCD). Clinical ACS/BCV robustly recruits irregular otolith afferents (transient system), generating a contralateral oVEMP n10 and an ipsilateral cVEMP p13–n23. In superior vestibular neuritis (SVN) the oVEMP n10 is reduced/absent beneath the contralesional eye while cVEMP is typically preserved; the complementary dissociation characterizes inferior vestibular neuritis (IVN). In MD, attack-phase testing often reveals utricular "enhancement" (oVEMP↑) with concurrent saccular reduction (cVEMP↓) relative to quiescence. For SCD, a single 4,000-Hz oVEMP trial is a rapid, specific functional screen, with a clear canal–otolith mechanism. Conclusions. Interpreting VEMPs within the sustained/transient framework clarifies why 500-Hz stimuli are efficient for routine testing and why reflex projection—not stimulus type—confers utricular (oVEMP) vs saccular (cVEMP) weighting. A standardized oVEMP/cVEMP + vHIT battery improves lesion localization, follow-up, and triage for SCD (Curthoys et al., 2017; Curthoys et al., 2018; Manzari et al., 2013; Curthoys & Manzari, 2020).

Keywords: *oVEMP; cVEMP; utricle; saccule; bone-conducted vibration; vestibular neuritis; Ménière's disease; semicircular canal dehiscence; sustained/transient otolith systems.*

Abbreviations

AR = asymmetry ratio; **TR** = tuning ratio (A1kHz/A500Hz); **ΔTR** = interaural tuning ratio difference; **Fz** = midline forehead; **ACS** = air-conducted sound; **BCV** = bone-conducted vibration; **SCM** = sternocleidomastoid; **SVV** = subjective visual vertical; **OCR** = ocular counter-roll; **vHIT** = video head-impulse test; **CHL** = conductive hearing loss.

Introduction

VEMPs are brief, stimulus-locked myogenic potentials recorded over the sternocleidomastoid (cVEMP) or infra-orbital fields (oVEMP) after air-conducted sound (ACS) or bone-conducted vibration (BCV). They assay otolith receptor function together with the short polysynaptic vestibulo-spinal and vestibulo-ocular projections that drive

the muscles under the electrodes. At clinical stimulus levels, both maculae can be activated by ACS/BCV; it is the **reflex projection** that confers relative organ weighting—**oVEMP** (contralateral, excitatory n10) reflects the utriculo-ocular pathway, while **cVEMP** (ipsilateral, inhibitory p13–n23) reflects the sacculo-collic pathway (Kushiro et al., 1999; Kushiro et al., 2000; Uchino & Kushiro, 2011; Curthoys et al., 2018).

A unifying physiological framework distinguishes **sustained** vs **transient** otolith subsystems. Regular (extrastriolar/type II) afferents encode low-frequency/static stimuli (tilt, gravito-inertial loads), whereas **irregular striolar** afferents (type I, calyx) show low thresholds and precise phase-locking to brief **≈500-Hz** ACS/BCV bursts, dominating VEMP generation (Curthoys et al., 2017; Curthoys et al., 2019). In humans, the diagnostically salient components occur at short latencies—~6–7 ms for the oVEMP n10 and ~8–13 ms for the cVEMP p13—underscoring the importance of rapid stimulus onsets and fast synaptic transmission at type I–calyx endings.

Specificity of cVEMP for saccular function at clinical settings.

A comprehensive 2025 review integrating single-unit recordings across species, lesion/electrical-stimulation studies, and human clinical data concludes that, with standard clinical frequencies and intensities (e.g., 500-Hz tone bursts; clicks around 100–110 dB SPL), semicircular canal contributions are negligible and the cVEMP specifically indexes saccular/inferior nerve function (Curthoys et al., 2025). Apparent reports of canal activation by ACS arose from non-comparable stimulus regimes (e.g., rat clicks at ~130 dB SPL and species with markedly different audibility curves). When intensities are matched to human-relevant levels, otolith—but not canal—afferents dominate the early spike-rate response that generates VEMPs. Conversely, after a third-window (e.g., superior canal dehiscence), high-frequency sound/vibration can phase-lock canal afferents and augment both oVEMPs and cVEMPs—physiology that explains the diagnostic value of 4,000-Hz oVEMP in SCD screening.

Clinical implications. Because striolar type I receptors are metabolically vulnerable (e.g., to intratympanic gentamicin), VEMPs provide a rapid, objective window on **transient** otolith function, complementing video head-impulse testing (vHIT) and static measures (e.g., SVV/OCR). Throughout this narrative review we apply the sustained/transient framework to the interpretation of standard **500-Hz Fz-BCV/ACS** paradigms in vestibular neuritis and Ménière's disease, and to high-frequency paradigms in third-window disorders. Foundational selective-stimulation studies from Uchino's group delineated utricular and saccular circuits to extraocular and neck motoneurons—integrating ipsilateral inhibitory sacculo-collic and crossed utriculo-ocular/collic projections within a VO/VC/VOC/VS neuronal framework—providing the anatomical–physiological basis for the short-latency VOR/VCR components measured as VEMPs (Uchino & Kushiro, 2011).

Methods

Scope and guiding questions

This narrative review synthesizes evidence on the physiological bases and clinical applications of **cVEMP** and **oVEMP**, with emphasis on (i) the **sustained vs transient** otolith framework, (ii) standard **500-Hz** paradigms (Fz-BCV and ACS) for peripheral vestibulopathies, and (iii) **high-frequency (4,000 Hz)** paradigms for third-window disorders.

