vHIT, Calorics & Rotational Chair Testing:

A Comprehensive Clinical Review

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Section 3B — VOR & Canal Assessment | Vestibular Function Testing Series

How to Use This Review

This document is the companion clinical literature review to the video head impulse testing, caloric irrigation, and rotational chair assessment video series on the ADC education hub at www.australiandizzinessclinics.com. It is designed for vestibular physicians, audiologists, and neurologists building expertise in laboratory vestibular function testing.

The review follows clinical testing sequence: from theoretical foundations and neural substrates through methodology, normative values, interpretation frameworks, and clinical application. Callout boxes throughout identify clinically high-yield points and evidence-based pearls.

Callout box guide:

 Clinical Insight: *Clinically relevant observations derived directly from the basic science — the bridge between laboratory findings and patient management.*

 Clinical Pearl: *High-yield, memorable clinical points — the key facts that separate a competent clinician from an expert in vestibular function testing.*

 Key Point: *Foundational concepts and summary statements that anchor the clinical framework. Master these to interpret the full testing battery.*

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Introduction

This comprehensive review examines the three primary diagnostic pillars of vestibular assessment: the **Video Head Impulse Test (vHIT)**, **Caloric Testing**, and **Rotational Chair**

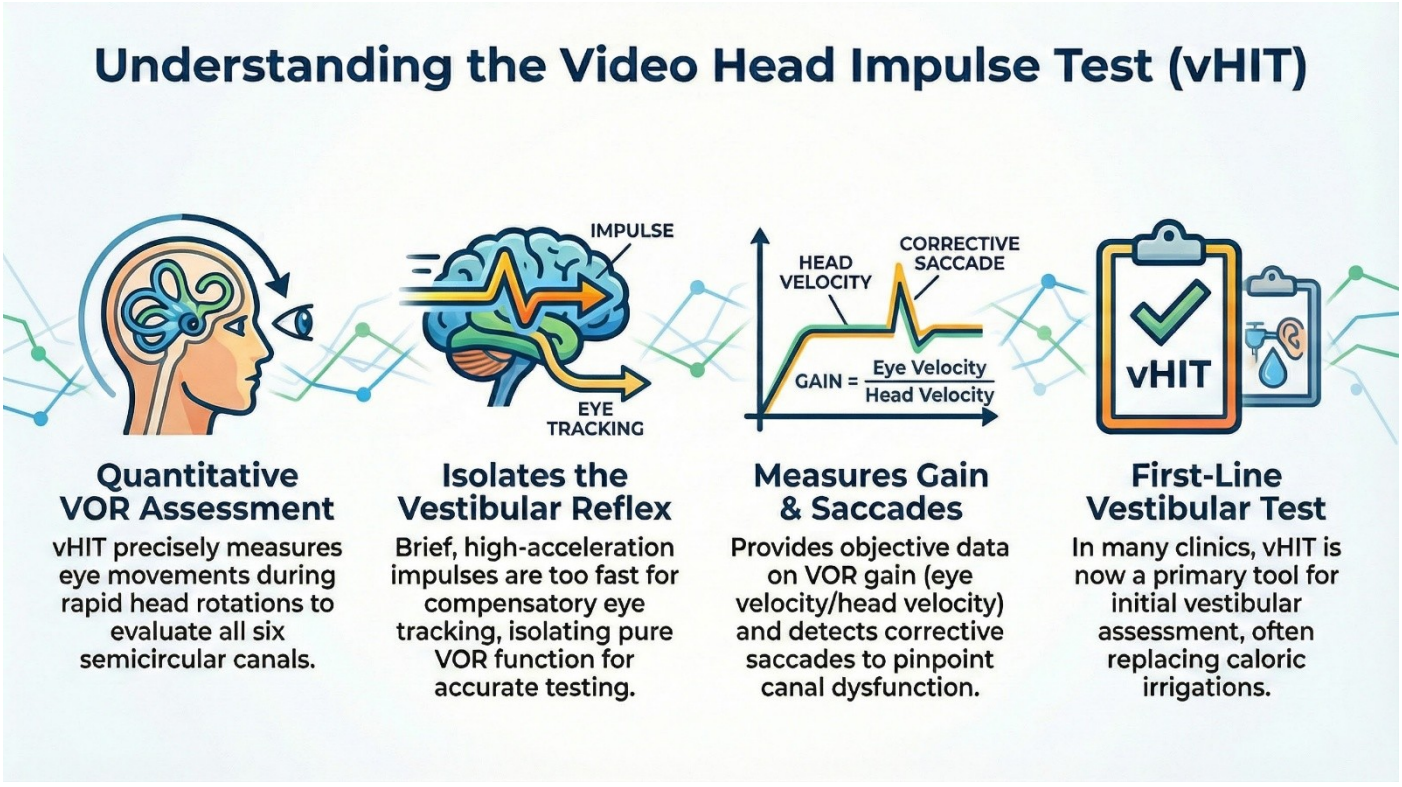
Testing. In modern clinical practice, these modalities form a "triad" of evaluation, allowing clinicians to probe the Vestibulo-Ocular Reflex (VOR) across its entire functional frequency spectrum—from the ultra-low frequency of caloric irrigation to the high-acceleration, physiologic demands of head impulses.

While the **vHIT** offers rapid, canal-specific data at high frequencies and is essential for acute diagnosis, the **Caloric Test** remains the gold standard for identifying subtle unilateral weaknesses at low frequencies. **Rotational Chair Testing** bridges these gaps, providing a global assessment of bilateral function and central compensation. By integrating these tests, clinicians can precisely localize lesions, differentiate peripheral from central pathologies, and design targeted rehabilitation strategies for patients suffering from balance and dizzy disorders.

Video Head Impulse Test (vHIT)

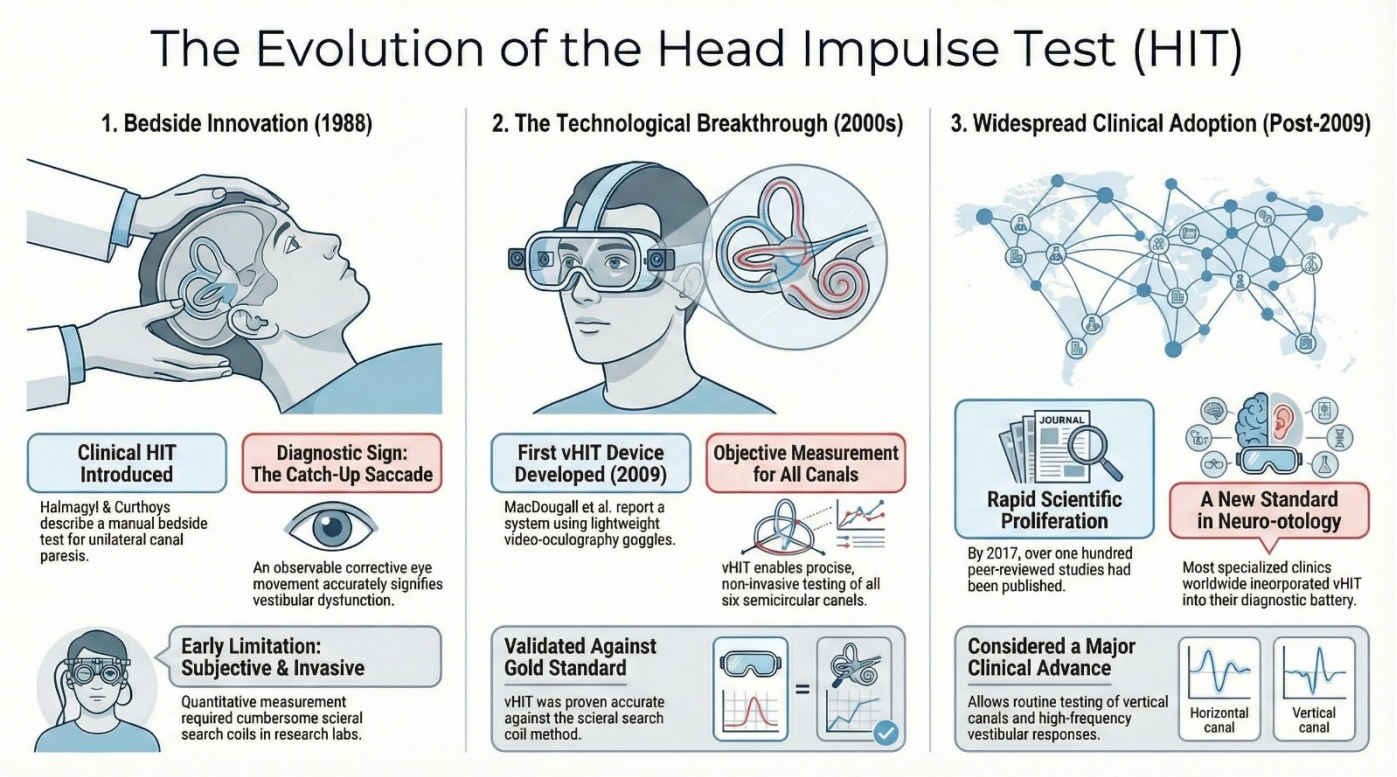
Introduction

The video head impulse test (vHIT) is a modern tool for quantitatively evaluating the vestibulo-ocular reflex (VOR) by measuring eye movements during rapid, passive head rotations. It is essentially an instrumented version of the bedside head impulse (or “head thrust”) test first described in the late 1980s, now expanded to assess all six semicircular canals. vHIT delivers high-acceleration head impulses (up to 2,000–4,000°/s²) that are too brief for the patient’s predictive eye tracking or other oculomotor mechanisms to compensate, thereby isolating the vestibular contribution to gaze stability. By recording eye velocity with high-speed video and comparing it to head velocity, vHIT provides an objective measure of VOR gain (eye velocity/head velocity) and detects corrective saccades, allowing precise assessment of semicircular canal function in patients with dizziness and balance disorders. In many clinics, vHIT has become a first-line vestibular test, often supplanting caloric irrigations for initial assessment of peripheral vestibular function.



Historical Development and Major Contributors

The clinical head impulse test (HIT) was introduced by Halmagyi and Curthoys in 1988 [1] as a bedside manoeuvre to reveal unilateral semicircular canal paresis. Using rapid, small-amplitude head rotations, they showed that an absence of the normal compensatory eye movement (with a corrective catch-up saccade instead) accurately signified vestibular hypofunction of the tested canal. For about two decades, precise quantitative HIT was only possible in research laboratories via scleral search coils – a cumbersome and invasive technique available to only a few specialists. The development of lightweight video-oculography in the 2000s was a breakthrough. MacDougall et al. (2009) [2] reported the first vHIT device, enabling objective measurement of head impulse responses for all six canals with a simple video goggles system. Halmagyi and colleagues subsequently refined the video HIT in 2009 [3] and 2013 [4], demonstrating its clinical utility and paving the way for widespread adoption. Other key contributors include Ian Curthoys [30] (physiology of head impulses) and Konrad Weber [5] and Michael Strupp's [6] groups (validating vHIT against the gold-standard coil recordings). Since its introduction, vHIT technology has rapidly proliferated: by 2017 [7], over a hundred peer-reviewed studies had been published, and most neuro-otology clinics worldwide had incorporated vHIT into their diagnostic battery. This innovation is considered a major advance, allowing clinicians to test individual vertical canal function and high-frequency vestibular responses in routine practice for the first time.



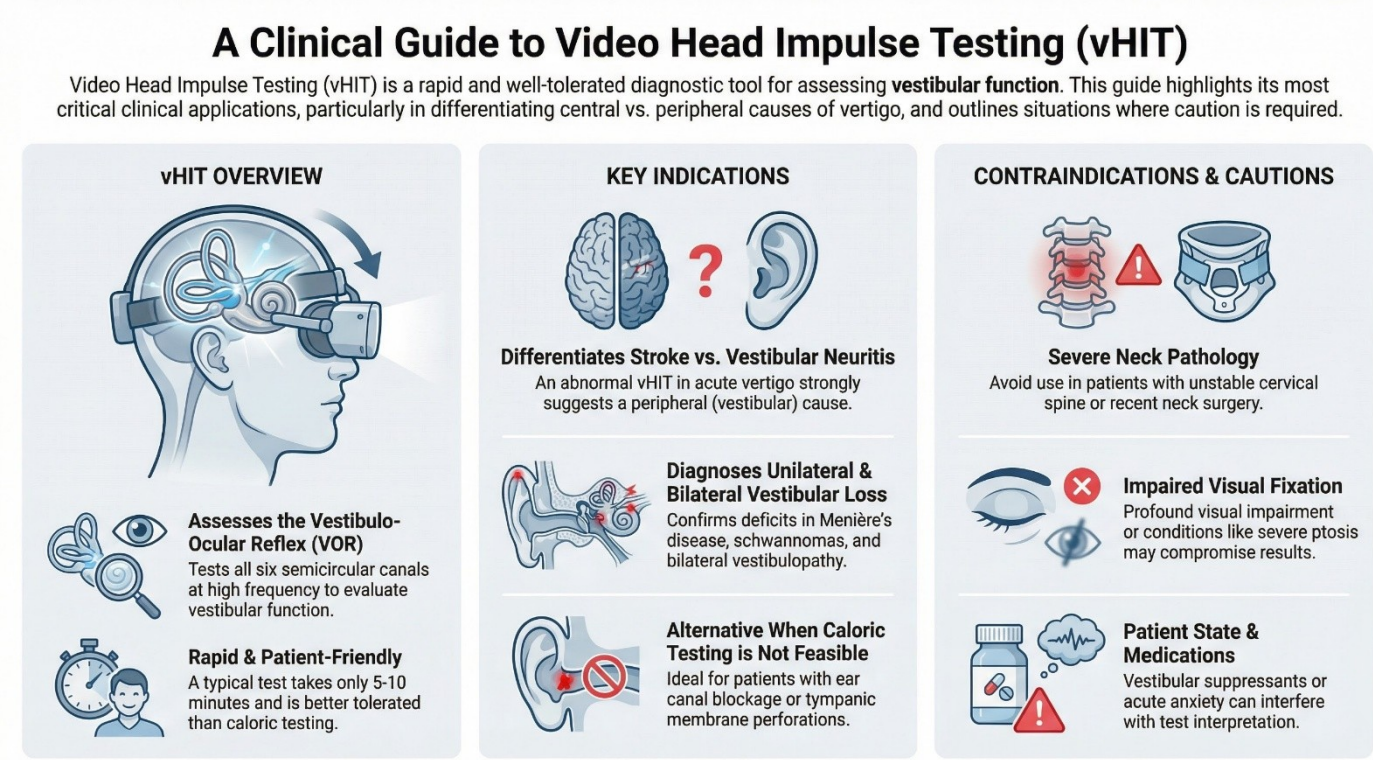
Indications

vHIT is indicated in the evaluation of virtually any patient with suspected vestibular dysfunction, especially those with vertigo, unexplained dizziness, chronic imbalance, or oscillopsia (visual blurring with head movements). It is particularly useful for acute vestibular syndrome: in an emergency setting, the pattern of vHIT results can help distinguish vestibular neuritis (peripheral acute unilateral loss) from stroke (central cause of acute vertigo). An abnormal head impulse (low gain with catch-up saccades) strongly supports a peripheral vestibular lesion, whereas a normal vHIT during acute continuous vertigo raises concern for a central lesion. Clinicians also

rely on vHIT for diagnosing unilateral vestibulopathies such as vestibular neuritis/labyrinthitis, Menière's disease (during and between attacks), and vestibular schwannomas, as it can confirm a deficit in the affected ear's canals. In addition, vHIT plays a critical role in diagnosing bilateral vestibular loss: patients with bilateral vestibulopathy show reduced VOR gain in both horizontal directions (and often vertical canals as well), with catch-up saccades, which vHIT can rapidly confirm. This is a major advantage over caloric testing, which is time-consuming and often intolerable in bilateral cases. vHIT is frequently the first vestibular test performed in outpatient clinics because it is quick (~5–10 minutes), well tolerated, and does not require the patient to be in darkness or prone to vertigo as calorics do. It can be repeated to track recovery or progression of vestibular deficits over time, making it useful not only for diagnosis but also for monitoring rehabilitation in vestibular disorders. Finally, vHIT is indicated when caloric testing is contraindicated or not feasible – for example, in patients with occluded ear canals, tympanic membrane perforations, or anatomical variations that preclude caloric irrigations. In such cases, vHIT can provide an alternative assessment of canal function. Overall, vHIT's ability to assess all semicircular canals (horizontal and vertical) at high frequency makes it an indispensable tool for comprehensive vestibular evaluation.

Contraindications

There are no absolute contraindications to vHIT, but certain conditions may limit the test's use or require caution. Patients with severe neck pathology (unstable cervical spine, recent neck surgery, advanced cervical spondylosis) should generally not undergo the rapid head rotations of vHIT to avoid injury. Similarly, significant atlantoaxial instability or rheumatoid arthritis affecting the cervical spine would preclude head impulse testing. In patients with limited neck mobility or severe neck pain, vHIT may be impractical; a rotational chair test can be considered instead in such cases. Eye conditions that interfere with tracking could pose problems – for example, profound visual impairment (the patient must be able to fixate on a target), severe ptosis, or ocular surface issues preventing proper calibration. However, moderate loss of visual acuity (if the patient forgets their glasses) does not significantly affect vHIT outcomes. If a patient cannot maintain fixation or has spontaneous nystagmus in primary gaze, the vHIT traces can be difficult to interpret reliably. Additionally, although not a strict contraindication, patients in an acute vertigo crisis (e.g. early Ménière's attack) or those extremely anxious about sudden head movements may not tolerate vHIT well – in such scenarios the clinician might defer the test until the patient is calmer or the acute phase has passed. It is also advised to withhold vestibular suppressant medications (benzodiazepines, antihistamines, etc.) before vHIT when possible, as these can reduce the VOR gain or saccadic responses and potentially mask deficits (similar to their effect on caloric responses). In summary, while vHIT is safe and noninvasive, one must use clinical judgment regarding neck stability and patient cooperation. If vHIT cannot be performed, other vestibular tests (caloric or rotational chair) may fill the gap.



Methods (Clinical and Lab Technique)

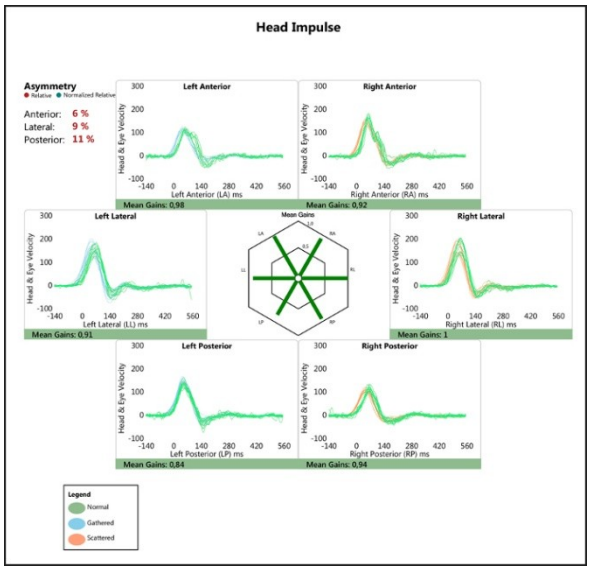
Setup: vHIT can be conducted in a well-lit room and does not require darkness because visual fixation does not suppress the high-acceleration VOR response. The patient wears a lightweight goggle frame equipped with a high-speed infrared camera (typically 250–300 Hz or higher) to record one eye and an inertial sensor to measure head motion. The examiner first calibrates the system by having the patient look at known target points so that eye position can be accurately measured. The patient is seated roughly 1 meter in front of a small fixation target at eye level, which they are instructed to stare at throughout testing.

Technique: The examiner stands behind or beside the patient and delivers a series of rapid, passive head impulses in the plane of the semicircular canal pair being tested. For horizontal canals, this means unpredictable small-amplitude (~10–20°) head rotations to the right and left (yaw axis), delivered at high velocity (~150–200°/s) and high acceleration (>2,000°/s²). Each impulse is a brisk "thrust" lasting only ~150–200 milliseconds. The key is that the patient does not know the timing or direction in advance, so they cannot pre-emptively refixate. The goggles simultaneously record head angular velocity and eye velocity; a normal response is one where the eyes move equal and opposite to the head, keeping gaze on target (VOR gain ~1.0). After several training impulses, approximately 10–20 impulses are performed to each side to ensure reproducibility. Vertical canals are tested by rotating the head in the plane of the anterior-posterior canal pairs ("RALP" for right anterior/left posterior, and "LARP" for left anterior/right posterior), which involves pitching the head diagonally while the patient fixates on a target straight ahead. Modern vHIT devices provide real-time feedback on technique (e.g. impulse speed and direction), helping the examiner deliver adequate impulses. Between impulses, the patient is reminded to

blink normally (to avoid excessive blinks during impulses) and keep fixing on the target. No vision-denial or dark room is needed, unlike caloric testing.