Sources and study selection

Sources comprised peer-reviewed works **provided by the author** and key related references spanning **1999–2025**, including: (a) mechanistic reviews on otolith physiology and VEMP generation (Curthoys et al., 2017; 2018; 2019; Curthoys et al., 2025); (b) classical **pathway studies in cats** (Kushiro et al., 1999; 2000; Uchino & Kushiro, 2011);

(c) translational single-unit evidence linking **irregular** otolith afferents to ACS/BCV; (d) clinical series on **SVN/IVN** and **Ménière's disease** using Fz-BCV/ACS at ~500 Hz (Manzari et al., 2010a; 2010b; 2010c; 2011); (e) third-window/SCD studies including **oVEMP at 4,000 Hz** (Manzari et al., 2013; Curthoys & Manzari, 2020); and (f) the **systematic review** on Ménière's disease (Perez-Carpena & López-Escámez, 2019).

Inclusion criteria (clinical): adult human studies reporting **cVEMP and/or oVEMP** elicited by **ACS** or **BCV** at **clinical intensities**, with at least one of: presence/absence, amplitude, latency (p13–n23; n10), **asymmetry ratio (AR)**, threshold, or frequency-tuning. We prioritized designs with clear diagnostic cohorts (SVN, IVN, MD, SCD), description of **phase** (attack vs quiescence for MD), and either contralateral ear or healthy **controls**.

Inclusion criteria (physiology): animal or human electrophysiology directly informative for clinical paradigms (e.g., irregular otolith afferent **phase-locking** to sound/vibration; cat sternocleidomastoid/ocular projections). Studies employing **non-clinical** intensities were considered for mechanism only and explicitly flagged when interpretation might not translate to human clinical settings.

Exclusion criteria: pediatric-only cohorts; central vestibular disorders; case reports without VEMP metrics; paradigms not comparable to ACS/BCV (e.g., galvanic stimulation) for the purposes of diagnostic performance; and studies where stimulus regimes were **far above human clinical levels** without translational relevance.

Data extraction and outcomes

From each eligible item we extracted: **stimulus parameters** (modality; frequency—**500 Hz**, **4,000 Hz**; burst shaping and **onset**; repetition rate; number of sweeps), **recording montage** (infra-orbital with upgaze for oVEMP; ipsilateral SCM with EMG monitoring/normalization for cVEMP), and **primary outcomes** (presence, amplitude, latency, **AR**, thresholds; frequency-tuning where applicable). We noted **quality/standardization** items (artifact rejection, replicate averages, electrode impedance, Fz coupling symmetry).

Synthesis approach

Given heterogeneity across paradigms and patient populations, we adopted a **qualitative, condition-oriented synthesis**, triangulating: (i) physiological plausibility from the **sustained/transient** framework, (ii) projection-based attribution (utricle→oVEMP; saccule→cVEMP), and (iii) clinical effect patterns in **SVN/IVN**, **MD** (attack vs quiescence), and **SCD** (4 kHz screen). Where available, we highlight quantitative cut-offs (e.g., oVEMP AR in SVN) and frequency-specific **rule-in** features for SCD.

Risk-of-bias and certainty considerations

Because this is a **narrative** review, no formal risk-of-bias instrument was applied. We qualitatively assessed: (a) potential **physiological confounders** (SCM tonic EMG for cVEMP; **upgaze** stability for oVEMP; stimulus symmetry at **Fz**), (b) **methodological heterogeneity** (burst design, repetition rate, averaging), and (c) **selection/reporting** issues (single-center cohorts, lack of blinding). For MD, conclusions were tempered by the **systematic review** showing modest pooled diagnostic performance of VEMPs and protocol heterogeneity (Perez-Carpena & López-Escámez, 2019). For the specificity of **cVEMP** as a saccular indicator, we relied on convergent mechanistic and clinical evidence at **human-relevant intensities** (Curthoys et al., 2025).

Results

Physiological Framework Sustained vs transient otolith systems

Irregular striolar afferents phase-lock to single cycles of sound/vibration into the kilohertz range; regular afferents are minimally responsive at clinical intensities. Central vestibular neurons mirror this sustained/transient dichotomy. These features underwrite short-latency VEMPs as **tran-**

sient-system readouts (Curthoys et al., 2017; Curthoys et al., 2019).

Commissural and cross-striolar inhibition (otolith-specific gain mechanisms)

Focal stimulation experiments in animal models reveal two "hidden" gain mechanisms in the otolith system: **commissural inhibition** (prominent in the utricular system), which augments sensitivity by integrating bilateral inputs, and **cross-striolar inhibition** (prominent in the saccular system), which sharpens directional tuning across the striola within a single macula. These motifs help explain robust VCR responses and the ipsilateral **inhibitory** cVEMP (p13–n23) from saccular activation, and they predict asymmetry patterns after unilateral lesions (Uchino & Kushiro, 2011). A compact schematic of these interactions and their clinical correlates is shown in **Figure 1**.