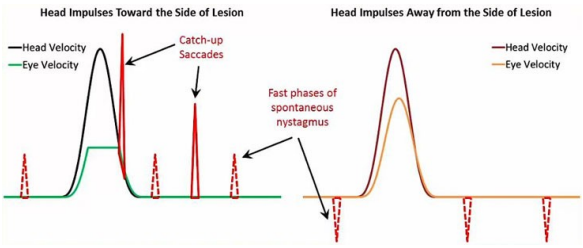
Recording and Data: The software plots head velocity and eye velocity versus time for each impulse. In a normal response, the eye velocity trace (dashed line) is nearly the mirror image of the head motion trace (solid line), indicating a compensatory eye movement equal in magnitude and opposite in direction (see Figure: normal vHIT). The device calculates gain either as the ratio of peak eye/head velocities or by integrating the area under the curve, depending on the manufacturer's algorithm. After multiple impulses, an average gain for each canal is obtained. Critically, the system also detects and charts any corrective saccades – rapid eye movements that the patient's brain generates if the VOR was insufficient to maintain fixation. These saccades can occur after the head stop (overt saccades) or during the head movement (covert saccades). The presence, timing, and amplitude of these catch-up saccades are as important as the gain value in interpretation. A complete vHIT battery tests all three canals on each side (horizontal, anterior, posterior) and typically takes only a few minutes once the patient is prepared. The test is considered finished when reliable, artifact-free traces have been obtained for each canal in both directions.

Interpretation of Results



Normal vHIT: A normal finding is a VOR gain close to 1.0 with no corrective saccades. For lateral (horizontal) canals, healthy individuals generally have gain in the range 0.9–1.0 (often a bit lower in older patients), and for vertical canals slightly lower (approximately 0.7–0.9) due to the more complex eye movement geometry. The eye velocity should match head velocity almost perfectly for the first ~100 ms of head turn, keeping the gaze on target. The vHIT report typically shows superimposed head and eye traces with the eye trace equal and opposite to head movement, and the computed gain falling within normal limits (e.g. >0.8 lateral, >0.7 vertical).

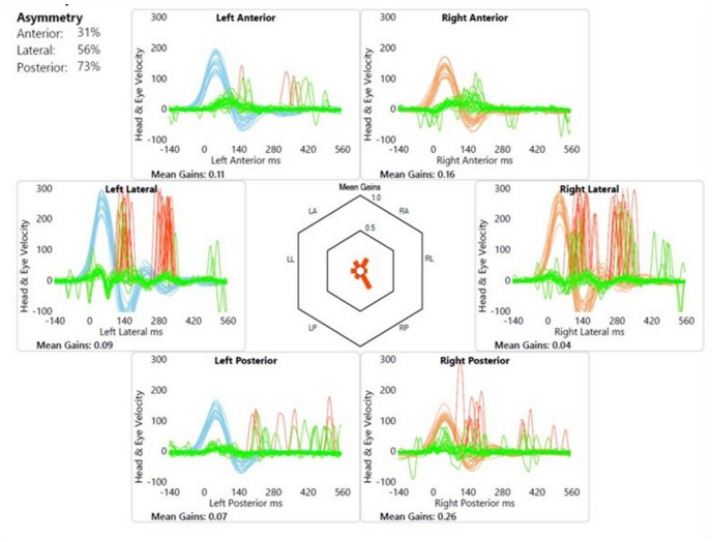
Peripheral Vestibular Loss: An abnormal vHIT is characterized by a reduced gain on rotations stimulating the affected canal, accompanied by corrective saccades. In a unilateral vestibular hypofunction (e.g. right vestibular neuritis), head impulses to the right (stimulating the right horizontal canal) will yield a low gain (eye movement smaller/slower than head) and the patient's eyes will slip off target toward the right with the head movement. The brain detects this slippage and triggers a catch-up saccade back to the target once the head stops (an overt saccade). If the patient has learned to compensate, a covert saccade may also occur during the head movement.



Overt saccades are visible on exam and indicate a deficient VOR; covert saccades are only seen on the vHIT traces (high-speed camera) and reflect partial compensation (the patient's cervico-ocular reflex or predictive strategies triggering a corrective eye movement sooner). Either type of saccade indicates an abnormal test. The pattern of affected canals can be very informative: for example, an isolated horizontal canal deficit with normal vertical canals might suggest a lateral semicircular canal lesion, whereas absent responses in both the horizontal and anterior canals of one ear (with preserved posterior canal function)

is a classic sign of superior vestibular nerve neuritis (selective involvement of the superior division of the vestibular nerve). vHIT thus allows localization to specific canal or nerve divisions, which caloric testing cannot achieve.

Bilateral vestibulopathy: vHIT can also reveal bilateral vestibular loss. If all canals on both sides show low gain and there are catch-up saccades after head impulses in both directions, this indicates bilateral dysfunction. Typically, the gain is depressed (often <0.5 bilaterally) and overt or covert saccades occur with impulses to either side. Sometimes the saccades in bilateral loss are small and scattered, reflecting that neither ear can generate a normal VOR. In advanced bilateral vestibulopathy, patients may have virtually absent VOR and instead make multiple corrective saccades for any head movement. vHIT provides a rapid confirmation of such cases, which is advantageous since caloric responses may be absent bilaterally and rotational chair testing (the gold-standard for bilateral loss) might not be readily available.



Central patterns: Generally, an intact VOR on vHIT (normal gain with no catch-up saccades) implies that the peripheral vestibular apparatus and the vestibular nerve are functioning. However, certain central lesions can produce distinctive vHIT findings. One example is in some cerebellar diseases or vestibular migraine, where an elevated VOR gain (>1.0) may be observed. An abnormally high gain, sometimes accompanied by what are called “anti-compensatory” saccades (saccades in the opposite direction of what a catch-up would be), suggests a central pathology interfering with normal VOR calibration. In other cases of central vestibular dysfunction, vHIT can be entirely normal despite significant symptoms, because the pathology lies beyond the peripheral reflex arc (for instance, a brainstem stroke sparing the vestibular nuclei can cause vertigo and nystagmus but still yield a normal vHIT). Thus, clinicians interpret vHIT in context: normal vHIT with ongoing vestibular symptoms may prompt a search for central causes, whereas peripheral lesions almost always show a deficit on vHIT if the lesion affects high-frequency canal function.

Abnormalities and Diagnostic Patterns

vHIT findings, combined with other tests, allow characterization of vestibular disorders:

Acute unilateral vestibulopathy (Vestibular Neuritis/Labyrinthitis): In the acute phase, patients have a deficient horizontal canal VOR on the affected side – vHIT shows significantly reduced gain and obvious overt saccades when the head is rotated toward the lesioned ear. Often both type I and II hair cell functions are initially impaired, so both vHIT (high-frequency) and caloric (low-frequency) tests will be abnormal acutely. Over weeks to months, partial recovery occurs preferentially in the high-frequency range: patients often regain near-normal vHIT gains (especially with covert saccades indicating central compensation), while caloric deficits may persist. The presence of only covert saccades on follow-up vHIT suggests compensation – the patient may experience fewer symptoms even though caloric testing still shows a weakness. This pattern (normalizing vHIT, persistent caloric weakness) is common in well-compensated unilateral vestibular loss. In contrast, a patient with persistent overt saccades on vHIT likely has an uncompensated or more severe deficit. By testing all canals, vHIT can also identify if the superior or inferior division of the vestibular nerve is affected. For example, a classic

superior neuritis yields abnormal vHIT in the ipsilateral horizontal and anterior canals but a normal posterior canal VOR, whereas labyrinthitis (involving the entire labyrinth) would affect all three canals on that side.

Ménière's Disease: A distinctive discrepancy often seen in early Ménière's disease is normal vHIT results alongside abnormal caloric results. Early in the disease, the high-frequency VOR may remain intact (normal vHIT gain, no saccades) because type I hair cells can still function, whereas low-frequency VOR is depressed (reduced caloric response) due to endolymphatic hydrops damping the low-frequency dynamics. Thus, a patient with early Ménière's may have a normal vHIT but significant unilateral weakness on caloric testing. As Ménière's disease advances or in more severe cases, both low- and high-frequency function become impaired – vHIT will then show reduced gains for the affected ear's canals and catch-up saccades, concordant with caloric loss. vHIT can also catch transient changes: during some acute Ménière's attacks, patients transiently have abnormal vHIT (due to sudden loss of function during the vertigo spell), which may improve between episodes.

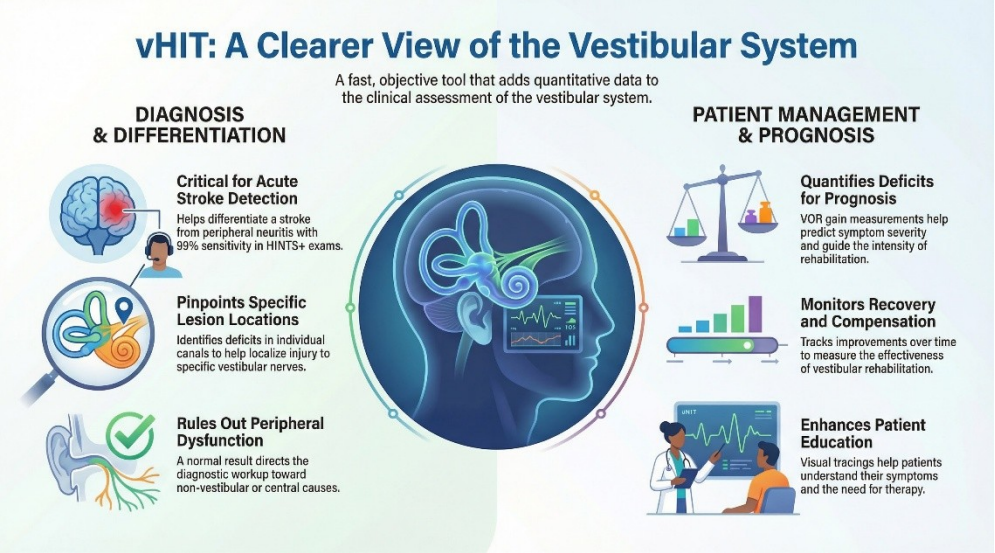
Bilateral Vestibular Loss: As noted, bilateral vestibulopathies (from ototoxic antibiotics, bilateral Meniere's, bilateral vestibular neuritis, or degenerative causes) manifest as reduced gains and bilateral catch-up saccades on vHIT. vHIT is a fast way to confirm bilateral loss, which is crucial for explaining symptoms like oscillopsia. It also allows grading of severity: for instance, a patient with severe bilateral loss will have gains near 0.2–0.3 and multiple large saccades, whereas mild bilateral loss may show gains around 0.7 with only small corrective saccades. Studies have shown vHIT can achieve 100% sensitivity [8] for detecting severe bilateral vestibulopathy when average gains are very low. However, vHIT might miss milder bilateral reductions (because the symmetric deficit yields symmetric, near-normal appearing responses if above the threshold for saccade generation). In those cases, rotational chair testing, which can quantify absolute gain, is more sensitive.

Central Vestibular Disorders: Certain central disorders can be suspected from vHIT patterns. For example, in progressive supranuclear palsy or certain cerebellar ataxias, patients can show an intact or even hyperactive horizontal VOR on vHIT but have balance issues and an inability to modulate VOR gain (e.g. impaired cancellation). If vHIT shows normal gain but the patient has gaze instability complaints, a central pathology such as a cerebellar flocculus lesion might be considered – such lesions often cause failure of VOR suppression rather than loss of VOR. Another central clue is disconjugate catch-up saccades or unusual eye movement profiles on vHIT, which might indicate brainstem pathology affecting ocular motor nuclei or internuclear pathways, rather than a peripheral vestibular lesion.

In practice, clinicians use vHIT in conjunction with caloric and other tests to discern patterns. For instance, a normal vHIT + abnormal caloric pattern points toward a selective low-frequency dysfunction (common in early Ménière's or small vestibular schwannomas), whereas an abnormal vHIT + abnormal caloric implies a broad frequency spectrum loss (as in vestibular neuritis or advanced hydrops). An abnormal vHIT + normal caloric is unusual for peripheral lesions (since severe high-frequency loss usually also depresses calorics), so such a finding might raise the possibility of a central issue or a technical problem. Careful analysis of vHIT saccades (latency, size, presence on both sides) and gain values helps resolve these diagnostic questions.

Clinical Implications

vHIT has significant clinical utility in vestibular diagnosis and patient management. Perhaps its most celebrated application is in the HINTS plus exam [9] for acute vestibular syndrome: in combination with nystagmus assessment and skew deviation test, the head impulse (bedside vHIT) helps differentiate stroke from peripheral vestibular neuritis in patients with continuous vertigo. A normal head impulse during an acute vestibular syndrome strongly suggests a central lesion (since an acute peripheral loss should cause an abnormal HIT); this insight has 99% sensitivity for stroke when combined with the other HINTS signs. In less acute settings, vHIT results guide the diagnostic localization: for example, if a patient has chronic disequilibrium and vHIT shows an isolated deficient posterior canal on one side, one might suspect a selective injury to the inferior vestibular nerve (perhaps due to a small infarct or neuritis confined to that branch). Meanwhile, normal vHIT across all canals in a dizzy patient would redirect the workup toward non-vestibular causes or central causes (such as anxiety, orthostatic hypotension, cerebellar ataxia, etc.), especially if other vestibular tests are also normal. Thus, a normal vHIT can sometimes reassure both physician and patient that the peripheral vestibular function is intact, and attention can be turned elsewhere.



vHIT's ability to quantify bilateral vestibular failure is very important for prognostication. A patient with bilateral VOR gains of 0.2 will likely have significant oscillopsia and require intensive vestibular rehabilitation and safety counselling (falls prevention, avoiding driving at night, etc.), whereas someone with gains of 0.8 might be much milder. Moreover, vHIT can monitor compensation and recovery. Improvement in VOR gain or disappearance of overt saccades over time indicates central compensation and vestibular rehab efficacy, while persistent deficits might prompt more rehab or consideration of vestibular implant trials in the future. Clinicians also find vHIT useful to guide further testing: for instance, if vHIT is clearly abnormal for the lateral canal, one may forego caloric testing or do a simpler monothermal caloric, since the presence of a significant deficit is already established. Conversely, if vHIT is normal but suspicion for vestibular dysfunction remains (e.g. a classic history of unilateral Menière's episodes), the clinician will ensure caloric testing or rotational chair testing is done to probe low-frequency function.

From a patient counselling perspective, showing patients their vHIT tracings can be very illustrative: they can see the corrective saccades their eyes must make, which validates their symptoms and the need for therapy. In cases of unilateral vestibular loss, documentation of an abnormal vHIT (and caloric) on that side not only clinches the diagnosis but also provides a baseline to gauge any future changes (for example, if a patient with Menière's disease later develops bilateral weakness). Because vHIT covers all canals, it can detect subclinical bilateral involvement (e.g. subtle anterior canal hypofunction in the contralateral ear of a unilateral case), which has implications for how aggressively to pursue preventative measures (like gentamicin ablation in Menière's might be avoided if the contralateral ear shows any deficit on vHIT). In summary, vHIT has become an invaluable extension of the vestibular bedside exam, confirming peripheral vestibular lesions, distinguishing central mimics, and informing prognosis and

management strategies – all with a fast, objective test that adds quantitative data to the clinical assessment.

Pitfalls and Limitations

Despite its usefulness, vHIT has several important limitations and potential pitfalls.

Sensitivity in mild lesions is a known issue: vHIT predominantly probes high-frequency function, and a vestibular lesion that is mild or mainly affects low-frequency dynamics may still yield a "normal" vHIT. Studies have noted [10] that if a unilateral caloric weakness is less than about 40%, the lateral canal vHIT is often within normal limits. In other words, early or partial deficits can be missed by vHIT, which could give a false sense of normalcy if used in isolation. This underscores that a normal vHIT does not entirely rule out vestibular impairment – complementary tests may be needed for low-frequency evaluation. Conversely, when vHIT is clearly abnormal, caloric testing is almost never normal. Another subtle point is that bilateral symmetric vestibular losses may be under-recognized: if both ears are equally impaired, vHIT will show reduced gain on both sides, but without a normal ear for comparison, it requires careful analysis to appreciate that gains are borderline and saccades, if present, may be small. It is possible for an inexperienced interpreter to overlook a mild bilateral hypofunction as "within normal," so one must examine the absolute gain values and look for any catch-up saccades on either side.

Technical and operator-dependent pitfalls are also significant. Proper calibration and goggle fit are critical – slippage of the goggles on the head can artifactually reduce VOR gain (because the camera moves relative to the eye). If the impulse is not truly "unpredictable" or if the patient anticipates it, they might suppress their VOR or generate pre-programmed eye movements, leading to erroneous results. Likewise, impulses that are too slow or too small in amplitude may not reach the threshold to fully engage the high-frequency VOR, again yielding falsely normal gains. Modern devices attempt to coach the operator with feedback (e.g. warning if head velocity was too low), but user training remains important. Ocular artifacts like blinks or interference by eyelids and eyelashes can distort the eye velocity recording. A common artifact is a refixation saccade being missed by the software or misattributed; another is an apparent high gain if the patient makes an opposite saccade during the impulse. Careful review of raw traces helps avoid these misinterpretations. It's recommended to focus on the presence of genuine catch-up saccades as the most robust sign of deficit, since small gain measurement errors can occur but do not typically produce full corrective saccades.

Patients with certain eye movement disorders (such as an impaired ability to generate saccades, or spontaneous nystagmus) present interpretation challenges. If a patient cannot generate a normal saccade due to a supranuclear palsy, for example, vHIT might show low gain without clear saccades, possibly mimicking bilateral vestibulopathy. On the flip side, a patient with baseline nystagmus (e.g. an acute vestibular lesion) will have that nystagmus superimposed on the head impulse traces, which can create asymmetry or directional preponderance artifacts. Many vHIT devices allow examiners to subtract out baseline nystagmus or exclude impulses where the traces were confounded. Visual fixation is another consideration – the patient must keep looking at the target; if they glance away or are not vigilant, the test is invalid. To ensure alertness (particularly in follow-up tests that can become routine), some clinicians have patients perform mental tasks or stare at optotype characters as the target.

Equipment limitations: vHIT cameras have finite resolution and sampling rate. Very rapid saccades (covert saccades with small amplitudes) might not be perfectly captured depending on frame rate. Also, vertical canal testing can be more variable because torsional eye movement components are not measured – the device only tracks the vertical component for anterior/posterior canals, which is an approximation. Therefore, vertical vHIT results must be interpreted with somewhat larger thresholds for abnormal (normal gain for vertical canals is a bit lower and more variable). Another limitation is that vHIT assesses primarily the lateral and vertical semicircular canals; it does not test otolith organ function (utricle and saccule) – other tests like the ocular and cervical VEMPs are needed for that.

False positives and negatives: With an inexperienced operator or suboptimal conditions, vHIT can yield false positives (e.g. an apparent low gain due to goggle slip or patient non-compliance, leading to an incorrect diagnosis of hypofunction). It can also yield false negatives in the situations discussed (compensated or mild deficits). To mitigate this, results that seem surprising or "too interesting" should be cross-checked and interpreted in light of physiology. For example, a unilateral low gain without any saccades is suspicious for artifact – a true vestibular loss should evoke catch-up saccades unless the patient is completely inattentive or using some

extraordinary compensation. If vHIT results are equivocal or conflict with other clinical findings, one should repeat the test or proceed to caloric/rotational testing rather than making a definitive conclusion.

In summary, while vHIT is extremely valuable, it is not foolproof. Awareness of its blind spots – low-frequency sensitivity, need for proper technique, and artifact recognition – is essential for the neuro-otologist. When used in conjunction with other tests and careful clinical correlation, the pitfalls can be navigated, and the vast majority of vestibular lesions can be accurately detected.

Future Directions

vHIT technology and applications continue to evolve. One new variant of the head impulse test is the suppression head impulse paradigm **(SHIMP)** [11], in which the patient is asked to track a moving target that moves with the head, rather than a fixed target. In a SHIMP test, a normal vestibular system will produce a high-gain VOR (eyes move opposite head) which is actually maladaptive for keeping eyes on the head-fixed target, so a healthy subject must generate a saccade toward the direction of head rotation (a “backup” saccade) to reacquire the target. By contrast, a vestibular-loss patient, whose eyes move with the head due to deficient VOR, will keep the target in view and need no saccade. In essence, SHIMP produces a reversed situation: it measures how well patients can not use their VOR. Early studies suggest SHIMP can complement traditional vHIT (HIMP) by providing additional information on any residual vestibular function and central compensation – patients with vestibular loss often show absent SHIMP saccades on the lesioned side (because there is no VOR to suppress), whereas their normal-ear SHIMP saccades indicate preserved function. This paradigm may better visualise covert compensatory saccades as well. SHIMP is one example of future refinements aiming to glean more diagnostic info from head impulses.

Another area of development is data analysis and integration. Efforts are underway to improve VOR gain calculation algorithms to reduce artifact influence (e.g. excluding the first few milliseconds to avoid impulsive transients or using regression methods). The goal is to enhance reliability so that small changes in gain (for example, in serial monitoring of a patient) can be confidently ascribed to real vestibular improvement or decline rather than noise. Additionally, incorporating catch-up saccade analysis quantitatively (their frequency, latency, and amplitude) can increase sensitivity. Research shows that including parameters like covert saccade latency can improve agreement between vHIT and rotary chair results. Machine learning approaches are even being explored to automatically classify vHIT traces into diagnostic categories based on the full waveform.

In terms of clinical use, one can expect vHIT to become more widespread in primary care or general neurology/ENT settings. As portable, lower-cost devices come on the market, general practitioners may use a simplified vHIT for vestibular screening, much like ECGs are used for cardiac screening, referring to specialists when abnormal. Also, vHIT might play a role in vestibular rehabilitation feedback – for example, using the device to provide real-time feedback to patients training gaze stability exercises, although this is still experimental.

Anatomical and physiological insights gleaned from vHIT are also shaping future research. The test has revealed new clinical patterns: for instance, a syndrome of selective anterior canal hypofunction or “hidden” bilateral hypofunction that only manifests at high frequencies. These findings prompt investigations into whether certain diseases (like early ototoxic injury or microvascular vestibulopathy) preferentially affect type I hair cells (high-frequency) versus type II (low-frequency). Indeed, a proposed explanation for vHIT/caloric discrepancies is the differential susceptibility of receptor cell types – type II hair cells (low-frequency responders) may

be affected sooner in gradual vestibulopathies, whereas type I cells (high-frequency) are spared until later. Understanding these differences could lead to new diagnostic criteria and targeted treatments before complete loss occurs.

Finally, we anticipate greater integration of vHIT with other modalities. For example, simultaneous video head impulse and vestibular-evoked myogenic potential (VEMP) testing can assess all canals and otoliths in one session to localize labyrinthine lesions (three canals via vHIT, two otolith organs via VEMPs). Additionally, combining vHIT with functional imaging or electrophysiology might illuminate central processing of vestibular impulses. Although speculative, one can envision future "vestibular labs" where vHIT is one component of a multimodal assessment including ocular motor testing, dynamic visual acuity, and balance platform testing, all integrated for a comprehensive profile of a patient's vestibular function.

In conclusion, vHIT will continue to advance in accuracy and scope. It represents a shift toward more physiologic, frequency-specific testing of the vestibular system and away from solely relying on the century-old caloric test. Its ongoing development – from new protocols like SHIMP to enhanced analysis – will further solidify vHIT as a cornerstone of vestibular diagnostics for years to come.

Other Relevant Considerations

It is important to maintain consistent terminology when discussing vHIT findings. An abnormal vHIT is often described as showing a "canal paresis" or "vestibular hypofunction," but to avoid confusion with caloric "canal paresis" (which refers to low-frequency loss), some authors specify "high-frequency canal paresis." Also, while vHIT provides evidence of dysfunction, it does not identify the underlying pathology – e.g. an abnormal vHIT could be due to vestibular neuritis, ototoxic damage, surgical labyrinthectomy, etc., and clinical context is required to pinpoint the cause. Anatomical relevance is clear: each canal's function can be separately evaluated. Clinicians should recall the innervation (for example, horizontal and anterior canals share the superior vestibular nerve, posterior canal the inferior nerve) – so a pattern of combined horizontal+anterior loss implicates the superior branch, whereas isolated posterior canal loss implicates the inferior branch. These nuances make vHIT a powerful tool for localization of vestibular lesions within the labyrinth or vestibular nerve.

Cost and accessibility are practical considerations. A vHIT device (high-speed camera goggles and software) is a moderate one-time investment, generally less expensive than a full rotary chair system and comparable to a caloric irrigator with video-oculography setup. The ongoing costs are minimal, as it does not require expendable supplies (unlike calorics which use water or air and disposables). Many private vestibular clinics and audiology centres have found it feasible to acquire vHIT equipment, and its portability even allows use at the bedside or in emergency departments. The learning curve for examiners should be acknowledged: proper training is necessary to produce reliable results. However, once mastered, the test is quick and convenient, with immediate results that can be shown to patients.

It should be noted that vHIT does not assess everything – otolith function (utricle and saccule) still requires other tests (VEMP, off-vertical axis rotation, etc.), and dynamic visual acuity tests complement vHIT by measuring functional impact of bilateral vestibulopathy. Also, patient factors like age need consideration: older individuals may have slightly lower gains or more frequent

covert saccades even without gross pathology. Age-related VOR changes are an area of ongoing research, and normative vHIT databases stratified by age are being developed.

In summary, the vHIT is a true vestibular function test targeting the high-frequency aVOR, with distinctive strengths and some limitations. It is best interpreted as part of a comprehensive test battery. When combined with caloric testing (low-frequency VOR) and possibly rotational chair testing (mid-frequency VOR), vHIT helps complete the puzzle of vestibular function across the spectrum of head movement frequencies. The following sections will explore the caloric and rotational chair tests, which together with vHIT form the triad of major VOR assessments in clinical vestibular diagnosis.

Caloric Test

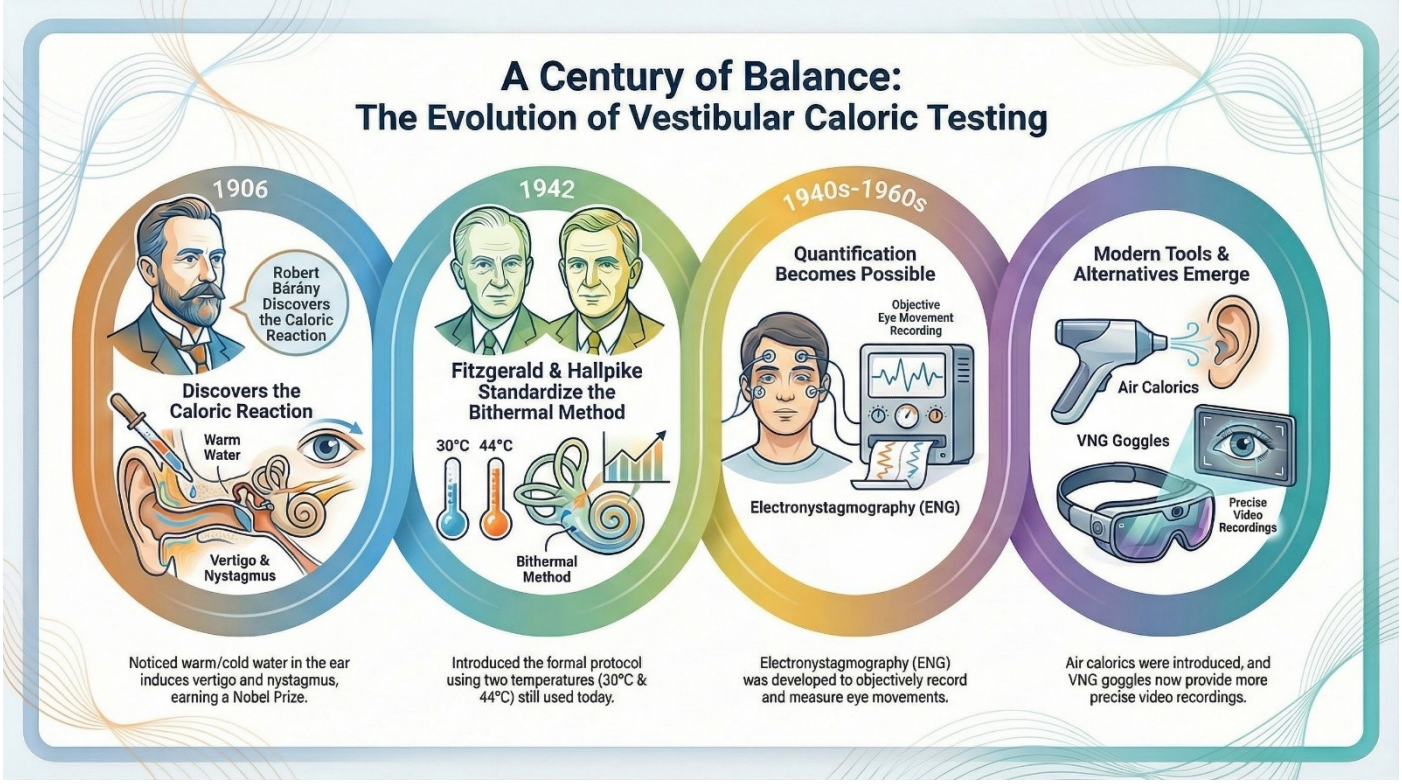
Introduction

Caloric testing is a classic vestibular examination technique that uses temperature stimuli in the ear canals to induce endolymphatic flow and provoke nystagmus, thereby assessing the function of each horizontal semicircular canal independently. First described over a century ago, the caloric test remains a cornerstone of the vestibular test battery. Unlike rotation or head impulse tests, caloric stimulation produces a very low-frequency vestibular input (**approximately 0.003 Hz),** well below the range of natural head movements. This aphysiologic low-frequency stimulus is nevertheless valuable because it allows isolation of each labyrinth's responsiveness. The caloric test primarily evaluates the lateral (horizontal) semicircular canal of each ear and, by extension, the superior vestibular nerve and central connections for that canal's pathway.

Clinically, caloric testing is often performed as part of videonystagmography (VNG) or electronystagmography (ENG) protocols in patients with dizziness. It has been historically regarded as the "gold standard" for detecting unilateral vestibular hypofunction due to its sensitivity to even partial canal paresis. In essence, a caloric exam involves irrigating each ear with warm and cold water or air and observing the resulting nystagmus (both its slow-phase eye velocity and its direction), then comparing the responses between ears. An asymmetric caloric response indicates a unilateral vestibular lesion, whereas bilaterally reduced responses indicate bilateral vestibulopathy. Caloric testing also engages central vestibular pathways: because the induced nystagmus can be modulated or suppressed by visual fixation and influenced by brainstem integrity, the test can yield insight into central vestibular function as well. Overall, despite being an "artificial" stimulus and somewhat uncomfortable for patients, the caloric test's ability to selectively test each ear's vestibular function at a low frequency makes it a unique and important diagnostic tool.

Historical Development and Major Contributors

The caloric reaction was first discovered by Robert Bárány [12] in the early 20th century. In 1906, Bárány noted that introducing warm or cold water into the external ear canal produces characteristic nystagmus and vertigo due to vestibular stimulation; this discovery of the caloric response was seminal and contributed to Bárány being awarded the Nobel Prize in 1914. Bárány's work laid the foundation for systematic vestibular testing. However, early caloric tests were not standardized in terms of temperature or technique and were performed qualitatively (observers simply noted presence or absence of nystagmus and patient sensations).



It was not until 1942 that Fitzgerald and Hallpike [13] formalized the caloric test protocol. Fitzgerald and Hallpike introduced the bithermal caloric testing method, using two temperatures (one warmer than body temperature, one cooler) delivered to each ear, with the patient supine and head elevated 30° to align the horizontal canal vertically. They determined that maximum nystagmic responses are obtained with water irrigations at approximately 7°C above and 7°C below body temperature (i.e. ~44°C warm and ~30°C cold water) for a fixed duration. This **Fitzgerald-Hallpike technique** remains the basis of caloric testing today. Over subsequent decades, researchers like **Jongkees** [14] refined the interpretation by introducing quantitative analysis of **canal paresis (labyrinthine preponderance) and directional preponderance**, complete with formulas to calculate percent differences between ears. The 1940s–1960s also saw the development of electronystagmography (using surface electrodes to record nystagmus), which enabled quantification of caloric-induced eye movements. Pioneers such as Claussen [15] and Dix-Hallpike [16] further expanded clinical use of calorics in diagnosing vestibular disorders. By the latter half of the 20th century, caloric testing had become ubiquitous in audiology and ENT clinics, often considered an essential test for any dizzy patient. In the 1980s and 1990s, air caloric irrigators were introduced as an alternative to water – addressing situations like eardrum perforations or patient discomfort with water – and standards for air caloric stimulus were developed (e.g. 50°C warm air, 24°C cool air for ~60 seconds). Throughout its history, caloric testing has remained largely unchanged in concept: its longevity is a testament to the importance of Fitzgerald and Hallpike's contribution in standardizing a reliable method. Today, caloric testing is typically performed with video-oculography (VNG goggles with infrared cameras) rather than electrodes, but the principles trace directly back to Bárány's discovery and Fitzgerald and Hallpike's protocol. These individuals are rightly regarded as giants in vestibular medicine for their role in developing the caloric exam.

Indications

Caloric testing is indicated whenever detailed assessment of unilateral vestibular function is needed, especially for low-frequency responsiveness of the horizontal semicircular canals. Classic indications include patients with vertigo or dizziness where a peripheral vestibular lesion is suspected. Caloric stimulation, by separately evaluating each ear, can localize a vestibular

deficit to one side – a critical capability if one is trying to confirm, for example, the side of a vestibular neuritis or the extent of vestibular loss in Menière's disease. In fact, caloric testing is often performed as part of the standard battery for evaluation of unilateral hearing loss with dizziness (such as in vestibular schwannoma workups), as caloric weakness commonly accompanies an acoustic neuroma on the affected side. It is also indicated in Menière's disease and other episodic vertigo disorders, both to document baseline function and to detect any progressive loss in the affected ear. Because caloric is very sensitive to partial vestibular deficits, it can uncover dysfunction when other tests (like bedside head impulse) appear normal – for instance, in patients with subtle imbalance or chronic dizziness of unclear origin, calorics can identify a previously uncompensated unilateral weakness.

Another key indication is differentiating central vs peripheral vertigo. Caloric testing stimulates the vestibular apparatus and the reflex arc through the brainstem; a completely normal caloric result in a patient with persistent vertigo might suggest that the issue is central (since the labyrinths are functioning symmetrically). Conversely, a significant asymmetry on calorics usually points to a peripheral (labyrinth or nerve) lesion. In practice, calorics are often done alongside ocular motor tests; patterns such as a unilateral caloric weakness strongly indicate a peripheral vestibular lesion, whereas certain caloric patterns like hyperactive responses or inability to suppress nystagmus may indicate central pathology.

Caloric testing is uniquely indicated for patients who cannot tolerate head movement-based tests. For example, if a patient has orthopedic or neurologic conditions limiting head or body motion (cervical spine issues, spinal cord injuries), caloric stimulation can still be performed since the patient remains supine and only the stimulus in the ear changes. As StatPearls notes [17], calorics can have better compliance in patients whose symptoms worsen with movement or who have limited cervical mobility. In these cases, vHIT or rotational tests might be infeasible, making caloric the primary means to test vestibular function.

Caloric testing is also part of the neurologic examination of comatose patients to assess brainstem integrity. This application, known as oculovestibular testing, involves instilling ice water into the ear canals to provoke eye deviation (the cold caloric reflex) as a test of the vestibulo-ocular reflex pathway through the brainstem. In unconscious patients, absence of caloric-induced eye movement can indicate brainstem dysfunction. Thus, in intensive care settings, calorics (often ice-water calorics) are indicated for brainstem testing in suspected brain death or coma, although this is a specialized usage distinct from the dizzy patient evaluation.

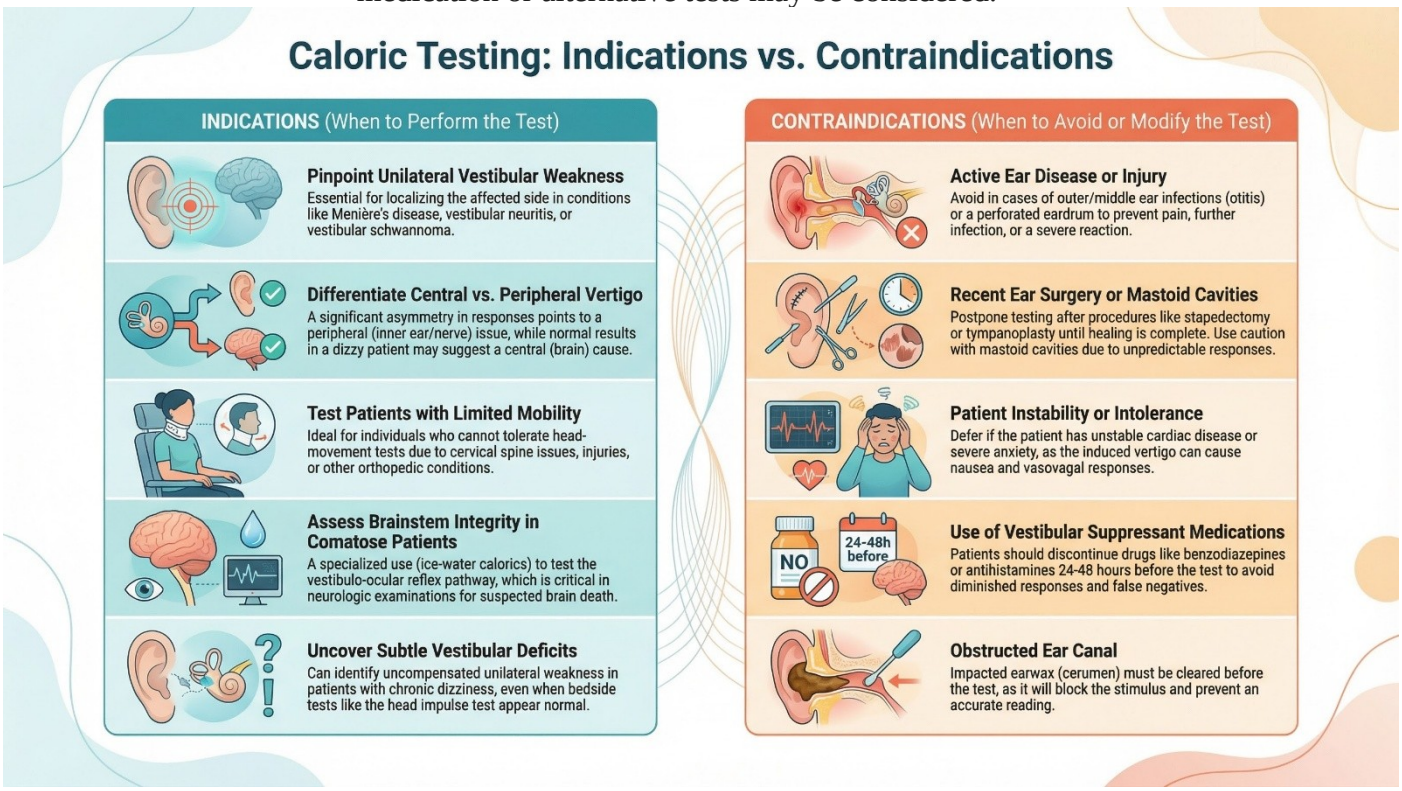
In summary, the main indications for caloric testing are: (1) evaluation of suspected unilateral vestibular weakness (vertigo, unilateral hearing loss with dizziness, episodic vertigo disorders), (2) as part of a comprehensive dizziness workup to complement higher-frequency tests (vHIT, rotational) especially if those are normal but suspicion remains, (3) patients who cannot undergo other vestibular tests due to mobility or tolerance issues, (4) differentiation of central vs peripheral causes of dizziness by assessing labyrinthine function symmetry, and (5) assessment of vestibulo-ocular reflex pathway integrity in comatose patients. It offers the unique advantage of isolating each ear's response, which is why it is still routinely indicated in vestibular clinics worldwide.

Contraindications

Caloric testing, while generally safe, has a few contraindications and precautions primarily related to the condition of the ears and patient safety. Active ear pathology is a major contraindication: an ongoing outer ear infection (otitis externa) or middle ear infection (otitis media) should preclude caloric stimulation, as introducing water or air could aggravate the infection or cause pain. Similarly, a perforated tympanic membrane is a contraindication for water calorics – irrigating through a perforation could introduce water into the middle ear and potentially the inner ear, risking infection or a severe caloric reaction. In cases of known tympanic membrane perforation or grommet (ventilation tube), water calorics should be avoided; if caloric testing is essential, air irrigation may be cautiously used or alternatively a “dry” thermal stimulation method (like closed-loop caloric or infrared calorics) can be employed. Some advanced systems use warm/cool air or even infrared radiation directed at the inner ear through the ear canal as an alternative caloric stimulus when eardrum integrity is an issue.