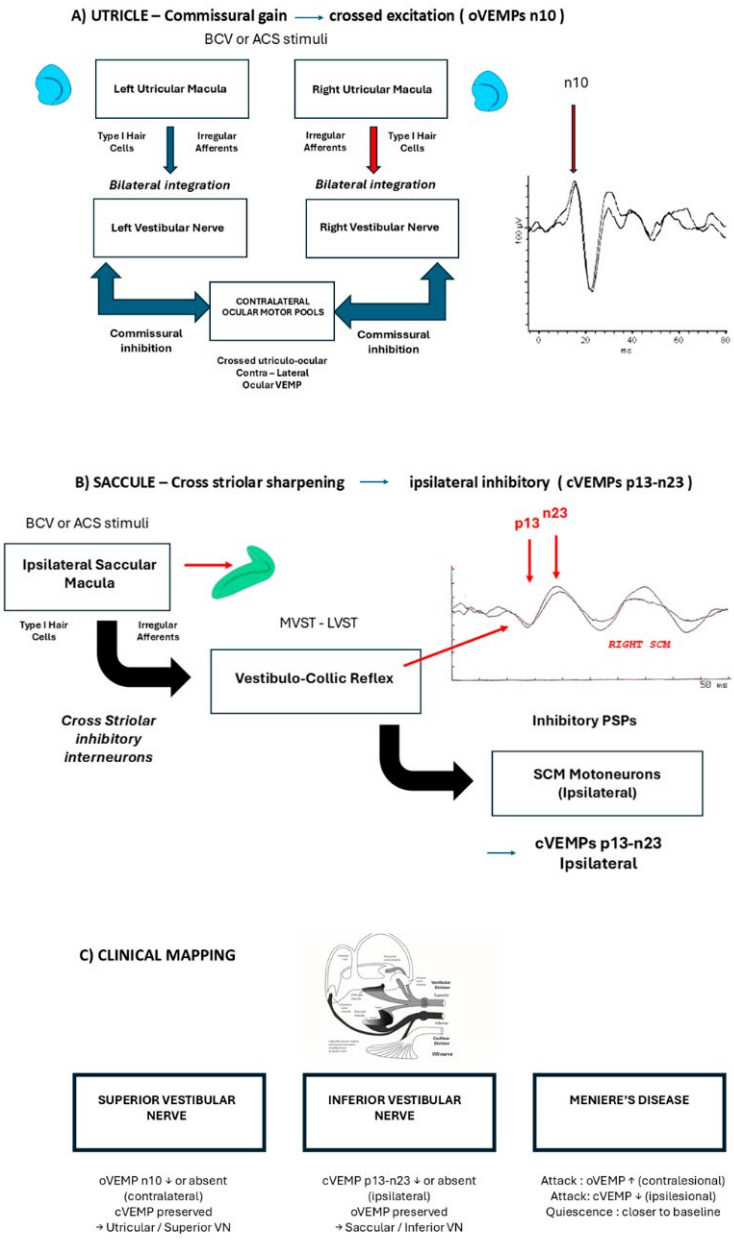


Figure 1. Commissural vs cross-striolar inhibition supporting oVEMP/cVEMP patterns. *(A) Utricle—commissural gain:* bilateral utricular inputs with strong **commissural inhibition** yield net **crossed excitation** of ocular-motor pools, explaining contralateral **oVEMP n10** and predicting **large oVEMP AR** after unilateral utricular loss with preserved canal function. *(B) Saccule—cross-striolar sharpening:* **cross-striolar inhibitory** interneurons within the saccular macula shape an **ipsilateral inhibitory** sacculo-collic output, explaining **cVEMP p13–n23** and predicting **reduced/absent cVEMP** in selective saccular/inferior nerve loss. *(C) Clinical mapping:* **SVN → oVEMP↓ with cVEMP preserved; IVN → cVEMP↓ with oVEMP preserved; MD → tuning shift (TR) and phase-dependent dissociation**.
VEMP = Vestibular Evoked Myogenic Potential, **oVEMP** = Ocular VEMP (contralateral **n10** negative peak at ~10 ms), **cVEMP** = Cervical VEMP (ipsilateral **p13–n23** biphasic peaks at ~13 & ~23 ms), **VN** = Vestibular Nucleus, **MVST / LVST** = Medial / Lateral Vestibulo-Spinal Tract, **SCM** = Sternocleidomastoid (muscle), **PSPs** = Postsynaptic Potentials, **SVN** = Superior Vestibular Neuritis, **IVN** = Inferior Vestibular Neuritis, **MD** = Ménière's Disease, **↓ / ↑** = Reduced / Enhanced (relative change).

Reflex projections and organ attribution

Organ attribution is derived from projection anatomy and physiology, not from stimulus type: oVEMP n10 reflects a crossed utriculo-ocular projection, and cVEMP p13–n23 reflects an ipsilateral sacculo-collic projection—consistent with cat pathway studies showing ipsilateral SCM inhibition from saccular/utricular inputs and crossed utricular excitation, and with convergent otolith inputs to vestibular neurons projecting to ocular and spinal targets (Kushiro et al., 1999; Kushiro et al., 2000; Curthoys et al., 2018). A schematic overview of these pathways and frequency-specific paradigms is provided in **Figure 2**.

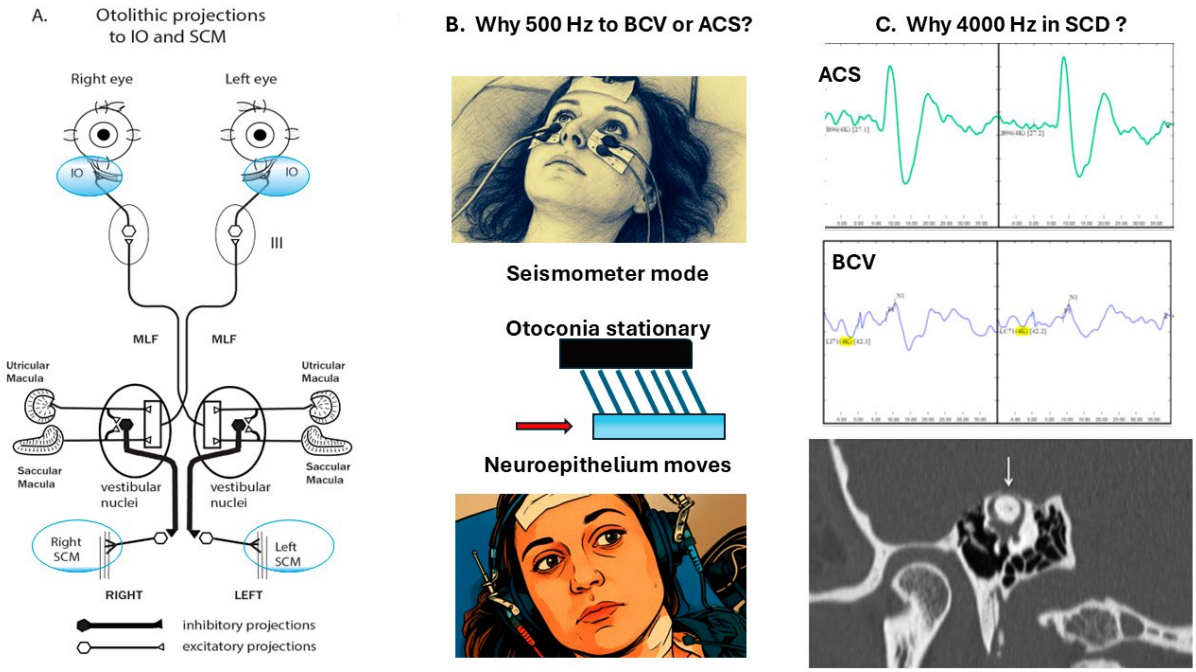


Figure 2. VEMP pathways and frequency-specific paradigms. *(A)* **Reflex anatomy:** crossed **utriculo-ocular** projection underlying contralateral **oVEMP n10** and ipsilateral **sacculo-collic** projection underlying **cVEMP p13–n23**; both driven predominantly by irregular otolith afferents (transient system). *(B)* **Why 500 Hz:** brief, rapid-onset **500-Hz** bursts act as a "**seismometer**" stimulus for the maculae; **Fz-BCV** delivers symmetric skull drive, while **ACS** requires middle-ear transmission; both efficiently generate robust VEMPs in healthy ears. *(C)* **Why 4000 Hz in SCD:** a **third-window** (e.g., superior canal dehiscence) lowers labyrinthine impedance, allowing high-frequency ACS/BCV to **phase-lock superior canal afferents** and amplify VEMPs; **4-kHz oVEMP** separates SCD from normals and is typically **abolished after canal plugging**.

Why 500 Hz at Fz and ACS?

Physiological sweet spot. Around 500 Hz, brief, rapid-onset bursts most efficiently recruit irregular striolar afferents (type I, calyx), which exhibit tight cycle-by-cycle phase-locking and low thresholds to both bone-conducted vibration (BCV) and air-conducted sound (ACS). This yields short-latency, high-SNR oVEMP n10 and cVEMP p13–n23 dominated by the otolithic transient system. The effect reflects receptor/afferent dynamics—not canal activation—in intact labyrinths (Curthoys et al., 2017; Curthoys et al., 2018; Curthoys et al., 2019).

Why Fz (midline forehead) for BCV. Fz coupling produces near-symmetric skull acceleration at both mastoids, minimizing

stimulus asymmetry and improving reliability of asymmetry ratio (AR) comparisons. It also reduces placement variability vs mastoid BCV and aligns the acceleration vector to effectively stimulate both maculae (Curthoys et al., 2009; Curthoys et al., 2018).

Why include ACS at ~500 Hz. Although middle-ear transmission is required, ACS 500 Hz complements Fz-BCV by: (i) confirming findings with a second modality; (ii) enabling testing where BCV hardware is unavailable; and (iii) providing threshold paradigms in some labs. Note that conductive components can attenuate ACS responses; in such cases, Fz-BCV is preferable.

Signal design matters. Use short bursts with very fast rise/fall (e.g., Blackman/raised-cosine shaping; total duration ≈ 2–7 ms). The onset carries most of the effective energy for driving n10/p13; excessive plateau adds time without improving synchrony. Typical repetition rates are 4–5 Hz with 50–100 sweeps per average (Curthoys et al., 2018).

Safety/comfort. 500-Hz ACS requires higher SPL than BCV acceleration to achieve comparable neural drive; adhere to local SPL and exposure limits. Fz-BCV avoids middle-ear loading and is generally well tolerated.

Key take-home. At **500 Hz**, both **Fz-BCV** and **ACS** efficiently interrogate the otolithic transient system; the measured projection determines the organ weighting (utricle→oVEMP; saccule→cVEMP), not the stimulus class (Curthoys et al., 2018; Curthoys et al., 2019).

Why 4,000 Hz at Fz or ACS?

Clinical motivation. In an intact labyrinth, very-high-frequency (≈4 kHz) stimuli rarely elicit measurable VEMPs, because otolith afferents—and downstream reflex pathways—behave effectively as low-pass systems beyond the high-hundreds to ≈1–2 kHz. By contrast, in third-window conditions (notably superior semicircular canal dehiscence, SCD) there is an abnormal fluid pathway that makes the inner ear highly responsive to high-frequency sound and vibration. As a result, oVEMP n10 at 4,000 Hz becomes large and reliable, while normal ears remain absent/near-zero, yielding an excellent signal-to-diagnosis ratio (Manzari et al., 2013; Curthoys & Manzari, 2020).