Patients who have had recent ear surgery (e.g. stapedectomy, tympanoplasty) should not undergo calorics until fully healed, to avoid disturbing surgical repairs or inducing vertigo that could disrupt post-op safety. Chronic mastoid cavities (from previous mastoidectomy surgery) are another caution: such ears may have altered thermal dynamics and often connect directly to the vestibule; many clinicians avoid irrigating mastoid bowl ears due to unpredictable (and sometimes intense) responses and the risk of caloric water entering the vestibule. If a mastoid cavity ear must be tested, air calorics or very cautious minimal water irrigation is preferred.

Another category of contraindication is patient factors: if a patient is in a medical condition where induced vertigo/nystagmus could be dangerous (for example, unstable cardiac disease or severe orthostatic hypotension), one might defer caloric testing. The test can cause a temporary autonomic response – patients often feel nausea, occasionally changes in blood pressure or heart rate can occur (usually mild). While rare, calorics have been reported to provoke vasovagal responses in very anxious individuals. Thus, extreme anxiety or an inability to handle vertigo is a relative contraindication; pre-medication or alternative tests may be considered.



Medications that depress vestibular responses (vestibular suppressants such as benzodiazepines, antihistamines, certain anticonvulsants) do not strictly contraindicate the test but will diminish caloric nystagmus. It is standard practice to ask patients to discontinue such drugs 24–48 hours before caloric testing. If they cannot (for example, due to seizure medications), the clinician should interpret reduced responses with caution, considering medication effect. Similarly, alcohol and sedatives should be avoided pre-test to prevent false negatives.

One practical contraindication is an occluded ear canal – impacted cerumen or debris can block the irrigant and prevent proper stimulation. Therefore, before caloric testing, the external canals should be examined and cleared of wax or obstructions (this is part of test preparation). If removal is not possible or safe, the test might need to be rescheduled or foregone.

In summary, caloric testing should not be performed in ears with acute infection, perforation, recent surgery, or other conditions that could cause harm with irrigation. Caution or alternative methods are warranted in those cases. Additionally, ensure patients are off vestibuloactive medications, and consider the patient's overall condition – if they are unable to tolerate the induced vertigo (e.g., severe anxiety or medical fragility), then alternative vestibular tests like vHIT or rotational chair (which are gentler) might be favoured. When caloric testing is indicated but contraindications are present, modifications such as using air stimulus or dry thermal stimuli can sometimes allow the test to proceed safely.

Methods (Clinical and Lab Technique)

The caloric test is typically performed as part of a VNG/ENG battery in a dedicated exam room.

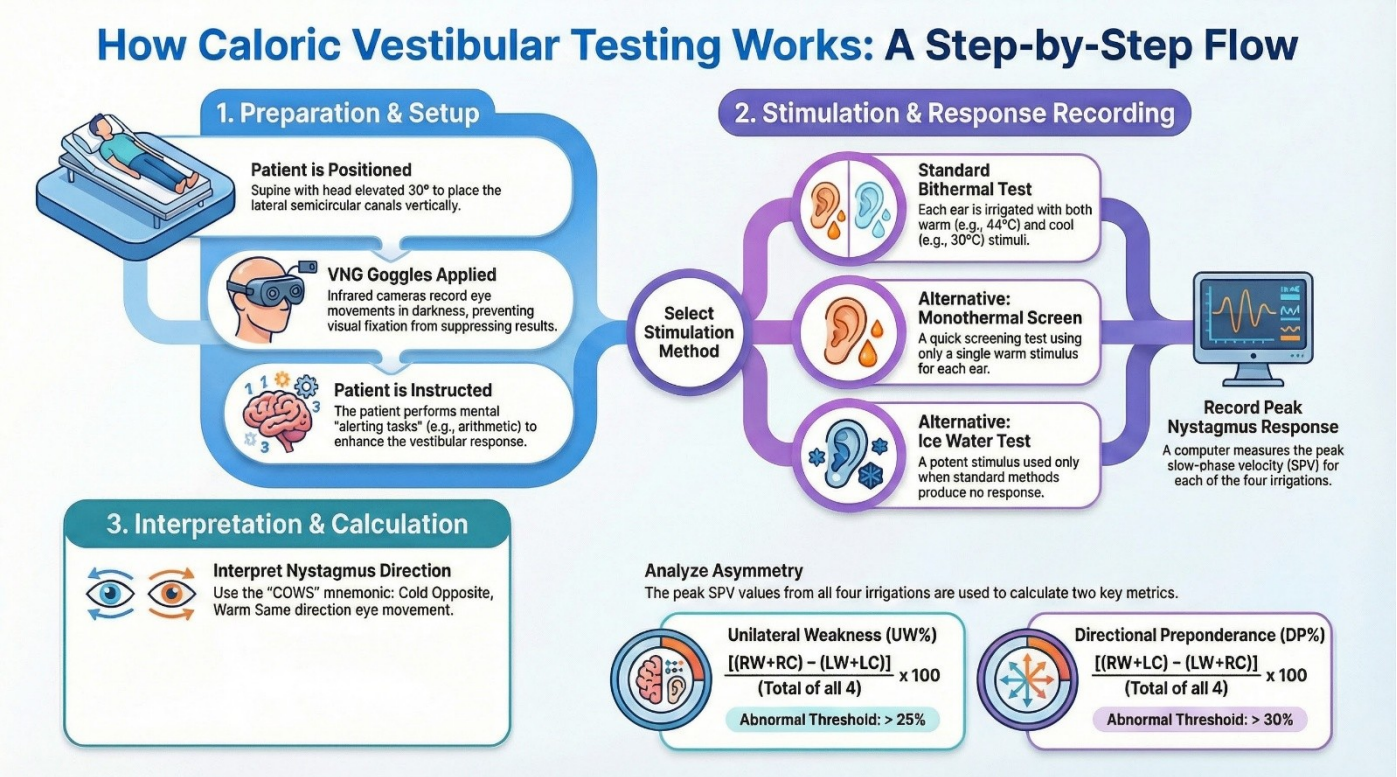
Preparation: The clinician first inspects the external auditory canals and tympanic membranes with an otoscope to ensure they are clear and intact. Any cerumen is removed. The patient is positioned supine on an examination table (or reclined in a chair) with their head elevated ~30° from horizontal (the neck is propped up), which places the lateral semicircular canals in a vertical orientation. This orientation is crucial; with the canal nearly vertical, the thermal convection currents induced by caloric irrigation will maximally deflect the cupula. During ENG in the past, electrodes would be placed around the eyes to record nystagmus. Nowadays, VNG goggles with infrared cameras are placed on the patient to record eye movements in darkness. The patient wears the goggles, and vision is occluded (most systems either have the patient close eyes and the camera views under infrared, or the goggles have eye covers) – vision must be eliminated to prevent fixation from suppressing the induced nystagmus. The patient is instructed about what to expect: the test will likely make them dizzy briefly, and they should mentally focus on a task (like arithmetic or recalling a list) during the stimulation to keep the mind alert, which paradoxically enhances the vestibular nystagmus by preventing central suppression.

Irrigation Protocol: In the standard bithermal caloric test, each ear is irrigated twice – once with a warm stimulus and once with a cold stimulus. For water calorics, the typical parameters are 250 mL of water at 44°C (warm) or 30°C (cool) irrigated into the ear canal over about 30 seconds. Many commercial caloric irrigators have a nozzle or irrigation syringe that delivers a controlled flow rate. The ear is irrigated and the water allowed to drain out into a catch basin (for open-loop systems). Nystagmus usually begins about 20–30 seconds after onset of irrigation, once the temperature gradient has penetrated to the lateral canal. The nystagmus intensity builds and typically peaks around 60–90 seconds after irrigation, then slowly subsides. During this time (around 90s to 2 minutes post-irrigation), the patient is asked to perform an alerting task (like

naming boys' names starting with A, B, C, etc., or doing mental calculations). This task prevents them from suppressing the nystagmus and also helps avoid vestibular habituation. After the response declines, a common practice is to check fixation suppression: the goggles are briefly lifted (or a fixation light is turned on) at ~2 minutes post-irrigation to see if the patient can suppress the nystagmus by looking at a target. Normally, visual fixation dramatically reduces caloric nystagmus; failure to do so suggests a central lesion (cerebellar or brainstem) affecting the vestibulo-ocular reflex suppression mechanism. The patient is then allowed a rest period (usually about 3–5 minutes or until nystagmus fully stops and dizziness fades). The procedure is then repeated for the other ear with the same temperature. Then the two opposite temperature irrigations are done in similar fashion, often alternating ears (some protocols do RLWR – Right Warm, Left Warm, Right Cool, Left Cool, etc., to allow symmetry in any order effects). Alternatively, some do all one ear then the other. The exact order is less important as long as adequate rest is given and the sequence is symmetrical.

For air caloric stimulators, the method is analogous: a calibrated air irrigator blows warm air (typically ~50°C) or cold air (~24°C) into the ear canal for a longer duration (about 60 seconds). Because air is a less efficient thermal conductor, the temperature differentials are usually set larger (about ±13°C from body temp) and the irrigation lasts longer to achieve sufficient endolymph temperature change. Air calorics also often use a fixed flow rate (e.g. 8 litres/min) delivered via a nozzle with a speculum tip. The rest of the procedure (monitoring nystagmus, alerting tasks, etc.) is the same as for water. Water generally yields more robust and less variable responses, but air is useful for patients who cannot have water (e.g. perforations) and can be more comfortable (no wetness).

In cases where standard bithermal testing cannot be done or yields no response, special caloric tests can be performed. The monothermal caloric test is sometimes used as a screening: only one temperature (usually warm) is given to both ears. If the responses are symmetric and strong, the test may be stopped, as this implies normal function in both ears. However, if monothermal results are equivocal or asymmetric, the full bithermal battery should be done because monothermal can miss some unilateral weaknesses (sensitivity can be as low as ~54% in some reports, meaning it may fail if used alone when an intermediate probability of pathology exists). Another is the ice water caloric: if no nystagmus is observed with standard stimuli (which could indicate a severe bilateral loss or dead ear), 2 mL of ice-cold water (0°C) can be injected into the ear canal as a last attempt to provoke any response. The patient lies with that ear upward, the ice water remains in the canal ~30 seconds, then the head is turned back to supine and any nystagmus is observed. Ice water caloric is a very intense stimulus and can detect even minimal residual function; if even ice fails to elicit nystagmus, the labyrinth is essentially areflexic on that side. (Ice water is also used in coma stimulation tests due to its potency.)



Throughout these procedures, careful observation or recording of the eyes is done. With VNG, the computer will measure the slow-phase velocity (SPV) of the caloric nystagmus. Typically, the peak SPV of nystagmus in each irrigation is measured as the key quantitative outcome. The direction of nystagmus follows the mnemonic **COWS ("Cold Opposite, Warm Same"),** meaning the fast phase of nystagmus beats to the opposite side of the stimulated ear with cold, and to the same side with warm irrigation (assuming the head is supine). For example, warm water in the right ear causes right-beating nystagmus; cold in right ear causes left-beating nystagmus. This is because warm irrigation mimics an excitatory stimulus (endolymph in horizontal canal rises toward ampulla, causing ampullopetal flow, simulating a head turn toward that ear), whereas cold does the opposite (ampullofugal flow, akin to head turn away from that ear). Clinicians verify that these directional effects occur as expected; paradoxical reactions (like a warm irrigation causing the opposite direction nystagmus) can indicate technical error or occasionally central pathology ("caloric inversion").

Safety and patient comfort: An emesis basin is kept handy, as some patients become nauseated. The examiner should be ready to immediately stop if a patient feels they might vomit or if the distress is too great; often by ceasing irrigation and allowing the patient to fixate, the symptoms abate. Communication is maintained with the patient, reassuring them that the dizziness will be short-lived. Between each stimulus, enough time (usually 3–5 minutes or more) is given for the nystagmus to subside and the patient to feel relatively normal again. The total test time for a full bithermal caloric (4 irrigations) is often about 20–30 minutes including rests.

Data and calculations: After obtaining responses from all irrigations, the main calculated metrics are **Unilateral Weakness (UW) and Directional Preponderance (DP).** The Jongkees formula [14] is commonly used: **for canal paresis/UW% = ((Total warm + cold response of one ear) - (Total of opposite ear)) / (Sum of all four responses) × 100%.** A result above a certain threshold (**often >20–25%)** indicates a significant unilateral weakness on the side of the

smaller response. Directional preponderance is calculated as ((Right-beating total) - (Left-beating total)) / (Sum of all four) × 100%, representing if nystagmus beats more strongly in one direction than the other. **DP values beyond ~30%** are typically considered abnormal, though labs vary. Modern VNG software does these calculations automatically and will flag abnormal asymmetries.

In summary, the caloric test method involves precise, controlled thermal stimulation of each ear in turn, rigorous observation/recording of induced nystagmus, and quantitative comparison of responses. It requires patient cooperation (to endure the dizziness and perform mental tasks) and careful technique (ensuring stimulus actually reaches the tympanic membrane and is the correct temperature). When done properly, it yields reproducible measures of vestibular function in each ear separately.

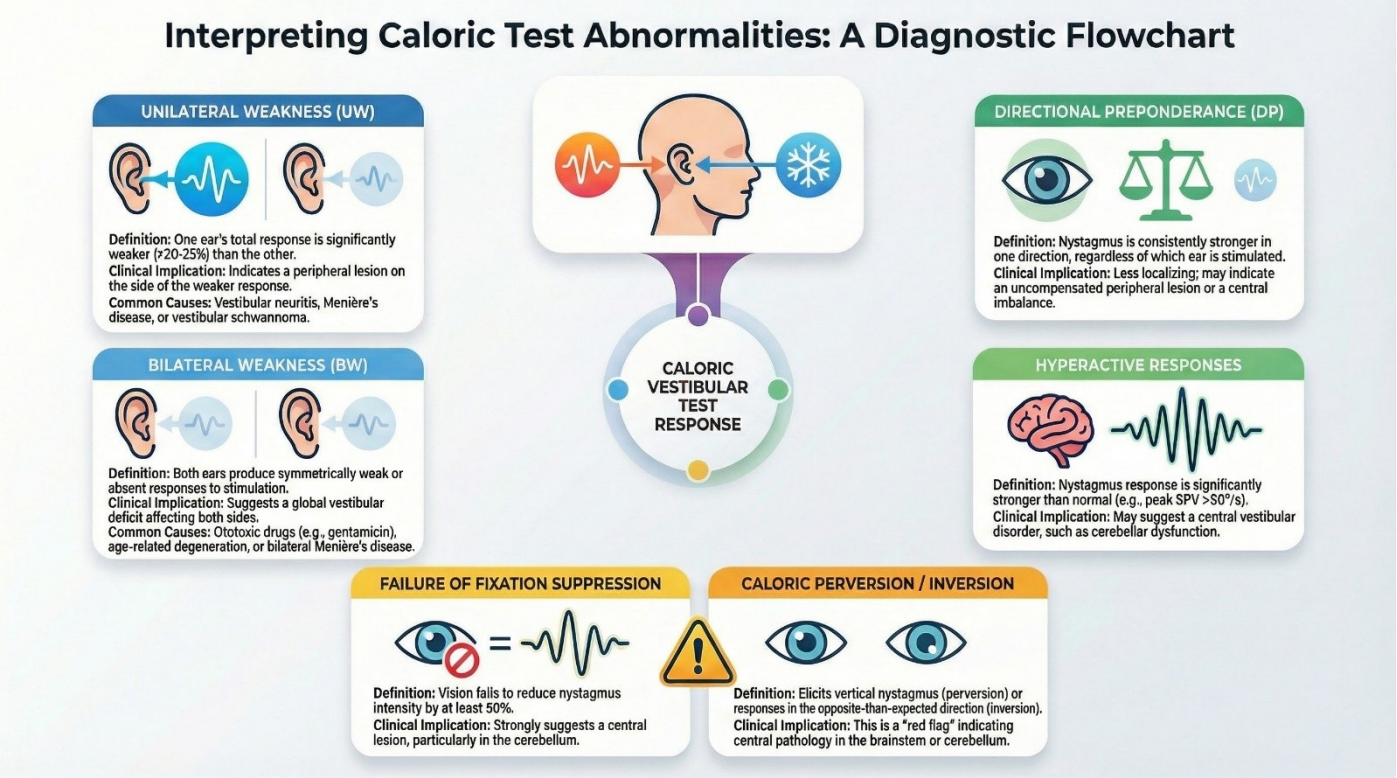
Interpretation of Results

Interpreting caloric results centres on identifying asymmetries and assessing overall responsiveness, in light of normative values and considering patient factors.

Normal Caloric Response: A normal exam is characterized by reasonably symmetric nystagmus responses between the two ears and appropriate nystagmus direction for each irrigation (COWS). Typical healthy individuals generate peak slow-phase velocities (SPVs) that might range, say, 15–30°/s for each irrigation (this can vary greatly with stimulus intensity and age). Crucially, the difference between the left ear total response (warm + cold SPV) and the right ear total response is small – usually <20% difference is considered within normal limits. Directional preponderance is also normally minimal (again <~25–30%). Essentially, both labyrinths should be roughly equal in excitability. Additionally, the presence of robust nystagmus with both warm and cold stimuli indicates intact bilateral vestibular function. When fixation is introduced, a normal subject will markedly suppress the nystagmus (>50% reduction in SPV) indicating proper central integration (cerebellar function). Normal caloric results in a patient with dizziness would suggest that the horizontal canal function is intact and symmetric, pointing either to a non-labyrinthine cause of symptoms or possibly involvement of structures not tested by caloric (vertical canals or otoliths).

Unilateral Weakness (Labyrinthine Paresis): This is the most clinically significant caloric finding. A **unilateral weakness (UW)** means one ear's responses are significantly weaker than the other's. In quantitative terms, if one labyrinth's total response is >20-25% lower than the others, it is considered a canal paresis on that side. For instance, if the right ear warm + cold yields 50°/s total and the left ear warm + cold yields 20°/s, the left has a ~43% weakness (since (50-20)/(50+20)=0.5*100%). This indicates a peripheral vestibular lesion on the left side. Clinically, a UW corresponds to reduced function of the horizontal canal or its afferent pathways on that side, commonly due to vestibular neuritis, labyrinthitis, unilateral Menière's disease (during or after episodes), vestibular schwannoma, or any insult affecting the labyrinth/nerve. The side of the weaker response is the side of the lesion (assuming no technical errors). A unilateral caloric weakness confirms the diagnosis of a peripheral vestibulopathy and localizes it. Often this finding correlates with the patient's history of vertigo toward the opposite side (e.g., a right UW typically manifests as past spontaneous nystagmus beating to the left, etc.). The magnitude of UW can be roughly graded: e.g., 25–40% mild paresis, 40–70% moderate, >70% severe. A 100% unilateral weakness (no measurable response from one ear) is caloric areflexia on that side – essentially indicating a dead labyrinth or nerve. The clinical term "dead ear"

sometimes refers to no auditory and no vestibular function on a side, and caloric areflexia would support that.



Directional Preponderance (DP): This measures whether the nystagmus tends to be stronger in one direction (regardless of which ear is stimulated). For example, if all four irrigations produce stronger right-beating nystagmus than left-beating, there is a directional preponderance to the right. DP can be caused by a pre-existing nystagmus at baseline (if the patient had, say, a right-beating spontaneous nystagmus, it will add to right-beating calorics and subtract from left-beating calorics). It can also result from a central vestibular tone imbalance. Normal DP values vary by lab but often up to 25–30% are accepted. A significant DP (e.g., 40% rightward) means that when you irrigate either ear, the response is biased in one direction. By itself, DP is less localizing: it might indicate an uncompensated peripheral lesion that still has a tonic bias (hence spontaneous nystagmus), or a central lesion in the vestibular nuclei commissural pathways. Many clinicians interpret DP alongside other findings: for instance, if there is a UW, any DP is usually attributed to that; but if there's no UW yet a strong DP, it could mean the patient had a recent unilateral insult and still has spontaneous nystagmus, or possibly a central process. For example, a patient in acute phase of right vestibular neuritis might show both a left UW (right weakness) and a right-beating DP (because they have a spontaneous right-beating nystagmus that adds to warm-left/cold-right). As the patient compensates, the DP might disappear while the UW remains. So DP is dynamic and less specific. Some consider DP as indicating the presence of a bias (static lesion) whereas UW indicates a loss (dynamic impairment).