Biomechanical/physiological rationale. A dehiscence lowers labyrinthine impedance and allows high-frequency ACS/BCV to phase-lock superior canal afferents and to shunt acoustic energy that would otherwise be poorly transmitted. These canal signals project to ocular and cervical motoneuron pools, amplifying oVEMPs and cVEMPs at kilohertz frequencies. In healthy labyrinths, this canal-mediated high-frequency drive is negligible, explaining the binary separation between SCD ears and controls at 4 kHz.

Diagnostic performance. A single 4,000-Hz oVEMP trial (often ~50 bursts at 4 Hz; test time ~13 s) identified all CT-verified SCD ears and none of 22 healthy controls in the original cohort (sensitivity/specificity = 1.0). Subsequent mechanistic work accounts for this selectivity and supports using 4 kHz as a rapid functional screen to triage patients for imaging and surgical counseling (Manzari et al., 2013; Curthoys & Manzari, 2020).

Fz-BCV vs ACS at 4 kHz. Both modalities work:

- **ACS 4 kHz** is simple to deploy, but responses depend on middle-ear transmission; **conductive loss** can yield false-negatives.
- **Fz-BCV 4 kHz** bypasses the middle ear and delivers a symmetric skull drive, useful when conductive components or effusions are present. In either case, the contralateral oVEMP n10 is the primary readout; cVEMP at 4 kHz can be supportive but is more variable due to SCM EMG dependencies.

Recommended protocol (pragmatic). Upgaze 25–30°; infra-orbital montage; 4,000 Hz, short raised-cosine/Blackman

burst (rise/fall ≈ 2–3 ms, minimal plateau), repetition 4–5 Hz, 50–100 bursts, artifact rejection as per lab routine. Report presence/absence and amplitude; AR is optional because normals typically show no response at 4 kHz. (**see Table 2** for a practical checklist).

Pitfalls and differentials. A positive 4 kHz oVEMP indicates a third-window physiology, not exclusively SCD: enlarged vestibular aqueduct or other canal dehiscences may also test positive. Confirm with temporal-bone CT and clinical correlation. Post-operative canal plugging usually abolishes the 4 kHz response, providing an objective marker of surgical success (Curthoys & Manzari, 2020).

Stimuli and Transduction Salient to VEMPs

Both ACS and BCV activate utricular and saccular receptors at clinical levels; there is no strict "ACS→saccule / BCV→utricle" selectivity. The differential readout (oVEMP vs cVEMP) arises from projections. Mechanistically, otoliths act as seismometers at high frequency (sound/vibration) and accelerometers at low frequency (tilt/translation), explaining tight neural phase-locking and the efficiency of short, high-frequency bursts (Curthoys et al., 2018; Curthoys et al., 2019).

After SCD, high-frequency ACS/BCV additionally phase-locks superior canal afferents, augmenting VEMPs and shifting tuning toward kilohertz—absent in intact labyrinths (Manzari et al., 2013; Curthoys & Manzari, 2020).

Practical Recording Considerations

cVEMP: Ipsilateral SCM EMG monitoring/normalization; 500-Hz ACS/BCV bursts; report p13–n23 amplitude and AR (± thresholds where relevant).

oVEMP: Infra-orbital montage with up-gaze; ACS or Fz-BCV; report n10 amplitude and AR; **frequency-tuning** (including high-frequency trials) increases diagnostic yield in third-window suspects (Curthoys et al., 2018).

Clinical Signatures

Key patterns are summarized in Table 1.

Superior vestibular neuritis (SVN)

Prospective series with Fz-BCV 500 Hz show that, in unilateral SVN, the oVEMP n10 is reduced/absent beneath the contralesional eye while cVEMP remains typically preserved, consistent with utricular (superior nerve) involvement and spared saccular (inferior nerve) inputs. In a cohort of 133 SVN patients, an oVEMP n10 asymmetry-ratio (AR) cut-off of 46.5% yielded ~94% accuracy for side identification (AUC ≈ 0.94). Follow-up demonstrates normalization of oVEMP with recovery, mirroring restoration of dynamic utricular function and canal VOR gains (Manzari et al., 2010a; Manzari et al., 2011; Curthoys et al., 2019).

Inferior vestibular neuritis (IVN)

The complementary pattern—pathologic cVEMP (p13–n23 ↓/absent) with preserved oVEMP—supports selective saccular/inferior nerve dysfunction. Importantly, the same dissociation is seen with BCV or ACS, underscoring that projections, not stimulus type, determine organ weighting (Manzari et al., 2010b).

Ménière's disease (MD)

Within-patient comparisons during acute attack vs quiescence reveal a robust dynamic dissociation: oVEMP n10 increases contralesionally, while the cVEMP p13 decreases ipsilesionally during attacks, relative to intercritical periods. Healthy controls tested twice show stable symmetry. These findings suggest differential regional effects within the otolith organs across disease phases (Manzari et al., 2010c).

Beyond these physiological signatures, a recent systematic review emphasizes that VEMPs in MD have supportive value but an unestablished stand-alone diagnostic role due to heterogeneous protocols and phase-dependent variability; pooled performance across studies is modest (≈50% sensitivity; ≈49% specificity), underscoring

the need to integrate VEMPs with audiometry, calorics and vHIT in clinical decision-making (Perez-Carpena & López-Escámez, 2019).

Frequency-tuning as a supportive biomarker. Several studies show that cVEMP frequency tuning in hydropic ears may shift from a 500-Hz peak toward ~1,000 Hz, and that a simple tuning ratio (amplitude at 1 kHz divided by amplitude at 500 Hz) can separate many MD ears from healthy controls; oVEMP tuning abnormalities have also been reported. Physiologically, endolymphatic hydrops alters the mechanical loading and striola/extra-striolar contribution to sound and vibration, changing the effective receptor–afferent drive. However, effect sizes and cut-offs vary across laboratories and equipment; accordingly, frequency-tuning should be used as a supportive marker within a multimodal battery, not as a sole diagnostic criterion (Curthoys et al., 2018; Curthoys, 2020; Perez-Carpena & López-Escámez, 2019).

Practical implications for MD: document the disease phase at testing; use oVEMP/cVEMP to phenotype utricular vs saccular involvement and to track intra-individual change across attacks; consider comorbid subtypes (e.g., autoimmune, migraine) that may modulate VEMP profiles; and avoid over-interpreting isolated cross-sectional abnormalities—prefer within-patient longitudinal comparisons.

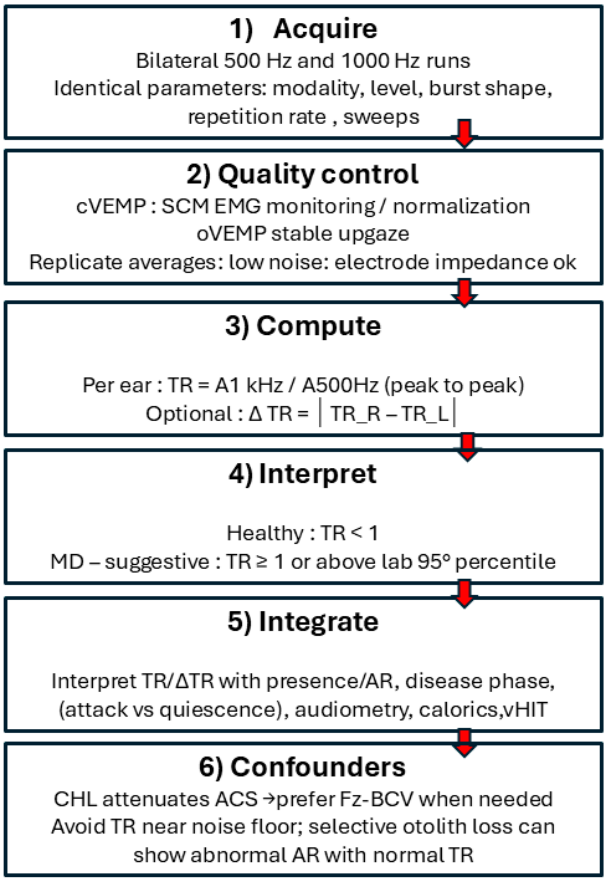


Figure 3. VEMP tuning workflow for Ménière's disease (MD). *(1) Acquire* bilateral **500 Hz** and **1,000 Hz** runs with **identical parameters** (modality, level, burst shape, repetition rate, sweeps). *(2) Quality control:* ensure **SCM EMG monitoring/normalization** (cVEMP), stable **upgaze** (oVEMP), replicate averages, and low noise. *(3) Compute:* per-ear **TR = A1kHz/A500Hz** (peak-to-peak); optionally **ΔTR = |TRR − TRL|**. *(4) Interpret:* **Healthy** → TR < 1; **MD-suggestive** → TR ≥ 1 or above **lab 95th percentile**. *(5) Integrate:* interpret **TR/ΔTR** with **presence/AR**, disease **phase** (attack vs quiescence), audiometry, calorics, and vHIT. *(6) Confounders:* **CHL** attenuates ACS (prefer **Fz-BCV**); avoid TR at **noise floor**; selective otolith losses may show abnormal AR with normal TR.

TR = *Tuning Ratio* = $A_{1k}Hz$ / $A_{500}Hz$, **ΔTR** = $TR_r - TR^l$, **AR** = *Asymmetry Ratio,* **Fz** = *midline forehead,* **ACS** = *Air-Conducted Sound,* **BCV** = *Bone-Conducted Vibration,* **SCM** = *Sternocleidomastoid,* **CHL** = *Conductive Hearing Loss,* **vHIT** = *Video Head-Impulse Test,* **A□□□Hz / A□□Hz** = *peak-to-peak VEMP amplitude measured at 500/1000 Hz.*

Practice Box 1. Frequency-tuning in Ménière's disease: how to compute and report the VEMP tuning ratio

Goal. Capture hydropic "tuning shift" using a simple, reproducible metric.

Stimuli. Record bilateral 500 Hz and 1,000 Hz runs with identical parameters (modality, level, burst shape, repetition rate, sweeps). For cVEMP, monitor/normalize SCM EMG; for oVEMP, stabilize upgaze.

Amplitude. Use peak-to-peak (cVEMP p13–n23; oVEMP n10–p15). Ensure noise <10–20% of signal; otherwise repeat.

Within-ear tuning ratio (TR). (TR = A_{1,kHz} / A_{500,Hz}). If A500 = 0, report TR as not computable and comment on floor effects (consider BCV if ACS is attenuated by middle-ear status).

Between-ear comparison. Optionally compute **ΔTR = |TR_{R} − TR_{L}|**.