Bilateral Vestibular Reduction: If both ears produce weak caloric responses (yet somewhat symmetric), this suggests bilateral vestibular hypofunction. Quantitatively, each ear's total may be far below normal ranges – many labs **consider bilateral weakness if the average SPV of each side's warm or cold is under a certain threshold (like <6°/s peak or sum of all four <20°/s, etc.).** In such cases, the calculated UW might be small (because both sides are equally reduced), but the absolute responses are low. This pattern can occur in bilateral vestibulopathies

(e.g. due to ototoxic drugs like gentamicin, bilateral age-related degeneration, or bilateral Ménière's in advanced stages). The key is noticing that even warm irrigations, which normally provoke strong nystagmus, are abnormally weak. Patients with bilateral loss often have absent or very slight nystagmus to all calorics and might report minimal vertigo during the test (since neither ear is functioning well). Documenting bilateral caloric loss is important, as it confirms a global vestibular deficit and correlates with symptoms like oscillopsia. However, caloric is a very low-frequency stimulus; sometimes, patients with bilateral loss at high frequencies (detected by vHIT) can still have some caloric response if low-frequency function remains via velocity storage. But in general, significantly reduced calorics bilaterally indicates a major bilateral vestibular problem. It is also noteworthy that bilateral caloric areflexia can be a false finding if the patient was not alert (fell asleep or was on sedatives) – this emphasizes maintaining alertness during the test.

Hyperactive Responses: Occasionally, caloric responses are much stronger than normal. Some define hyperactivity if the SPV exceeds a certain high threshold (e.g. **>50°/s** with standard irrigation) or if the sum of all responses is extremely high. Hyperactive calorics can be due to central vestibular disorder (lack of central damping). For instance, **cerebellar dysfunction (especially nodulus or uvula lesions)** can result in augmented and prolonged caloric nystagmus because the normal velocity storage damping is impaired. Another cause can simply be **anxiety or hyperventilation** during the test; an anxious patient might have an exaggerated response (one study noted ~10% [18] of subjects with "hyperactive" calorics had no pathology, attributing it to anxiety). Thus, mild hyperactivity may not be clinically significant if symmetric. If markedly hyperactive and especially if fixation does not suppress well, a central pathology should be considered.

Failure of Fixation Suppression: During caloric nystagmus, if allowing the patient to fixate (open eyes on a target) does not reduce the nystagmus substantially, this suggests a central lesion (likely in cerebellar flocculus/para-flocculus or brainstem pathways that enable VOR cancellation). Normally, fixation should reduce caloric nystagmus by >50%. An impaired fixation suppression (sometimes called abnormal Vestibular Ocular Reflex Suppression, VORS) often points to cerebellar disease even if peripheral vestibular function is normal or also impaired.

Caloric Perversion or Inversion: Rarely, caloric stimulation can produce vertical or perverted nystagmus (e.g., upbeat or torsional rather than horizontal). This is not normal and usually indicates central pathology in the brainstem or cerebellum. Caloric inversion refers to all responses beating the opposite direction of expected (e.g. warm producing contralateral nystagmus), and caloric perversion refers to vertical nystagmus elicited by caloric stimulus. These patterns are red flags for central involvement of vestibular pathways (such as a lesion in the vestibular nuclei or interconnecting tracts).

In practice, the most common interpretation issue is simply confirming a unilateral vestibular lesion. For example, an ENG report might say "25% reduced vestibular response on the right (right canal paresis)" which is taken as evidence of a right peripheral vestibular weakness. This correlates with, say, a patient's history of right-sided vestibular neuritis. Another scenario: a patient with episodic vertigo and fluctuating hearing suspected for Menière's may have an inter-ictal caloric test that shows 30% weakness on the affected ear, supporting the diagnosis of unilateral hydrops-related vestibular loss. If calorics are normal in a dizzy patient, and especially if vHIT is also normal, it suggests looking for non-peripheral causes like central disorders or even migraine.

Importantly, one must integrate the caloric results with the clinical picture. A unilateral caloric weakness has localizing value – it tells us which side likely has the lesion – but not diagnostic aetiology by itself. Additional tests (hearing, MRI, etc.) pinpoint why that weakness exists. On the other hand, a bilateral caloric weakness dramatically narrows differential diagnosis to bilateral vestibulopathy causes (e.g. ototoxic drugs, bilateral inner ear disease, bilateral neuropathy like CANVAS [19]).

Normal caloric responses in a symptomatic patient prompt reconsideration of diagnoses (could this be migraine, TIA, anxiety, etc., since labyrinths seem fine). However, as noted, normal caloric does not entirely exclude vestibular disorder – e.g., a superior canal dehiscence or selective utricular disorder could cause dizziness with normal calorics. But by and large, calorics being normal indicates no significant horizontal canal paresis.

In summary, interpretation revolves around: Is there a unilateral weakness? If yes, which side and how severe? Is there a directional preponderance? If yes, could it be due to a still-compensating lesion or a central bias? Are responses present bilaterally? If absent or diminished in both, think bilateral loss. And are there central signs (hyperactive, poor suppression, unusual nystagmus direction)? These elements together allow the clinician to conclude, for example: “The caloric test shows a significant left vestibular paresis with otherwise normal findings, consistent with a left peripheral vestibular lesion,” or “Calorics are normal bilaterally, making peripheral vestibular loss unlikely; consider central causes for the patient’s symptoms.”

Abnormalities and Diagnostic Patterns

Specific vestibular disorders often yield characteristic caloric test patterns:

Vestibular Neuritis: This typically results in a unilateral caloric weakness on the affected side (often >50% reduced response). For example, acute right vestibular neuritis will show a marked reduction or absence of nystagmus on right ear irrigation (both warm and cold) compared to the left. Directional preponderance is usually present acutely (with nystagmus beating toward the healthy ear predominating). Over time, the directional preponderance may resolve as spontaneous nystagmus subsides, but the unilateral weakness can persist chronically for months or years despite central compensation. Caloric testing is very sensitive to this; even patients who compensate and have minimal symptoms may still show, say, a 40% weakness long after an acute neuritis. Thus, calorics are often used to confirm the diagnosis of a past vestibular neuritis and gauge residual deficit.

Labyrinthitis: If the infection or insult involves the entire labyrinth (both auditory and vestibular end-organs), caloric testing will similarly show a unilateral weakness (assuming one side is affected). Because labyrinthitis often causes hearing loss too, the finding of a unilateral caloric paresis plus ipsilateral sensorineural hearing loss strongly suggests a peripheral lesion (like bacterial labyrinthitis or herpes zoster oticus, etc.) affecting that ear. In suppurative labyrinthitis, the affected ear might be essentially areflexic (100% weakness).

Ménière’s Disease: Interictally (between attacks), Ménière’s can show normal or mild caloric reductions; it often produces **fluctuating caloric responses**. In early stages, caloric might be normal or only a slight weakness. After repeated attacks or in later-stage disease, a unilateral

weakness on the affected side is common, reflecting cumulative hair cell loss. It might be moderate (20–50%). Notably, as mentioned earlier, **Ménière's can cause a disproportionate loss of low-frequency function** – so caloric could be abnormal while vHIT remains normal early on. During an acute Ménière's attack, if one attempted caloric (impractical due to vertigo), the affected ear might paradoxically show a reduced response or even a temporary direction preponderance as the hydropic ear's function fluctuates. Over the long term, a significant caloric weakness in a Ménière's ear often portends a "burned out" stage where vertigo attacks subside but hearing and vestibular function are chronically impaired. Some clinicians use serial caloric testing in Ménière's to monitor progression or to decide on ablative treatment (e.g., if one ear's caloric is already very weak, the patient might tolerate gentamicin ablation better).

Vestibular Schwannoma (Acoustic Neuroma): These benign tumours on the vestibular nerve usually cause a unilateral caloric weakness in a majority of cases (the larger the tumour, the more likely and more severe the weakness). This is due to compression and gradual loss of vestibular nerve fibres. Often, an acoustic neuroma patient will have an asymmetry on calorics but might not report vertigo (due to slow compensation). A significant caloric weakness in a patient with unilateral hearing loss raises suspicion for a vestibular schwannoma if not already diagnosed. Caloric testing has also been used post-operatively to assess function of the remaining ear (important in bilateral tumours like NF2). In summary, calorics are abnormal (UW) in about 85% of vestibular schwannomas by some reports, making it a useful adjunct to audiometry and imaging.

Bilateral Vestibulopathy: Causes include ototoxic drugs (e.g., gentamicin), bilateral sequential vestibular neuritis, autoimmune inner ear disease, idiopathic degenerative conditions, or central aetiologies like CANVAS [19]. Caloric results show bilateral reduced or absent responses. If both ears produce essentially no nystagmus even to warm water, that is bilateral vestibular failure (areflexia). Some patients have partial bilateral loss: e.g., all responses present but small (**say peak SPVs 5–7°/s on each irrigation).** The term bilateral hypofunction would be used. In ototoxicity, for instance, one might see initially partial reductions that progress to total loss. Documenting a bilateral caloric loss is important for confirming the diagnosis and severity. However, remember caloric only tests horizontal canals – in some rare cases a patient could have specific selective losses (like bilateral superior canal loss sparing horizontal canals – rare but reported in some autoimmune inner ear diseases); caloric might be normal in such an unusual scenario, whereas vertical canal tests (vHIT on verticals) would be abnormal. Those are exceptional cases; generally bilateral caloric weakness equates to bilateral vestibular dysfunction affecting horizontal canals.

Central Vestibular Disorders: Pure central lesions (e.g., a stroke in the vestibular nuclei) typically will not cause a unilateral caloric weakness, because the labyrinths and nerves are intact. Instead, caloric responses might be relatively symmetric but there could be abnormalities in how the nystagmus presents. For example, an internuclear ophthalmoplegia (MLF lesion) could cause dissociated eye responses to caloric stimulation (one eye doesn't adduct properly during nystagmus), which might be noted on video. A cerebellar lesion could cause the caloric nystagmus to have atypical timing or inability to suppress with fixation. Central disorders like migraine usually produce normal caloric results, though some studies note mild caloric asymmetries can occasionally be seen in vestibular migraine patients (perhaps due to baseline nystagmus during migraine episodes). Another scenario is a brainstem stroke: if it involves the vestibular nuclei or their commissural fibres, one might see either a reduced response on one side (mimicking a peripheral weakness) or a curious combination of signs like normal calorics

but abnormal rotational chair (due to failure of central integration). One central sign as mentioned is caloric inversion or perversion – if a patient exhibits vertical nystagmus on caloric, that's strongly indicative of a brainstem lesion (likely in areas like the medial longitudinal fasciculus or rostral vestibular nucleus which integrate vertical vestibular signals).

Persistent Postural-Perceptual Dizziness (PPPD) or other functional dizziness: These have normal caloric tests since vestibular function per se is intact; any abnormalities on caloric in such patients usually mean there is an underlying vestibular loss that perhaps triggered PPPD initially.

Superior Canal Dehiscence (SCD): Interestingly, SCD (a third-window pathology) can cause enhanced sound and pressure sensitivity, but caloric tests in SCD are usually normal or can even be slightly hyperactive because the dehiscence might alter fluid dynamics (though not always). Generally, SCD patients have normal caloric responses as the horizontal canal and nerve are intact, so caloric is not the test of choice for SCD (VEMP and CT imaging are).

Multiple Sclerosis: If MS plaques hit central vestibular pathways, calorics remain normal (since peripheral end organ works), except in rare MS lesions of the root entry zone of the VIII nerve, which could mimic a unilateral peripheral loss. But that's uncommon; MS usually spares calorics.

In summary, caloric test patterns are quite diagnostic: a unilateral weakness points to a peripheral lesion on that side (neuritis, labyrinthitis, etc.), a bilateral weakness points to bilateral involvement (ototoxicity, bilateral inner ear disease), and abnormal nystagmus characteristics or an isolated directional preponderance may indicate central issues or incomplete compensation. Integrating these findings with vHIT and rotational chair results yields a comprehensive picture: for instance, a patient with a 40% left caloric weakness but normal vHIT could be early Ménière's (as high-frequency preserved); a patient with bilateral caloric areflexia and bilaterally low vHIT gains obviously has bilateral vestibular loss. A patient with normal caloric and abnormal vHIT might have a selective high-frequency deficit (or a central vestibular problem). Each abnormal pattern guides the clinician toward the correct diagnosis and subsequent management.

Pitfalls and Limitations

Although caloric testing is a powerful diagnostic test, it comes with several limitations and potential pitfalls that can affect results and interpretation:

Physiological limitations: Calorics stimulate the lateral semicircular canals in a very low-frequency, non-physiologic manner. The induced endolymph convection current corresponds to an extremely slow head rotation (~0.003 Hz). Humans do not typically move their heads this slowly; thus, caloric responses depend heavily on the vestibular system's velocity storage mechanism to produce sustained nystagmus. This means caloric results may not reflect vestibular performance at higher, more functional frequencies. For instance, a patient could have a normal caloric test yet have deficits at higher frequencies detectable by vHIT – the two tests are not measuring the same frequency range. Therefore, one limitation is that calorics examine only a narrow slice of vestibular function. It ignores vertical canals and otoliths entirely, focusing only on horizontal canal function. A patient with an isolated posterior canal loss or utricular dysfunction would have normal calorics.

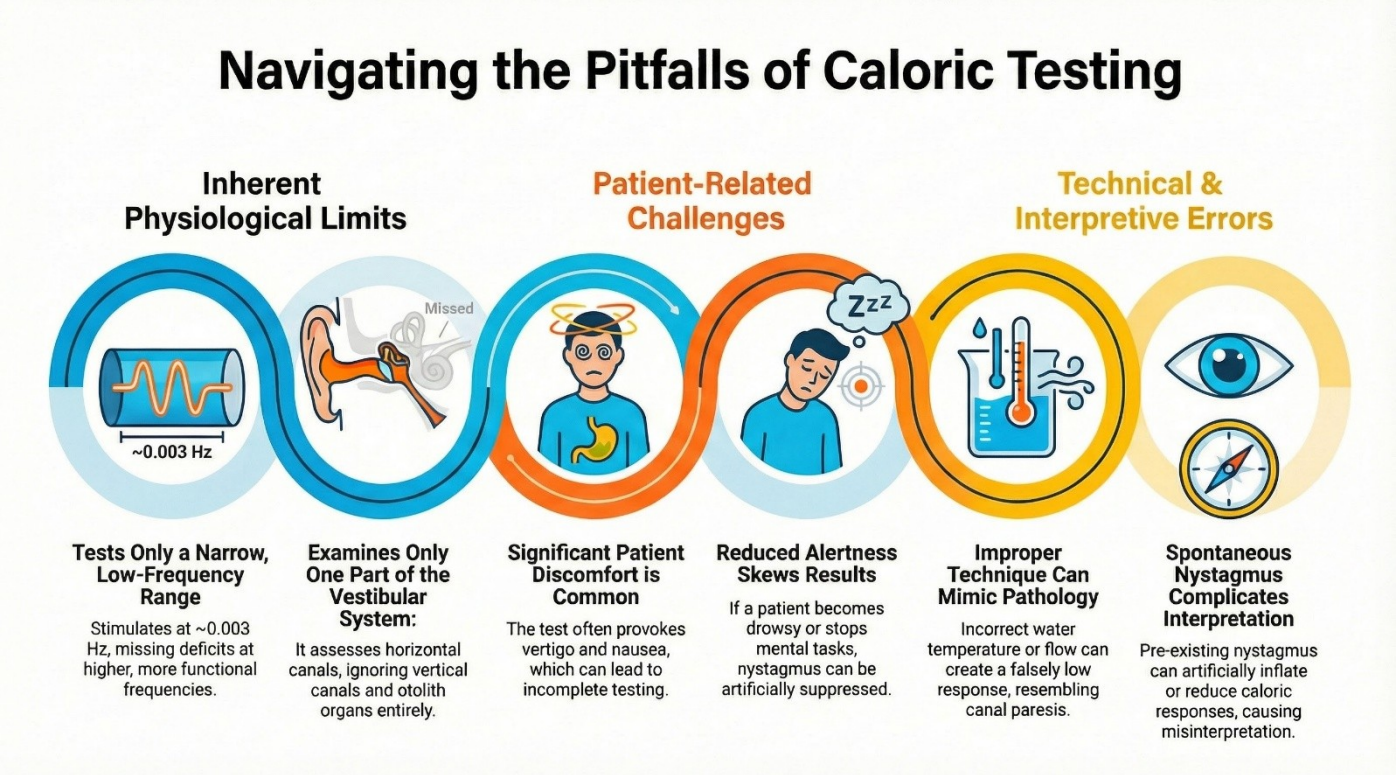
Patient tolerance and compliance: Caloric testing is notorious for provoking vertigo, nausea, sweating, and general discomfort. Some patients hyperventilate or panic during the test, which can alter results (e.g., hyperventilation reduces cerebral blood flow and can transiently suppress vestibular responses, or anxiety can increase variability). If a patient cannot tolerate the full set of irrigations and the test is aborted early, diagnostic information will be incomplete. Even subtle patient behaviours can interfere: for example, if the patient closes their eyes tightly or scratches (producing artifact on ENG electrodes), it could mask nystagmus or add noise. Also, patients must keep mentally alert; if they become too nauseated and stop engaging in the mental task, their nystagmus might diminish artificially due to central suppression. Ensuring the patient remains alert (sometimes challenging if they feel ill) is a necessary but difficult task – failure to do so is a common pitfall leading to underestimation of responses.

Technical pitfalls: Proper technique is crucial. If the water is not exactly at the correct temperature or the flow rate is off, the magnitude of stimulation can vary. An irrigation that doesn't fully contact the tympanic membrane (e.g., if the nozzle wasn't inserted correctly or water leaked out) might yield an abnormally low response, mimicking a canal paresis. Conversely, if warm water remains pooled in the ear or the room is warm, the ear may not cool back to baseline, affecting the next irrigation (thus usually one waits sufficiently or alternates ears to let things equilibrate). For air calorics, a common pitfall is insufficient stimulus – if the canal wall isn't heated/cooled adequately (perhaps due to a narrow canal or operator not aiming the air properly at the TM), the response could be falsely low. Additionally, air calorics have more variability; if one ear's anatomy yields better heat transfer than the other, it might introduce an artificial asymmetry. It's known that water calorics are more repeatable and have less inter-subject variability than air. So, switching between water and air protocols can make comparing absolute values tricky. Each lab tends to establish its own normative cutoffs; quoting literature thresholds (like 25%) assumes standardized conditions.

Spontaneous nystagmus and baseline drift: If a patient has a pre-existing nystagmus (from an acute vestibular lesion, say), it will sum with or subtract from caloric-induced nystagmus. For example, an acute left neuritis patient at rest has right-beating nystagmus. If you calorically stimulate the right ear with warm water (which also causes right-beating nystagmus), the measured response will be artificially higher (baseline plus induced). If you then do left ear warm (which causes left-beating nystagmus), the baseline right nystagmus will subtract, yielding a smaller observed response. This scenario produces a directional preponderance favouring the baseline nystagmus direction. If not recognized, one might misattribute that to a central finding, when in fact it's due to an uncompensated acute peripheral lesion. The timing of caloric testing is thus important: doing it in the first few days of an acute vestibular syndrome will invariably show both UW and DP due to spontaneous nystagmus. Some protocols recommend using the baseline nystagmus correction (subtracting the pre-irrigation nystagmus velocity from post-irrigation) or at least noting its presence. If the baseline nystagmus is large (>5°/s), interpretation of DP becomes unreliable.

Bell's phenomenon and eye closure artifacts: During ENG days, some patients would have an issue where upon eye closure (necessary for ENG), their eyes rolled up (Bell's phenomenon) and this could reduce measurable nystagmus, or they'd blink a lot. With VNG goggles, this is largely mitigated, but eye closure can still be an issue if goggles aren't totally dark or if patients' peek. Modern VNG systems keep eyes open in darkness, which helps.

Central compensation: In chronic unilateral vestibular losses, the central nervous system compensates for the asymmetry at rest, but caloric testing will still reveal the weakness. However, central compensation can reduce overt nystagmus by enhancing visual-vestibular interactions or promoting predictive mechanisms. If a patient has been well rehabilitated, they might subconsciously suppress some caloric-induced nystagmus by using cervico-ocular reflexes or mental strategies. It's important to keep them distracted (alert) to avoid this. On the flip side, patients with certain central lesions might exaggerate responses – e.g., a bilateral vestibular loss patient might show a small nystagmus which ordinarily would fatigue quickly, but if velocity storage is paradoxically prolonged due to a missing cerebellar clamp, they might maintain some nystagmus longer than expected.



Habituation: Repeated caloric irrigations can lead to reduced responses (the vestibular system habituates somewhat to the stimulus). If a second round of calorics is done immediately, the response can be blunted compared to the first. That's why one waits sufficient time or does alternating sides. But even alternating, by the fourth irrigation the patient often has a bit less response as they get used to the sensation. Labs try to mitigate this by sequence planning, but slight habituation could conceal a small asymmetry. It's also why monothermal screening (one irrigation each) can be valid if normal, but if done fully, the second irrigation on each side might be slightly weaker than the first just from adaptation (not pathology).

Environmental factors: The test environment should be controlled. Air drafts or differences in room temperature can influence an air caloric. Noise or other distractions can cause the patient to inadvertently fixate or become unalert.

Misinterpretation pitfalls: As mentioned, one must be careful not to over-interpret a directional preponderance without context. A DP in isolation doesn't tell you side of lesion; it might not even be peripheral. Over-reliance on caloric alone could also be a pitfall: e.g., diagnosing "normal

vestibular function" just because calorics are normal, when the patient might have bilateral high-frequency loss missed by caloric. That's why comprehensive testing is emphasized.

Also, a common pitfall is equating caloric weakness to "percent vestibular loss" in a linear way. A 50% caloric weakness doesn't necessarily mean the ear has exactly 50% loss of hair cells or fibres: it's a relative measure at low-frequency stimulus. So, it's more a functional measure than an anatomical one. Two patients with the same 40% UW might have very different clinical pictures depending on chronicity and compensation.

In summary, caloric testing's limitations include: it is time-consuming (compared to vHIT, for instance, which is done in minutes), provokes discomfort, and only tests a portion of vestibular function. It has numerous potential technical pitfalls, but with a skilled examiner, these can be minimized. Awareness of these limitations is crucial in ensuring that the caloric test is used and interpreted appropriately. Many modern vestibular evaluations incorporate calorics as one piece of the puzzle – acknowledging its sensitivity for unilateral loss but also its blind spots and patient tolerance issues. Clinicians must sometimes decide to skip or modify calorics (e.g. do monothermal or ice-water only) in frail patients to avoid excessive discomfort, balancing diagnostic yield against patient wellbeing.

Future Directions

Although caloric testing is a mature technique, there are ongoing efforts to improve and modernize it, as well as discussions about its future role given newer vestibular tests.

One area of development is alternative stimulation methods that could replace water/air irrigations. Research has been done on using **microwave or infrared thermal stimulation** delivered through the external canal to induce endolymph temperature changes without actually instilling any substance. For instance, near-infrared laser caloric stimulation has been experimented with: it involves directing an infrared beam at the bony canal, causing a slight warming that in theory induces a caloric effect. Early studies suggest this method can yield caloric nystagmus and would be especially useful in cases of perforated eardrums or when water cannot be used. If perfected, such techniques might make caloric testing less messy and more tolerable (no water rushing in/out) while still obtaining low-frequency data.

Another innovation is **closed-loop caloric irrigation systems**: rather than an open flow of water into and out of the ear, some devices circulate water in a caloric canal tip that sits in the ear (without water overflowing). This can precisely control temperature delivered to the ear canal and potentially reduce variability. Such systems have been used in research to get more repeatable results and even to do prolonged caloric stimulation for therapy (e.g., caloric vestibular stimulation for neurologic therapeutic trials).

There has also been discussion of automating caloric analysis and integrating it with other tests. Modern VNG software already computes canal paresis and DP, but future systems might use machine learning to interpret caloric results in context of vHIT/rotational results to give clinicians a synthesized report of vestibular function across frequencies. For example, an algorithm could combine a 30% caloric weakness with a borderline vHIT gain to estimate overall percentage of vestibular function loss in that ear in a more holistic manner, rather than treating them separately.

In terms of clinical practice evolution, one future direction is more judicious use of calorics. There is a trend toward performing vHIT first (as it is quick and well-tolerated) and using calorics more selectively – e.g., only if vHIT is normal but clinical suspicion remains, or to quantify a deficit if vHIT shows bilateral involvement. Some experts advocate for a "tasking caloric test" approach: do monothermal calorics initially and if it indicates symmetry, skip the rest (saving the patient discomfort). Indeed, studies show monothermal warm caloric screening [21] has high negative predictive value when properly done. We might see updated guidelines that encourage monothermal screening to improve patient tolerability and clinic efficiency, with full bithermal reserved for positive or unclear cases. This would effectively cut test time in half for many patients.

Another future consideration is how caloric testing fits into comprehensive vestibular care as new tests for otolith function (like VEMPs) and high-frequency canal tests (vHIT) are common. The caloric will likely remain relevant for certain scenarios (like unilateral vestibulopathy confirmation and bilateral assessment), but its use might decline in routine dizzy evaluations that are clearly central or benign positional in nature. On the other hand, calorics may find new life in therapeutic realms. There is emerging interest in using caloric vestibular stimulation for rehabilitation or even treatment of certain neurological conditions (e.g., there are experimental protocols of repeated caloric stimulation to improve gait in Parkinson's or to modulate migraine – based on the concept of vestibular neuromodulation). If such unconventional uses pan out, caloric devices might become therapeutic tools too.

From an anatomical and physiological research perspective, caloric tests in the future might incorporate more detailed measurements. For instance, 3D video-oculography can record not just horizontal but also torsional components of caloric nystagmus, which could give insight into the state of vertical canals via cross-coupling. Additionally, combining caloric with imaging like functional MRI is being explored to map which brain regions are activated during caloric vestibular stimulation – potentially useful for understanding spatial cognition and even psychiatric effects (caloric vestibular stimulation has interesting transient effects on body perception and mood in research settings).

Finally, the equipment for calorics may be refined. Current caloric irrigators are relatively simple (water baths or air heaters). Future devices might incorporate more precise temperature control with feedback (ensuring the ear canal reaches target temp), or small portable caloric stimulators for bedside use in hospitals (for coma exams, etc.). There's also a possibility of vestibular implants in the future – if vestibular prosthetics become common for bilateral loss, caloric testing would be one way to assess the implanted ear's residual function or effectiveness in low-frequency ranges.

In summary, while caloric testing is old, its future likely involves making it more patient-friendly (through alternative stimuli and streamlined protocols) and maintaining its role as a complementary test alongside newer technologies. Its unique ability to isolate ear function at low frequency means it will probably remain in our armamentarium, albeit used in more focused ways. The future may see calorics being done less routinely, but more strategically – for example, confirming mild losses that vHIT misses, evaluating candidates for vestibular implants, or research into brain responses. The fundamental principle discovered by Bárány – using temperature to probe the inner ear – will likely endure, adapted to modern requirements.

Other Relevant Considerations

When performing and interpreting caloric tests, a few additional points are noteworthy:

Standardization and Norms: Each clinic often establishes its own normative data for caloric responses, because factors like ambient temperature, irrigation technique, and even patient population (age distribution) can affect results. For example, older patients generally have weaker caloric nystagmus than younger patients due to age-related decline in vestibular hair cells and central processing. Thus, a 12°/s response might be normal in an 80-year-old but considered low in a 20-year-old. Clinicians must use age-adjusted normative ranges or at least interpret with knowledge of this. Similarly, different equipment (air vs water) have different baseline magnitudes – a 25% weakness threshold is mostly derived from water caloric norms. If using air calorics, some labs might consider a slightly higher threshold (because air responses can be more variable). The British Society of Audiology (BSA) [20] and other bodies have published recommended caloric test protocols and interpretation guidelines, which emphasize local calibration of normal values. So it's important to consider that what constitutes an "abnormal" caloric can vary slightly – e.g., some use 25% as significant UW, others 20%.

Ear-specific considerations: Middle ear status can alter caloric responses. Even without perforation, differences in middle ear volume (e.g., a mastoidectomy cavity or a very sclerotic middle ear) can affect heat conduction. A patient with a large mastoid bowl might have somewhat dampened or delayed caloric responses because the heat dissipates in the larger air space. Conversely, a tight, sclerotic middle ear might transmit temperature changes more directly. Also, otosclerosis has been reported to sometimes reduce caloric responses, presumably by insulating the labyrinth. Awareness of these conditions can explain unexpected caloric findings (like a slight bilateral reduction in a patient with otosclerosis).

Time since lesion: As touched on, the timing of when caloric testing is done relative to an insult can influence the results. In acute phases, you might see a greater difference (since central compensation hasn't kicked in at all), whereas months later the difference persists but the patient's symptoms are less. Some clinicians consider repeating calorics in certain scenarios – for instance, after a vestibular neuritis, a follow-up caloric 6–12 months later can document whether any peripheral recovery occurred (sometimes small improvements are seen if partial vestibular function returns). In most cases, the weakness remains, but interestingly central compensation does not "normalize" caloric asymmetry the way it can normalize head impulse or rotational asymmetry in part. That is because the velocity storage asymmetry from a unilateral loss tends to persist. This is a nuance: caloric testing often remains abnormal long after a compensated lesion; it is sensitive to the history of a lesion even when patient is well.

Relation to symptoms: Patients with chronic unilateral vestibulopathy may actually be asymptomatic at rest but still have a caloric weakness. Meanwhile, directional preponderance often correlates with presence of spontaneous nystagmus (hence acute symptoms). A subtle but important consideration: a pure unilateral weakness without directional preponderance usually indicates a compensated lesion (no baseline nystagmus, but weakness appears when stimulated). A unilateral weakness with a directional preponderance (especially beating toward the good ear) often indicates an uncompensated or recent lesion. This can be clinically useful: for example, a caloric result that shows 40% weakness and a big DP suggests the lesion is probably recent or ongoing (like an acute unilateral loss not yet compensated), whereas 40% weakness with no DP suggests the brain has compensated the resting tone but the dynamic loss remains.

Cross-checking with other tests: As a rule in vestibular testing, one correlates caloric outcomes with vHIT, rotational, VEMP, etc. Often, caloric and vHIT complement each other as high vs low frequency – disparate results can yield insight. For example, patients with superior vestibular neuritis (affecting horizontal and anterior canals) will have an abnormal horizontal vHIT and a caloric weakness on that side, both aligning. Patients with infranuclear lesions (like a specific horizontal canal plug or postsurgical loss of lateral canal function) similarly will show both abnormal vHIT and caloric on that side. But if vHIT is normal and caloric is abnormal, one thinks of frequency-dependent loss (as in early hydrops). If vHIT is abnormal and caloric normal, consider selective high-frequency loss (maybe central adaptation at low freq), or a central issue. Also, one cross-check is with rotational chair: rotational chair at 0.01 Hz should theoretically parallel caloric results. Indeed, if a rotational chair test (low-frequency SHA) is normal but caloric shows a big weakness, one must question if the caloric had a technical issue or if something like a middle ear problem dampened calorics. In many cases, rotational testing confirms a bilateral loss that calorics suggested or can quantify bilateral deficits when caloric just says “absent bilaterally.” It’s reassuring when multiple tests align, discrepancies often prompt re-testing or careful analysis.

Use in children: Caloric testing in very young children is difficult because they won’t tolerate the discomfort and need to be alert (and you cannot easily get a toddler to do mental tasks while dizzy). Therefore, calorics are rarely done below age ~5. In older children (school-age), calorics can be performed but require much coaching. Some paediatric vestibular clinics avoid calorics in favour of rotational chair testing which is better tolerated by kids. However, if inner ear dysfunction is suspected in one ear of a child (like vestibular loss associated with a cochlear implant or meningitis), calorics might be attempted to gauge any residual function. This is a consideration because more paediatric patients with hearing loss are getting evaluations for vestibular function nowadays (e.g., before cochlear implantation).

Vestibular suppressant medication influence: We mentioned withholding meds. To expand: benzodiazepines, barbiturates, some antiemetics (like promethazine) can depress the nystagmus response. Even caffeine and alcohol can alter responses (alcohol can induce a baseline nystagmus or change endolymph density transiently and produce a positional nystagmus – the “positional alcohol nystagmus” – which could confound a caloric). The test ideally is done med-free and sober. If a patient accidentally took a vestibular suppressant, results should be interpreted with caution or the test rescheduled if possible.

Compensation index concept: Some have proposed that the ratio of caloric to rotation response or caloric to vHIT might serve as an index of central compensation. For example, an uncompensated unilateral lesion might show large caloric asymmetry and similarly large low-frequency rotation asymmetry, but a partially compensated one might still show caloric asymmetry (peripheral deficit remains) but rotation asymmetry reduced (central compensation for that frequency). In practice, though, there is no simple numeric compensation index widely used; clinicians qualitatively judge compensation by presence of DP and by symptoms vs objective findings.

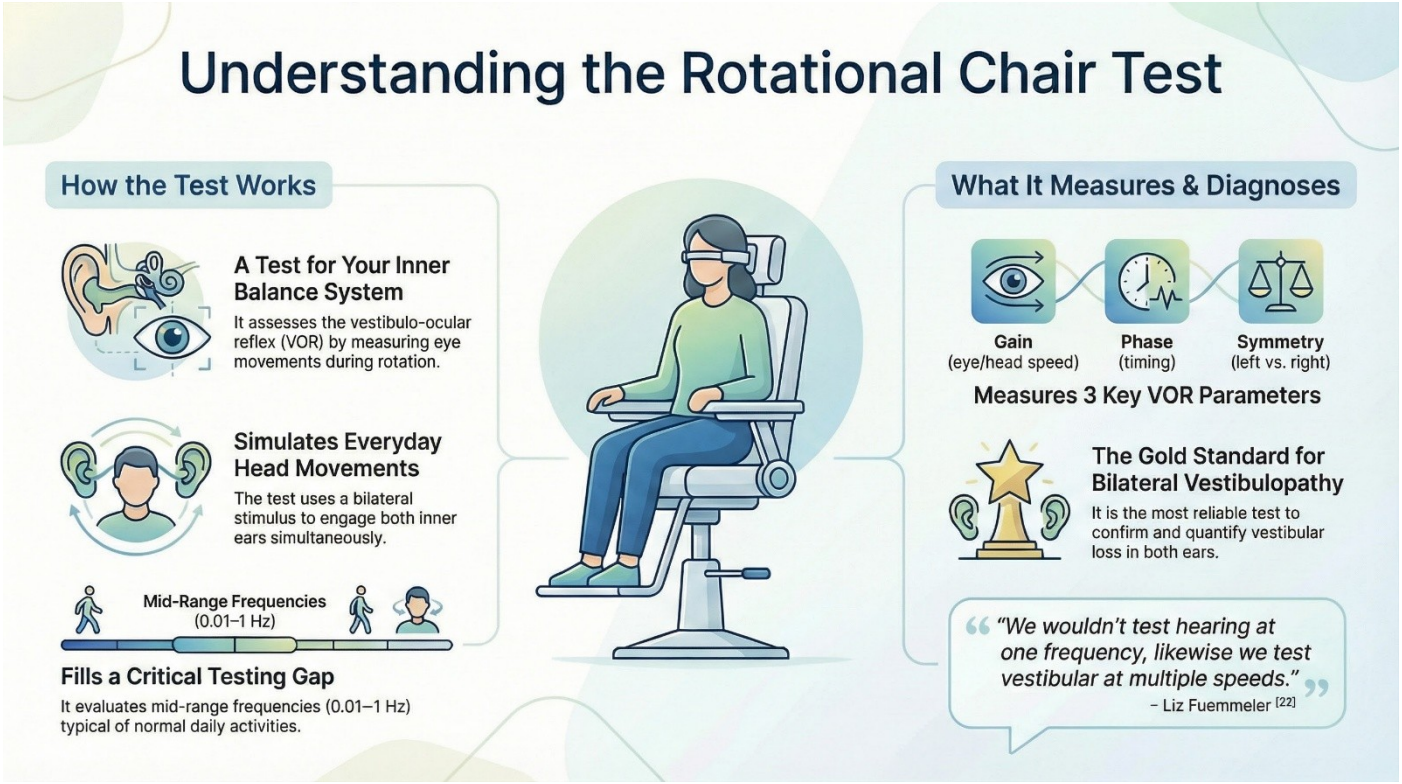
In conclusion, the caloric test is an established, nuanced piece of the vestibular test battery. It requires careful technique and thoughtful interpretation, integrating knowledge of vestibular anatomy, physiology, and compensatory mechanisms. When interpreted in context and

combined with other tests, caloric stimulation provides critical information about vestibular function that cannot be obtained otherwise. Despite its discomfort and limitations, it remains a key test, especially in diagnosing unilateral vestibular deficits and assessing bilateral vestibular loss, ensuring that the vestibular clinician can localize lesions and quantify the degree of impairment for accurate diagnosis and management.

Rotational Chair Test

Introduction

Rotational chair testing is a vestibular evaluation method that assesses the function of the vestibulo-ocular reflex (VOR) by subjecting the patient to controlled rotational movements and measuring the resulting eye responses. Unlike the head impulse or caloric tests, which stimulate one ear at a time or in highly specific frequency extremes, the rotational chair (often simply “rotary chair”) provides a physiologic, bilateral stimulus that engages both vestibular systems simultaneously across a range of mid-frequency rotations**. Everyday head** movements span frequencies roughly from **0.1 Hz up to 5–6 Hz.** The rotational chair test typically covers frequencies in the **0.01–1 Hz range,** bridging the gap between caloric (≈0.003 Hz) and head impulse (>1 Hz). This makes it particularly useful for evaluating the VOR in the frequencies of normal activities (like walking, turning the head, etc.). By analysing eye movement gain (eye/head velocity), phase (timing difference between eye and head motion), and symmetry (response differences when rotating left vs right), rotational testing can detect both unilateral and bilateral vestibular deficits, gauge the degree of central compensation, and provide a “global” sense of vestibular performance in a natural stimulus paradigm (physical rotation in darkness). It is considered the gold-standard test for diagnosing bilateral vestibulopathy, since it can confirm bilateral reductions in VOR gain and measure any remaining function. Additionally, rotational chair exams often include specialized subtests, such as velocity step testing (to measure how quickly the VOR decay – the time constant – which relates to central velocity storage) and optokinetic or visual-vestibular interaction tests (which assess central integration of visual and vestibular cues). Overall, the rotational chair is a comprehensive, quantitative test that complements caloric and vHIT by examining the vestibular system’s performance dynamically over a continuous range of stimuli. Clinically, it is frequently used in specialized vestibular centres, in research, and for cases where precise quantification of bilateral function or monitoring of vestibular status over time is needed. Liz Fuemmeler [22] highlights: we wouldn’t test hearing at one frequency, likewise we test vestibular at multiple speeds.



Historical Development and Major Contributors

Rotational testing of the vestibular system has a long history, essentially as old as vestibular research itself. The concept of using rotation to provoke nystagmus dates back to the 19th century, but it was Robert Bárány [23] in 1907 who first systematically employed a rotating chair for clinical vestibular evaluation. The classic Bárány chair was a simple manual swivel chair that allowed an examiner to spin the patient at a certain speed then stop them abruptly, observing the post-rotational nystagmus. Bárány's use of rotation, along with calorics, was pioneering – he recognized that a sudden stop from rotation causes nystagmus (e.g., if you spin someone and suddenly stop, the endolymph continues moving briefly, simulating a head turn in the opposite direction). This qualitative use of rotation became part of early neurologic exams. Over subsequent decades, especially mid-20th century, investigators aimed to quantify rotational responses. In 1935, J. R. Schäfer [24] introduced one of the first motor-driven rotation devices, and during the 1940s–50s, researchers like Fitzgerald and Hallpike [13] (who were also caloric pioneers) studied nystagmus decay after rotation to understand vestibular physiology. The development of electronystagmography (ENG) in the 1940s–60s by thinkers such as H. Kornhuber [25] allowed more precise measurement of eye movements during rotation.

However, a major impetus for advanced rotational testing came from the space and military programs in the 1960s–1970s. Organizations like NASA and air force labs needed to evaluate pilots' vestibular function and adapt astronauts to weightlessness; this drove the creation of more sophisticated computer-controlled rotary chairs. Pioneers like Dr. Viktor Jirák [26] and Dr. Herman Schilder [27] contributed to early rotary chair designs. By the late 1970s, commercially available rotary chairs (e.g., Contraves, and later Neurokinetics and Micromedical/Interacoustics Orion systems) were introduced, featuring precise velocity control and computerized analysis. These chairs could deliver sinusoidal harmonic acceleration (SHA) tests at defined frequencies and record eye movements via ENG. In the early 1980s, researchers such as T. Hain [28], C.

Herdman [29], and colleagues refined rotational testing protocols, establishing normative data and clinical utility for detecting bilateral vestibular loss and monitoring unilateral lesion compensation. Dr. Ian Curthoys [30] and others also contributed to understanding of high-frequency head rotations, although that overlapped with the eventual development of head impulse tests.

The modern rotational chair test battery emerged through contributions from many. Notably, Richard Black [31] and Michael Leopold [32] in the 1980s delineated standard frequencies (like 0.01 to 0.64 Hz SHA) and step test protocols. The concept of using different frequencies to characterize the VOR frequency response (gain and phase plot) was developed in analogy to audiograms. This multi-frequency concept became a cornerstone taught by leading vestibular audiologists like Susan Herdman [33] and Gary Jacobson [34]. The advancement of video-oculography (VOG) in the 2000s allowed even more precise eye tracking during rotation (replacing or augmenting ENG electrodes).