Interpretation (principles). In healthy ears, responses typically peak at 500 Hz → TR <1. In many MD ears, tuning shifts toward 1 kHz → TR ≥ 1. Interpret against lab-specific normative percentiles (recommend reporting the 95th percentile of controls for TR and ΔTR).

Reporting template. Provide per-ear amplitudes at **500/1,000 Hz**, **TR**, replicate averages, SCM EMG level (cVEMP) and gaze (oVEMP), stimulus details, and a **brief note** on disease phase (attack vs quiescence).

Caveats. Hearing/conductive components reduce ACS amplitudes (prefer Fz-BCV); avoid TR when amplitudes are at the noise floor; consider that selective otolith losses may retain "normal" tuning despite abnormal asymmetry/presence—use TR alongside presence/AR and clinical context.

Selective otolith loss (utricle vs saccule): definition, detection, and clinical implications

Modern test batteries (6-canal vHIT + oVEMP/cVEMP) reveal that selective loss of individual otolith organs can occur—with otherwise normal canal function. A unilateral utricular loss (UUL) typically presents as a pathologic oVEMP n10 asymmetry to 500-Hz Fz-BCV with preserved cVEMP and normal vHIT. In such cases, the oVEMP AR often exceeds common laboratory normal limits (≈40%). Static otolith tests may be dissociated: depending on etiology and timing, SVV can be normal (isolated transient-system loss) or abnormal (combined sustained/transient involvement). By contrast, selective saccular loss presents as reduced/absent cVEMP with preserved oVEMP and normal vHIT.

Pathophysiology and compensation. The sustained/transient framework explains these dissociations: brief high-frequency stimuli primarily assay irregular striolar/type I pathways, whereas SVV/OCR probe regular/type II pathways. UUL may produce horizontal spontaneous nystagmus via commissural/central integration despite normal canal tests; over time, many patients functionally recover even when oVEMP asymmetry persists, reflecting central compensation (Curthoys et al., 2019; Curthoys, 2020; Curthoys, Burgess & Manzari, 2019). These selective patterns are mechanistically consistent with commissural and cross-striolar inhibition demonstrated in animal models, providing a physiological rationale for large projection-specific VEMP ARs despite preserved canal tests (Uchino & Kushiro, 2011).

Bilateral vestibular loss

Absent oVEMPs in patients with complete bilateral vestibular loss despite preserved hearing, alongside present VEMPs in totally deaf patients with intact vestibular function, confirm the vestibular (otolithic) origin of these responses (Curthoys et al., 2009; Manzari et al., 2010c).

Clinical condition	Stimulation/ recording paradigm (typical)	oVEMP (n10)	cVEMP (p13–n23)	Clinical/diagnostic notes
Superior Vestibular Neuritis (SVN)	**Fz BCV 500 Hz**, ~**7 ms** burst; bilateral infra-orbital recording (upgaze); cVEMP bilaterally on SCM	**Reduced/absent beneath the eye contralateral** to the affected ear ⇒ utricular dysfunction/ superior nerve involvement	**Preserved** bilaterally (saccule and **inferior** nerve intact)	In a cohort of **133** SVN patients, **oVEMP n10 AR cut-off ≈46.5%** yielded **~94%** accuracy for side identification.
Inferior Vestibular Neuritis (IVN)	Same paradigm (**Fz BCV 500 Hz**, 7 ms) replicated with **ACS** for comparison	**Preserved** and symmetric	**Reduced/absent ipsilateral** to the affected ear (saccular/**inferior** nerve)	**Mirror dissociation** vs SVN; pattern consistent for **BCV** and **ACS**, supporting different vestibular origins of oVEMP vs cVEMP and **non-specificity** of the stimulus (both ACS/BCV activate both otoliths).
Ménière's disease (attack vs quiescence)	**Fz BCV 500 Hz**, bilateral cVEMP and oVEMP in **two sessions** (attack/ quiescence)	**Increased (enhanced) contralateral** to the affected side during the **attack**, with higher **asymmetry** vs quiescence	**Reduced** ipsilateral during the **attack** vs quiescence	**Dynamic utricle↑ / saccule↓ dissociation** during attacks. Healthy controls tested twice show no significant changes. Robust results in **15** patients tested in both phases.
Ménière's disease — frequency tuning profile	**Fz-BCV or ACS at 500 and 1,000 Hz** with **identical parameters**; bilateral runs; **50–100 sweeps**; **SCM EMG normalization** (cVEMP) and **upgaze** (oVEMP)	May be normal or reduced; **oVEMP tuning abnormalities** reported; interpret using **TR** and **lab norms**	**cVEMP peak may shift** from **500 Hz → ~1,000 Hz**; compute **tuning ratio TR = A1kHz/ A500Hz**	Healthy ears: **TR < 1**; many MD ears: **TR ≥ 1** or above lab **95th percentile**. Report **TR per ear** and **ΔTR**; use alongside **presence/AR** and disease phase; prefer **Fz-BCV** if ACS is attenuated by **CHL**.