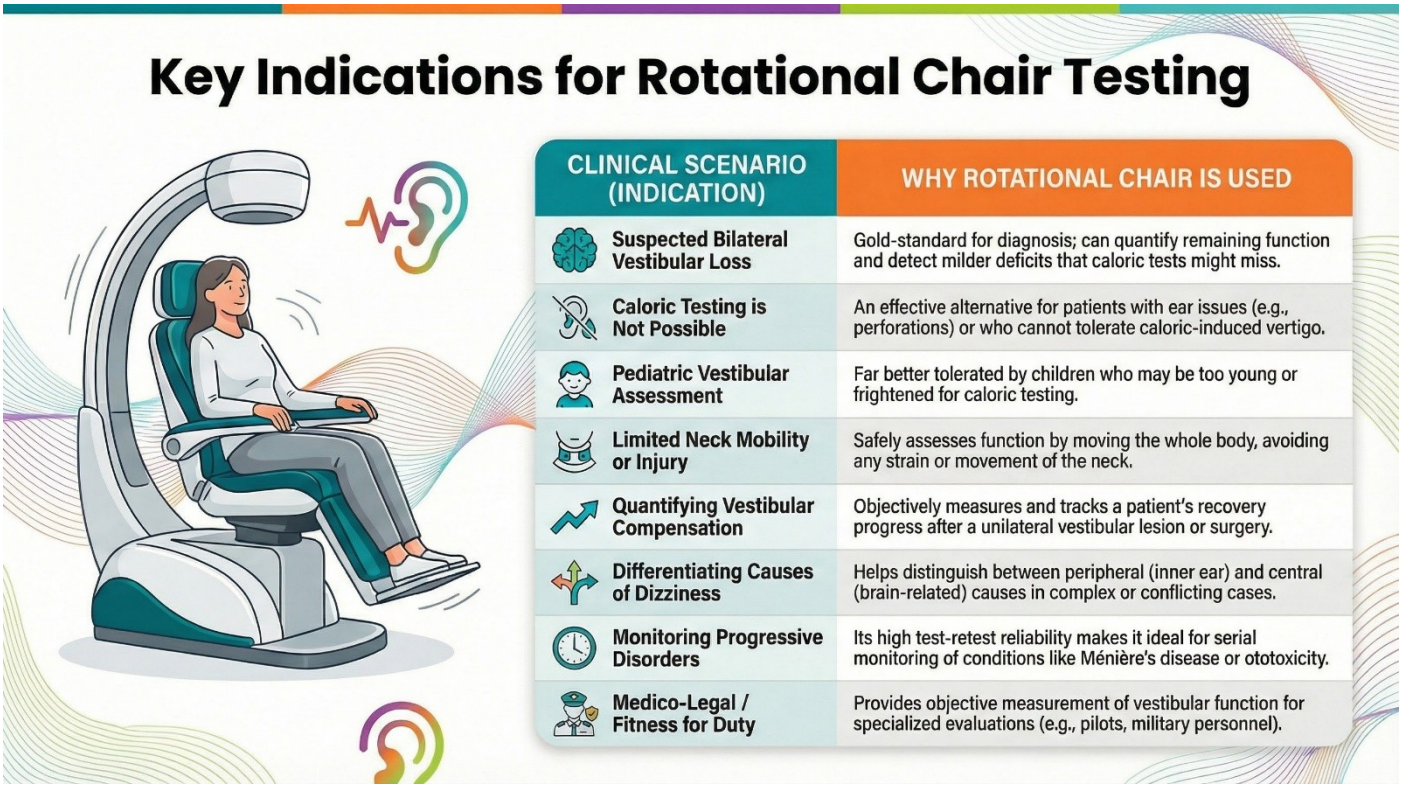
Indications

Rotational chair testing is indicated in a number of clinical scenarios, particularly when information about **bilateral vestibular function or vestibular performance over a range of stimulus frequencies** is desired:

Suspected Bilateral Vestibulopathy: Perhaps the most important indication is when bilateral vestibular loss is suspected (for example, in patients with oscillopsia, gait instability in dark, or a history of ototoxic antibiotic exposure). Rotary chair can confirm bilateral reduction in VOR gain and quantify the remaining function, whereas caloric tests might show absent responses but cannot differentiate a total loss from a partial one easily. In fact, rotational testing is often considered the gold-standard to diagnose bilateral vestibular hypofunction because it can detect milder bilateral deficits that caloric might miss and provide objective data across frequencies. For instance, gentamicin ototoxicity monitoring: a baseline rotary chair and follow-ups can catch declines in gain early, prompting cessation of the drug to prevent complete loss.

When caloric testing is not feasible or is contraindicated: If a patient cannot undergo calorics due to ear issues (e.g. perforated eardrums, mastoid cavities, external ear malformations) or simply cannot tolerate the intense vertigo of calorics, rotational chair is indicated as an alternative to assess vestibular function. As mentioned, active middle ear disease or anatomical variants that block caloric stimulus make rotation a preferable test because it bypasses the middle ear and directly stimulates the labyrinth via head motion. Similarly, in children, calorics are difficult, so paediatric vestibular testing often relies on rotational chair, which is far better tolerated (the child can be gently secured and perhaps watch videos in darkness with small lights for fixation tests, etc.). So rotational testing is indicated for paediatric vestibular assessment, especially in kids too young or frightened for calorics.

Limited cervical mobility or orthopaedic issues: Patients who cannot have their head moved rapidly (for vHIT) or who have neck injuries that prevent head impulse testing can still be evaluated with a rotational chair. The rotational chair moves the entire body en bloc, so it does not strain the neck. For example, an elderly patient with severe cervical spondylosis or a patient in a cervical collar could be tested on a rotary chair to assess vestibular function without violating neck precautions. Thus, any situation where bedside head thrusts or calorics are contraindicated might indicate using the chair.



Quantification of vestibular compensation: In a patient with a known unilateral vestibular lesion, rotational testing can be used to evaluate how well compensation is progressing. The chair can measure improvements in gain, reduction in asymmetry and phase normalization over time. For instance, after a vestibular neuritis, one might do an initial rotary test to get baseline low-frequency deficit and then a follow-up months later to document recovery (often gain improves at higher frequencies, asymmetry diminishes at low frequencies as central compensation occurs). This can help tailor rehabilitation: if compensation is incomplete (still large asymmetry or low gain) months out, perhaps more therapy is needed. Similarly, after vestibular schwannoma surgery (in which one vestibular nerve is removed), rotational chair can track how the remaining ear and central adaptation compensate.

Distinguishing peripheral from central causes in complex cases: Rotational tests can sometimes shed light on whether an abnormal finding is likely peripheral or central. For example, phase lead abnormalities at low frequencies are common in peripheral vestibular loss, whereas grossly normal phase but inability to cancel the VOR with fixation indicates a central issue (cerebellar). If a patient has conflicting results (maybe mild caloric weakness but severe symptoms), a rotational test might help clarify if the vestibular deficit extends across frequencies or not. Also, central disorders like CANVAS [19] (cerebellar ataxia, neuropathy, vestibular areflexia syndrome) present as bilateral loss on both caloric and rotary, but often rotary will show specific patterns like very shortened time constants that hint at central integration loss beyond just peripheral areflexia.

Monitoring progressive disorders: Some vestibular disorders are progressive (e.g., bilateral Ménière's disease, ototoxic effects, degenerative vestibulopathy). Rotational testing is ideal for serial monitoring because it is repeatable and has high test-retest reliability. For example, if a patient has autoimmune inner ear disease under treatment, doing rotational tests every few months can indicate if vestibular function is declining despite therapy. Or in Meniere's, to see if

the contralateral ear is starting to show deficits. The repeatability of rotary chair is high because the stimulus is standardized and not operator-dependent like head impulse, and not as variable as caloric (where slight positioning or irrigation differences can alter outcome).

Medico-legal or fitness for duty evaluations: In contexts like assessing pilots, drivers, or military personnel, a rotational chair test might be indicated to objectively measure vestibular function as part of a fitness exam. For example, some specialized aerospace centres use rotary chairs to ensure pilots don't have bilateral vestibular deficits that could compromise their function. Similarly, in disability evaluations, having both caloric and rotary data can strengthen the case – e.g., showing bilateral vestibular loss on rotary chair supports a claim of severe functional impairment.

Research and special vestibular investigations: Indications also include research settings – e.g., investigating vestibular reflexes in Parkinson's, migraine, etc., often involves rotational paradigms to see subtle central processing differences. Also, testing velocity storage mechanism and VOR suppression are standard in many rotational protocols; if one specifically wants to test those (like suspicion of a cerebellar problem that affects VOR cancellation), the rotational chair's visual vestibular ocular reflex (VVOR) or fixation test is indicated.

In summary, rotational chair testing is indicated whenever a comprehensive assessment of vestibular function is needed, especially for bilateral involvement or in populations where caloric/vHIT are less feasible (children, certain medical conditions). It's also indicated as an adjunct when other tests yield conflicting or incomplete results. Many balance clinics reserve rotary chair tests for cases that require additional clarity – for example, persistent dizziness with normal calorics and vHIT might lead them to do a rotational test to check for a subtle bilateral reduction or a central integration issue.

Contraindications

Rotational chair testing is generally safe and non-invasive, but there are a few contraindications and precautions:

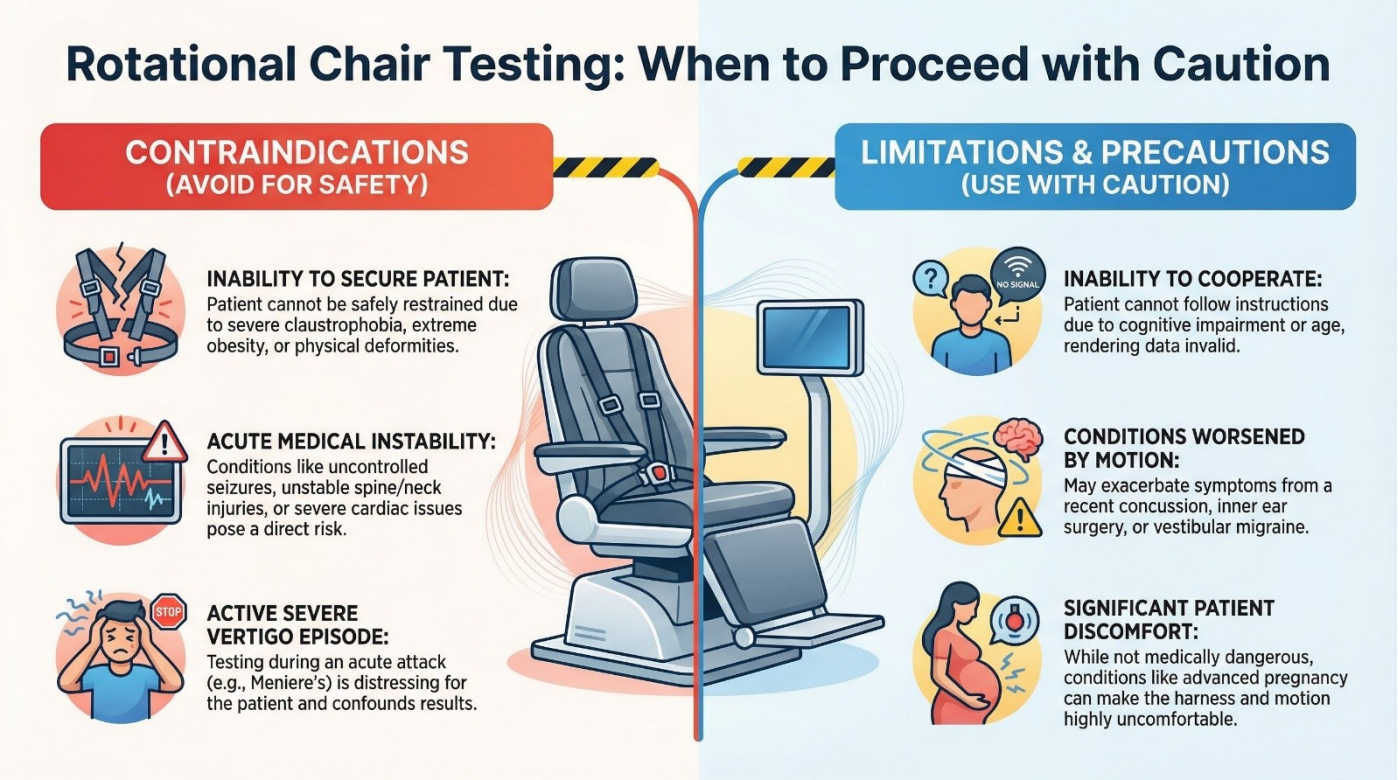
Inability to secure the patient safely: Patients who cannot be properly restrained in the chair (due to extreme obesity beyond the chair's harness capacity, severe orthopaedic deformities, or claustrophobia severe enough that they cannot tolerate being strapped in a dark enclosure) may not be suitable for the test. Safety is paramount because the chair will move; a patient who might try to unstrap themselves or panic could risk injury. Severe claustrophobia is a relative contraindication – some patients cannot tolerate being in the dark booth with the chair; mild anxiety can often be managed with reassurance, but true claustrophobic panic can abort the test.

Uncontrolled seizures or syncope: Because the test is done in darkness and involves moving stimuli, there is a theoretical risk of provoking a seizure in someone with photosensitive epilepsy (though the lights used are usually static or slow-moving dots, not rapid flashes). If a patient has a history of reflex seizures or is extremely prone to syncope with motion, caution is advised. A recent vestibular migraine or propensity to motion-provoked migraines might not be a full contraindication, but one would be cautious as the chair could trigger symptoms.

Acute orthopaedic or neurologic instability: If a patient has a condition like atlantoaxial instability (e.g., severe rheumatoid arthritis in cervical spine) – even though the whole body moves together

in the chair, some minor relative movements can occur. Usually, the head is supported by a headband to minimize any neck movement, so it's generally safe, but if any rotational movement could be dangerous (like unstable spine fracture or severe neck injury), the test should be postponed. Similarly, in patients with a recent concussion or head injury, one might avoid early rotational testing if it exacerbates symptoms, though it's not an absolute contraindication.

Severe cardiac or vestibulo-vasovagal sensitivity: While rare, rotation can cause autonomic responses. If someone has a history of passing out with motion (like Mal de Débarquement or other sensory conflict triggers) or has serious cardiac arrhythmias triggered by stress, there is a slight concern. Usually, screening for cardiovascular stability is wise if one is going to induce vertigo/dizziness – for instance,



we ensure no uncontrolled angina or such. That said, rotational testing is typically gentler than calorics in terms of autonomic stress, but one should still be mindful.

Pregnancy: There is no radiation or physical intrusion, just motion, so pregnancy is not a strict contraindication. However, a heavily pregnant patient might find the harness uncomfortable or might not fit well. And severe nausea from the test could be extra unpleasant in pregnancy. But medically, it's considered safe in pregnancy (unlike, say, radiologic tests).

Inability to keep eyes open or participate: The test requires the patient to keep looking (in darkness) and follow instructions like "count the lights" during certain parts. If a patient is unable to follow these due to cognitive impairment, very young age, or other issues, the data might be uninterpretable. For example, an uncooperative small child might just shut eyes or look around, messing up the data. In such cases, sedation is sometimes considered – but sedation greatly reduces the VOR response, making the test useless for quantitative measure (like a sedated child will have dampened nystagmus). Thus, an untestable patient due to cooperation issues is effectively a contraindication because it won't yield valid data.

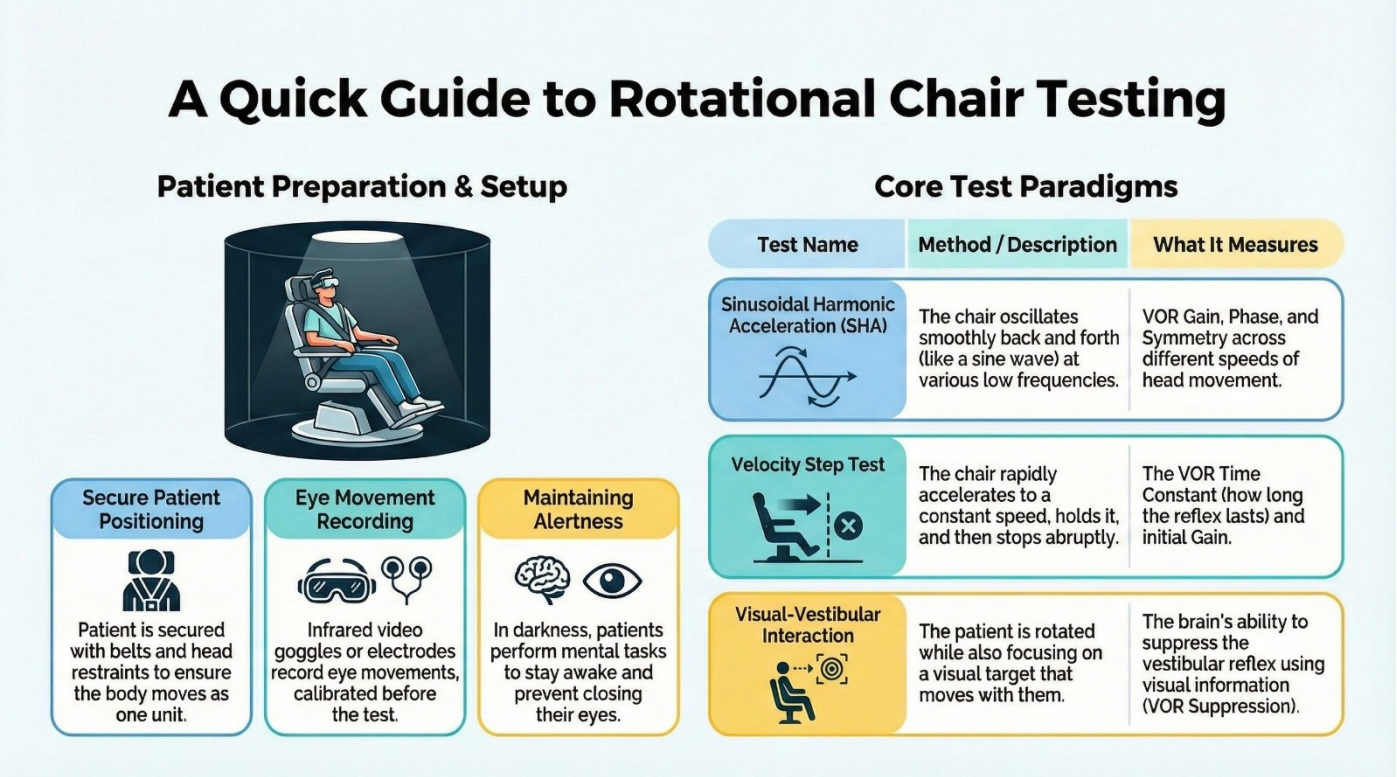
Ear conditions? Unlike calorics, middle ear problems are not a direct contraindication, since the test bypasses the ear canal. However, if someone has had recent inner ear surgery (labyrinthectomy or stapes surgery, etc.), the motion might be uncomfortable or possibly provoke a perilymph fistula (if that's a concern). A patient with a known perilymph fistula or recent inner ear surgery might be asked to avoid heavy motion; whether a gentle rotational test could worsen it is unclear, but caution suggests avoiding it until cleared. Typically, after inner ear surgery, one would wait weeks before doing any vestibular testing (including rotation).

Vertigo spells from other causes: If a patient is in the midst of an acute vertigo episode (like an active Ménière's attack or vestibular migraine attack), rotational testing is not advisable – not only will it be torturous for the patient, but results will be confounded by the ongoing nystagmus or fluctuating function. Better to wait until baseline.

In summary, while rotational chair testing is broadly applicable, it should be avoided or delayed in scenarios where patient safety or ability to endure the test is in question. Ensuring the patient can be properly restrained and is medically stable enough for induced dizziness is key. Compared to calorics, rotation is generally better tolerated and has fewer contraindications (e.g., no ear-specific issues), but the main ones revolve around patient cooperation, physical fit, and safety. If these are addressed, rotational testing can be done in most dizzy patients.

Methods (Clinical and Lab Technique)

Equipment and Setup: The patient is seated in a motorized rotational chair, usually within a light-proof booth or enclosure. They are secured with seat belts over the shoulders and lap, and often additional padding or a footrest to minimize body movement. The head is restrained typically by a head strap or cushion to minimize any independent head motion relative to the chair (the goal is the whole body moves as one). The patient wears either ENG electrodes around the eyes or, more commonly now, infrared video goggles to record eye movements in darkness. If using video goggles, the cameras are calibrated with the patient fixating on known points prior to starting. It's crucial that the goggles are secure so they don't slip during motion, as that could introduce artifact.



Test paradigms: The rotational chair battery usually includes several subtests:

Sinusoidal Harmonic Acceleration (SHA): In this test, the chair oscillates back and forth in a sinusoidal (sine-wave) motion at various frequencies. Common frequencies tested might include 0.01 Hz, 0.02 Hz, 0.04 Hz, 0.08 Hz, 0.16 Hz, 0.32 Hz, and 0.64 Hz. At each frequency, after a few cycles, the eye movement data is captured. Analysis: For each frequency, the system computes the VOR gain and phase and symmetry (or bias). A normal result typically shows near-unity gain at mid-frequencies and relatively small phase lead.

Velocity Step Test (also called Step Velocity or Impulse Rotation): In this test, the chair is accelerated rapidly to a constant velocity and then, after a period, decelerated or stopped abruptly. A common protocol is: rotate the chair to the right to a velocity of e.g. 60°/s within a second, maintain that constant velocity for e.g. 45–60 seconds, then suddenly stop. Key metrics are the time constant (Tc) of VOR decay and the gain at a specific time point. A normal vestibular system has a time constant of around 15–20 seconds.

Visual-Vestibular Interaction Tests: Many chairs have the capability to present a visual stimulus in the dark, like an optokinetic drum or projector, or a fixation light. For example, Optokinetic nystagmus (OKN) test. One common subtest is VOR suppression (also called VOR cancellation) – the patient is rotated, but a visual target moves exactly with them. The normal response is that nystagmus should be greatly suppressed.

Off-Vertical Axis Rotation (OVAR): In specialized centres, there are protocols to test otolith function by rotating the chair with the axis tilted off-vertical.

During Testing: The patient is instructed to keep eyes open, looking straight ahead. For SHA and steps, it's critical they do not fall asleep or close eyes – they're in darkness, so some labs have the patient perform mental tasks similar to caloric alerting tasks to ensure alertness.

Data Recording: The eye movements (nystagmus slow phase) are either automatically analysed by software or later by the examiner. For SHA, typically at each frequency, the eye velocity vs head velocity is plotted and a best-fit sinusoid derived to compute gain and phase.

For velocity steps, the decay of nystagmus is fit to an exponential. The symmetry or bias is usually reported as a percentage.

Interpretation of Results

Interpreting rotational chair results involves looking at patterns of gain, phase, and symmetry across the tested frequencies, as well as results of step tests and VOR suppression.

VOR Gain (SHA):

Normal gain: Typically, around 0.6–0.8 at 0.1 Hz, increasing to ~1.0 by 1 Hz.

Reduced gain: If the gain is globally reduced, this suggests a bilateral vestibular hypofunction.

Phase Lead (SHA):

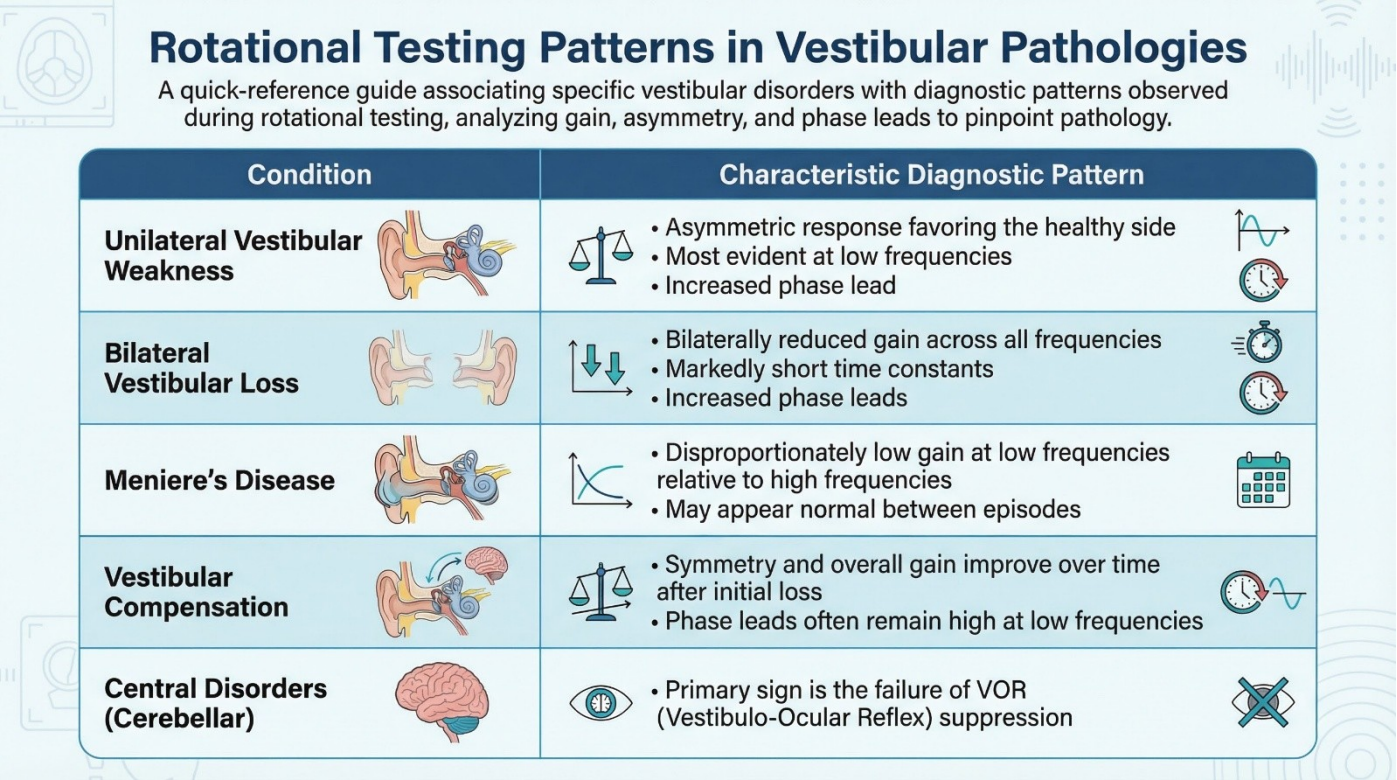
Normal phase: There's usually a phase lead that is larger at low frequency and decreases as frequency increases.

Increased phase lead: Peripheral vestibular lesions (unilateral or bilateral) often cause an excess phase lead at lower frequencies.

Symmetry (SHA): Rotary chair symmetry refers to whether responses are larger when rotating one way vs the other. Unilateral vestibular loss typically shows a significant asymmetry. Bilateral vestibular loss should show little asymmetry.

Velocity Step test interpretation: A reduced time constant indicates that nystagmus decays faster than normal. A very short TC (e.g., 5–6 s) is classic for bilateral vestibular loss.

Visual Suppression (VOR cancellation) test: If the patient cannot adequately suppress the VOR during rotation with a visual target, it indicates a central impairment, likely in the cerebellar flocculus/parafloccullus.



Abnormalities and Diagnostic Patterns

Rotational testing can reveal characteristic patterns for various vestibular pathologies:

Peripheral Unilateral Vestibular Weakness: The classic pattern is an asymmetric response favouring rotations toward the healthy ear, most evident at lower frequencies. Increased phase lead is common.

Bilateral Vestibular Loss: Hallmark is a bilaterally reduced gain across all frequencies, accompanied by markedly shortened time constants and increased phase leads. There is minimal asymmetry.

Vestibular compensation patterns: After unilateral loss, high asymmetry and low gain are seen; after weeks, symmetry improves and overall gain may improve, though phase leads often remain high at low frequencies.

Meniere's Disease: May show normal interictally, but a recognized pattern is disproportionately low gains at low frequencies relative to high.

Central Disorders: Cerebellar flocculus lesions show failure of VOR suppression. CANVAS [19] shows both bilateral loss and poor suppression.

Clinical Implications

Rotational chair test results have significant clinical implications:

Diagnosis and Localization: Confirming and localizing vestibular lesions, especially bilateral loss which calorics might miss.

Treatment decisions: Directs rehabilitation approaches and informs decisions regarding ablative therapy or vestibular implants.

Prognosis: Helps predict functional recovery based on residual gain and central adaptation (time constant).

Guiding Rehabilitation: Detailed frequency data allows physical therapists to tailor specific exercises.

Differentiating central pathology: VOR suppression testing is crucial for identifying cerebellar involvement.

Tracking disease progression: Ideal for serial monitoring in chronic or progressive conditions like ototoxicity.

Patient counselling and validation: Provides tangible evidence of the balance reflex deficit, improving acceptance and compliance.

Fitness for work and driving: Objective data helps determine safety for high-risk occupations or driving.

Pitfalls and Limitations

Limited availability and cost: Chairs are expensive and non-portable, limiting access to specialized centres.

Bilateral stimulation ambiguity: Stimulates both ears together, requiring asymmetry to identify the side of lesion.

Lack of otolithic information: Primarily tests semicircular canals (yaw axis).

Cooperation and alertness: Requires high patient alertness; inattention can lead to false low gains.

Calibration and mechanical issues: Goggle slippage or mechanical motor issues can introduce artifacts.

Interpretation pitfalls: Complex multi-parametric data (gain/phase/symmetry) can be prone to over-interpretation without experience.

Future Directions

The future likely involves expanded frequency ranges, portable rotational devices, and routine inclusion of otolith testing via OVAR. Integration with AI for automated diagnosis and combining rotation with imaging (fMRI/EEG) are emerging research areas. Furthermore, rotational chairs may transition from purely diagnostic to therapeutic tools by running habituation and VOR training protocols.

Other Relevant Considerations

Consistency of Terminology: Using precise terms like "low-frequency hypofunction" and specifying test-specific metrics is vital.

Complementary Nature: Caloric, rotary, and vHIT cover different frequency ranges and should be used together for a complete picture.

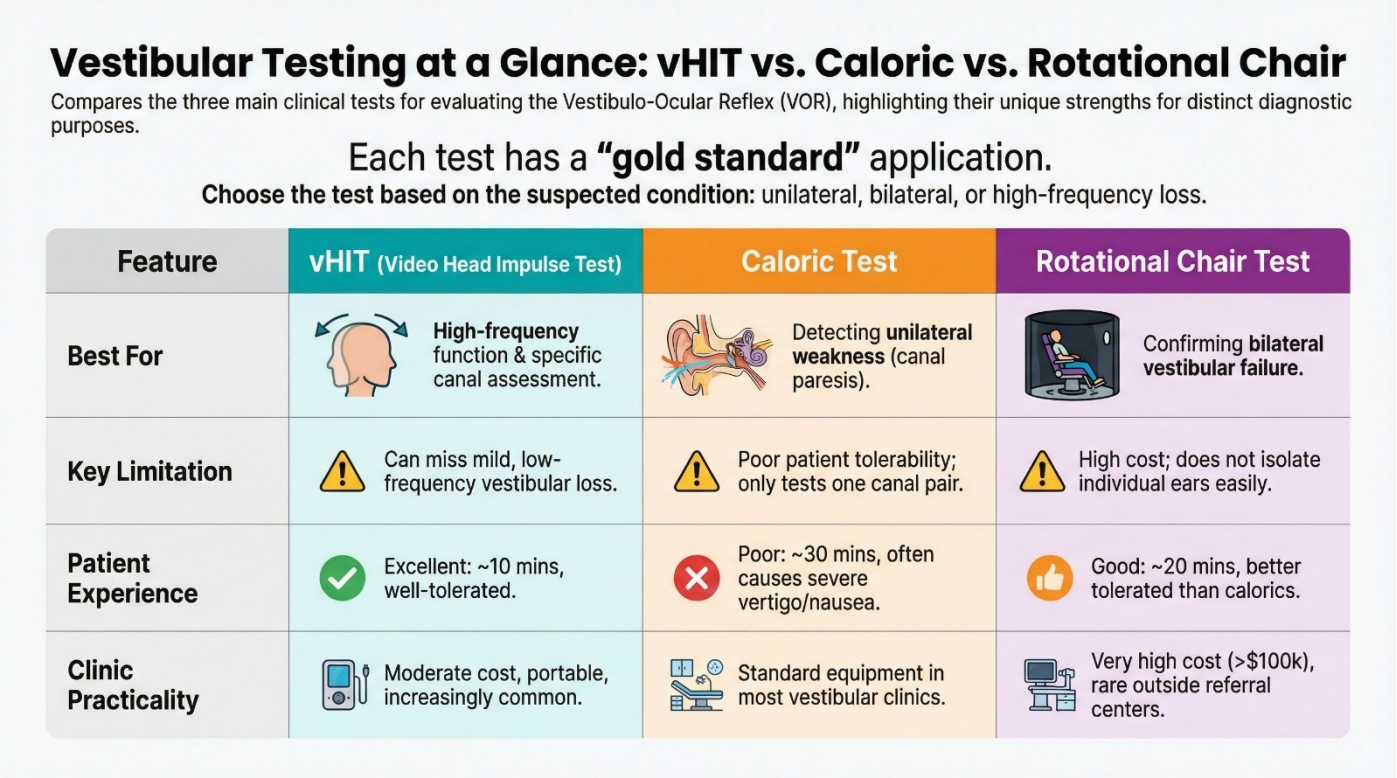
Anatomy: Different tests localize to specific nerves or canals (e.g., vHIT for vertical canals, caloric for superior nerve).

Patient Safety: Proper monitoring for nausea or vasovagal responses is required during testing.

Comparison of vHIT, Caloric, and Rotational Chair Tests in Private Vestibular Clinics

Aspect	Video Head Impulse Test (vHIT)	Caloric Test	Rotational Chair Test
Typical Stimulus	Rapid, high-acceleration head thrusts **(~5 Hz** frequency range). Tests VOR at physiologic high frequencies.	Thermal irrigation (warm & cool water/air) producing low-frequency (~0.003 Hz) endolymph convection.	Sinusoidal or step rotations (0.01–1 Hz). Stimulates both horizontal canals simultaneously.
Equipment & Cost	Video goggles and sensors (~USD $15–30k). Portable; clinic friendly.	Caloric irrigator + VNG (~USD $5–10k + $20k). Standard in many clinics.	Computerized chair in booth. High cost (> $100k); requires dedicated space.
Test Duration	Very brief – ~10 minutes.	Lengthy – typically ~30 minutes.	Intermediate – ~15–20 minutes.
Patient Tolerability	Excellent. Well tolerated; no sustained nausea.	Poor. Often induces strong vertigo, nausea, and sweating.	Moderate/Good. Generally, better tolerated than calorics.
Diagnostic Yield	High-frequency function; individual canal localization. Best for overt/covert saccades.	Gold-standard for unilateral canal paresis. Very sensitive to mild/partial losses.	Gold-standard for bilateral vestibulopathy. Tracks global VOR and compensation.
Use in Clinics	Increasingly common due to moderate cost and ease.	Long-time standard; part of comprehensive VNG battery.	Rare in private practice; mostly in referral/research centres.

Pros	Fast, well-tolerated, canal specific.	Highly sensitive to small asymmetries.	Comprehensive view; best for bilateral failure.
Cons	Can miss mild low-frequency losses.	Poor patient tolerability; tests only horizontal canal.	High cost; individual ear isolation requires asymmetry.



Reference List

1. Halmagyi GM, Curthoys IS. A clinical sign of canal paresis. Arch Neurol. 1988;45(7):737-9.
2. MacDougall HG, Weber KP, McGarvie LA, Halmagyi GM, Curthoys IS. The video head impulse test: diagnostic accuracy in peripheral vestibulopathy. Neurology. 2009;73(14):1134-41.
3. Weber KP, MacDougall HG, Halmagyi GM, Curthoys IS. Impulsive testing of individual semicircular canal function using video-oculography. Clin Neurophysiol. 2009;120(1):125-30.
4. Halmagyi GM, Chen L, MacDougall HG, Weber KP, McGarvie LA, Curthoys IS. The Video Head Impulse Test. Front Neurol. 2017;8:258.
5. Weber KP, et al. Video head-impulse test: it's not as easy as it looks. Neurology. 2013;81(4):306-7.
6. Strupp M, et al. Vestibular Neuritis and VPPB. Front Neurol Neurosci. 2013;34:108-14.
7. Alhabib SF, Sangawi MS. Role of Video Head Impulse Test (vHIT) in Evaluation of Vestibular Disorders: A Systematic Review. J Otol. 2017;12(4):179-85.
8. Blödow A, et al. Sensitivity and specificity of the video head impulse test. Neurology. 2013;80(1):9-17.

9. Kattah JC, et al. HINTS to diagnose stroke in the acute vestibular syndrome: three-step bedside oculomotor examination more sensitive than early MRI diffusion-weighted imaging. Stroke. 2009;40(11):3504-10.
10. McCaslin DL, et al. The Video Head Impulse Test (vHIT): Pitfalls of interpretation. J Am Acad Audiol. 2014;25(9):801-7.
11. MacDougall HG, et al. The suppression head impulse test (SHIMP): a novel mechanism to measure vestibular-ocular reflex function. Front Neurol. 2016;7:47.
12. Bárány R. Untersuchungen über den vom Vestibularapparat des Ohres reflektorisch ausgelösten rhythmischen Nystagmus und seine Begleiterscheinungen. Berlin: Coblentz; 1906.
13. Fitzgerald G, Hallpike CS. Studies in human vestibular function: I. Observations on the directional preponderance ("Nystagmusbereitschaft") of caloric nystagmus resulting from cerebral lesions. Brain. 1942;65(2):115-37.
14. Jongkees LBW. Which is the preferable method of performing the caloric test? Arch Otolaryngol. 1948;48(4):402-17.
15. Claussen CF. [On the history of the caloric test]. Laryngol Rhinol Otol (Stuttg). 1980;59(1):1-10.
16. Dix MR, Hallpike CS. The pathology symptomatology and diagnosis of certain common disorders of the vestibular system. Proc R Soc Med. 1952;45(6):341-54.
17. Ganti L, et al. Vestibular Testing. [Updated 2025]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2025.
18. Yardley L, et al. Relationship between anxiety and caloric hyperactivity. J Audiol Med. 1998.
19. Szmulewicz DJ, et al. CANVAS: cerebellar ataxia with neuropathy and bilateral vestibular areflexia syndrome. J Neurol Sci. 2011;301(1-2):115-22.
20. British Society of Audiology. Recommended Procedure: Bithermal Caloric Test. Reading: BSA; 2010.
21. Jacobson GP, et al. The negative predictive value of the monothermal warm caloric test. Am J Otol. 1995;16(1):18-27.
22. Fuemmeler L. Introduction to Rotary Chair Testing. AudiologyOnline. 2022.
23. Bárány R. Physiologie und Pathologie des Bogengang-Apparates beim Menschen. Leipzig: Deuticke; 1907.
24. Schäfer JR. [Early motorized rotational devices]. Z Laryngol Rhinol. 1935.
25. Kornhuber HH. [Physiology and pathology of the vestibular system]. Stuttgart: Schattauer; 1966.
26. Jirák V. [Clinical development of the rotary chair]. Cesk Otolaryngol. 1970.
27. Schilder H. NASA Vestibular Research and Rotary Stimuli. NASA Tech Rep. 1975.
28. Hain TC, et al. Sinusoidal harmonic acceleration in vestibular disorders. 1987.
29. Herdman SJ, et al. Clinical utility of the rotational chair test. 1986.
30. Curthoys IS. The physiology of the head impulse test. 2000.
31. Black FO, et al. Rotational chair protocols for clinical use. 1980.
32. Leopold M. Multi-frequency VOR characterization in humans. 1985.
33. Herdman SJ, Clendaniel RA. Vestibular Rehabilitation. 4th ed. Philadelphia: FA Davis; 2014.

34. Jacobson GP, Shepard NT. Balance Function Assessment and Management. 2nd ed. San Diego: Plural Publishing; 2015.
35. Halmagyi GM, et al. The Video Head Impulse Test. Front Neurol. 2017;8:258.
36. StatPearls. Vestibular Diagnostics and Testing Protocols. 2025.
37. Barany Society. Guidelines for Vestibular Function Testing. 2020.
38. Interacoustics Academy. Guide to Vestibular Testing and vHIT Interpretation. 2023.
39. Baloh RW, Honrubia V. Clinical Neurophysiology of the Vestibular System. 4th ed. Oxford University Press; 2011.
40. Whitney SL, Jacobson GP. Balance Function Assessment and Management. 2011.

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While every effort has been made to ensure the accuracy and completeness of the content, vestibular medicine is a rapidly evolving field. Clinicians are encouraged to verify specific protocols, normative values, and therapeutic recommendations against current published guidelines and primary literature.

References and Attribution

All referenced works are cited in good faith for educational purposes. Where specific normative data or clinical criteria are cited, the original sources should be consulted for full methodological detail and applicability to individual patient populations.

Version History

Version 3.0 — April 2026 | Full ADC standard rebuild with front matter, table of contents, callout boxes, and image-preserving reformatting.

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