Table 1. Synoptic summary of clinical patterns (oVEMP/cVEMP).
AR = asymmetry ratio; **TR = tuning ratio (A1kHz/A500Hz); ΔTR = interaural tuning ratio difference;** *Fz = midline forehead; ACS = air-conducted sound; BCV = bone-conducted vibration; SCM = sternocleidomastoid.*

Item	ACS (air-conducted sound)	Fz-BCV (midline forehead bone-conducted vibration)	Notes / Criteria
Stimulus frequency	**4,000 Hz**	**4,000 Hz**	Maximizes separation between SCD ears and normals.
Waveform	Short tone burst; fast rise/fall (~2–3 ms); minimal/zero plateau (e.g., raised-cosine/ Blackman)	Same	Sharp onset enhances neural synchrony and n10 detectability.
Repetition rate	~4–5 Hz	~4–5 Hz	Balances averaging time and myogenic/ EEG contamination; aligns with published protocols.
Sweeps per run	50–100	50–100	Acquire ≥2 averaged runs per condition to confirm reproducibility.
Recording montage	Infra-orbital electrodes; upgaze 25–30°	Same	Standard oVEMP setup; maintain stable gaze and low impedance.
Primary outcome	Presence/absence of **contralateral n10** at 4 kHz	Presence/absence of **contralateral n10** at 4 kHz	Amplitude optional; **AR usually unnecessary** because normals typically show no response at 4 kHz.
Positivity criterion	Clear, replicable contralateral n10 at 4 kHz	Clear, replicable contralateral n10 at 4 kHz	Indicates **third-window physiology** → triage for temporal-bone CT.
When to prefer	Fast to deploy; but reduced by **conductive** components (risk of false-negatives)	Bypasses middle ear; useful with CHL/ effusion; symmetric skull drive	Choose modality based on middle-ear status and lab hardware.
Pitfalls/differentials	Other third-window lesions may also be positive; conductive loss can mask ACS responses	Poor skull coupling can reduce BCV; ensure firm, midline placement	Interpret in clinical context; confirm with imaging.
Post-operative marker	Typically **abolished** after canal plugging	Typically **abolished** after canal plugging	Objective indicator of surgical success.

Table 2. Recommended high-frequency (4,000 Hz) oVEMP test parameters for third-window screening (suspected SCD). ACS = air-conducted sound; BCV = bone-conducted vibration; Fz = midline forehead; AR = asymmetry ratio; CHL = conductive hearing loss. Key sources: Manzari et al., 2013; Curthoys & Manzari, 2020.

Beyond ACS/BCV: Low-Intensity Focused Ultrasound (LiFU)

LiFU can drive utricular and saccular afferents via acoustic radiation force with waveform control spanning “vibration-like” (transient) to “tilt-like” (sustained) regimes, offering a laboratory probe of otolith subsystems without moving the subject. Clini-

cal translation is preliminary (Iversen et al., 2017).

Clinical Algorithm (pragmatic)

1. **Routine otolith screen: Fz-BCV 500 Hz** oVEMP (contralateral n10) + cVEMP (ipsilateral p13–n23), report amplitudes/latencies/AR with EMG and gaze documentation.
2. **Add 6-canal vHIT** for canal-specific dynamics; consider SVV/OCR if static otolith function is clinically relevant (Curthoys, 2020).
3. **If SCD suspected:** include one **4,000-Hz oVEMP** trial to triage for CT (Manzari et al., 2013; Curthoys & Manzari, 2020).
4. **Interpretation by projection:** utricle ↔ **oVEMP**, saccule ↔ **cVEMP**; avoid attributing organ by stimulus type (Curthoys et al., 2018).

Limitations

AR cut-offs and normative data are lab-specific; cVEMP depends on tonic SCM, oVEMP on upgaze and electrode placement. Absence of a VEMP does not prove complete organ loss—corroborate with complementary static and canal tests. LiFU evidence remains pre-clinical (Curthoys et al., 2018; Iversen et al., 2017).

Conclusions

Framed within the sustained/transient physiology of the otoliths, VEMPs offer rapid, objective readouts of transient (irregular, striolar) function using brief ≈500-Hz ACS/BCV bursts. Attribution to utricle vs saccule stems from reflex projection—not stimulus class—so that oVEMP (contralateral n10) reads out the utriculo-ocular pathway and cVEMP (ipsilateral p13–n23) the sacculo-collic pathway (Curthoys et al., 2017; 2018; 2019). At clinical stimulus levels, convergent mechanistic and human data indicate that the cVEMP is a specific indicator of saccular/inferior nerve function, with negligible canal contribution in intact labyrinths (Curthoys et al., 2025).

Clinically, standardized Fz-BCV/ACS 500 Hz protocols reveal reproducible dissociations in SVN and IVN, capture phase-dependent changes in Ménière's disease, and—together with frequency-tuning paradigms—support phenotyping of hydropic ears. In MD, VEMPs remain supportive rather than stand-alone diagnostics due to protocol heterogeneity and modest pooled accuracy (Perez-Carpena & López-Escámez, 2019). Integrating oVEMP/cVEMP with 6-canal vHIT refines topodiagnosis and longitudinal follow-up. For third-window disorders, a 4,000-Hz oVEMP provides a time-efficient rule-in screen with a robust canal–otolith physiological basis; a positive result indicates third-window physiology and should prompt imaging confirmation (Manzari et al., 2013; Curthoys & Manzari, 2020).

Implementation hinges on technical standardization (SCM EMG monitoring/normalization, stable upgaze, symmetric Fz coupling, electrode/impedance quality) and on lab-specific normative data with transparent reporting of stimulus design and averaging. Looking ahead, cross-platform calibration of asymmetry ratios, wider adoption of frequency-specific/tuning paradigms (including 4 kHz and 1-kHz vs 500-Hz comparisons), and exploratory modalities such as low-intensity focused ultrasound may further strengthen the physiological specificity and clinical utility of VEMP testing (Iversen et al., 2017).

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