Specialised Oculomotor Tests:
A Comprehensive Clinical Review

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How to Use This Review

This document is the companion clinical literature review to the specialised oculomotor subtests and advanced VNG 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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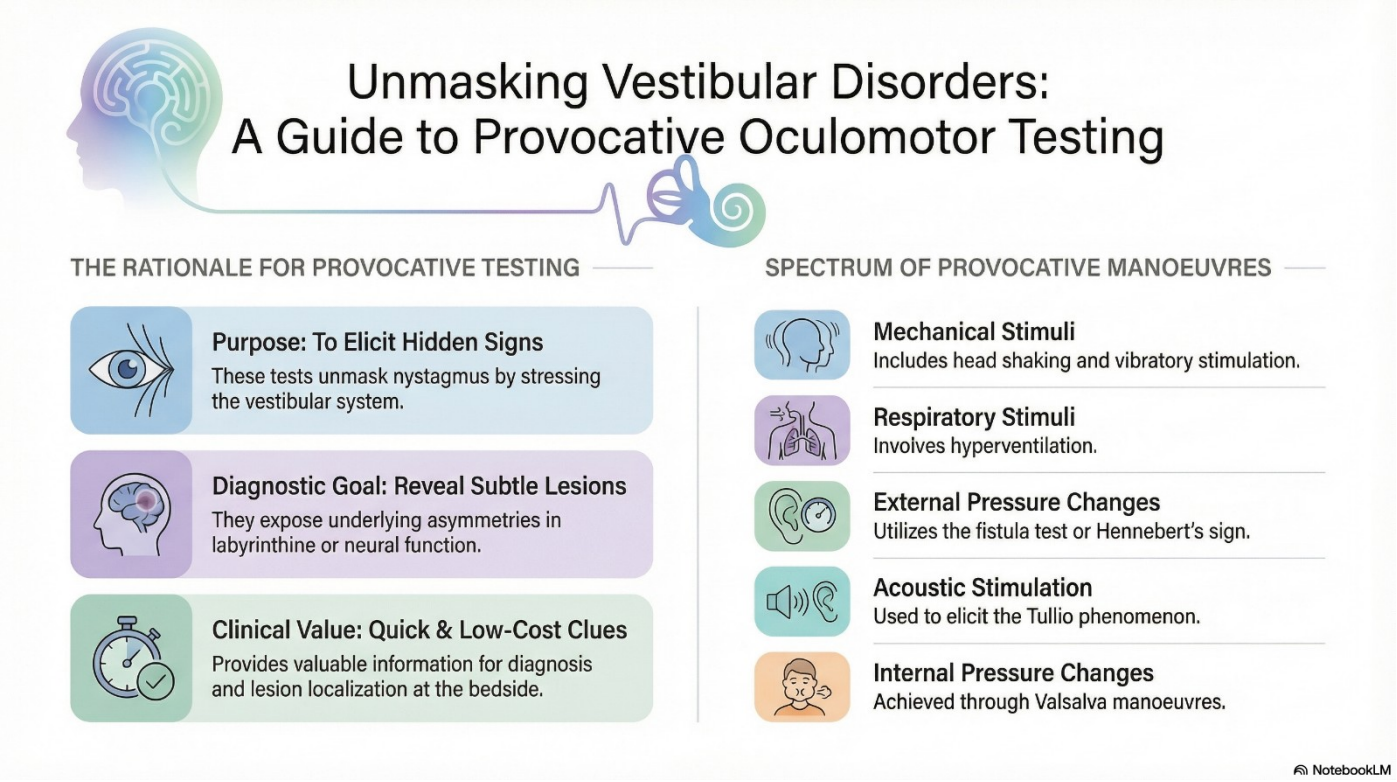
Introduction

Vestibular disorders often present with episodic vertigo or imbalance that may not be evident on routine examination. Provocative oculomotor tests are specialized bedside manoeuvres used to elicit or unmask nystagmus by applying physiological or mechanical stress to the vestibular system. These tests exploit underlying asymmetries in labyrinthine or neural function, revealing subtle vestibular lesions that might otherwise go undetected. They are especially valuable in neuro-otology and vestibular medicine, providing quick, low-cost clues for diagnosis and lesion localization [2, 17]. This manuscript focuses on “special” oculomotor provocation tests that induce nystagmus via targeted stimuli, highlighting their physiology, examination technique, and localization value.

The spectrum of provocation tests includes manoeuvres such as

1- Head shaking and vibratory stimulation (external mechanical stimuli),
2- Hyperventilation (a respiratory physiologic stimulus),
3- Pressure changes to the external auditory canal (fistula test/Hennebert’s sign),
4- Intense acoustic stimulation (Tullio phenomenon), and
5- Valsalva manoeuvres (internal pressure changes).



Vibration-induced nystagmus (VIN) and head-shaking nystagmus (HSN) are recognized members of this group but will not be detailed here, as they are covered separately. They are mentioned as part of the broad classification of provoked nystagmus because they also transiently perturb labyrinthine input to reveal vestibular imbalance [17]. Instead, this manuscript will emphasize four key tests in full detail:

1. **Hyperventilation-Induced Nystagmus (HVIN)** – nystagmus triggered by voluntary hyperventilation.
2. **Pressure-Induced Nystagmus (Hennebert's sign / fistula test)** – nystagmus caused by changes in external ear canal pressure.
3. **Sound-Induced Nystagmus (Tullio phenomenon)** – nystagmus provoked by loud sounds or acoustic energy.
4. **Valsalva-Induced Nystagmus** – nystagmus elicited by strain manoeuvres that raise intrathoracic/intracranial or middle-ear pressure.

These tests are organized herein by stimulus type (external mechanical, respiratory, external pressure, acoustic, internal pressure) to underscore common mechanisms. Each section will address the physiological basis, examination method, and interpretation of the respective test. Clinical relevance is highlighted first: these manoeuvres have significant diagnostic utility in conditions such as vestibular schwannoma, demyelinating vestibulopathies, Perilymphatic fistula, superior canal dehiscence (SCD) syndrome, and other "third window" lesions. We also discuss how nystagmus patterns differentiate peripheral vs. central aetiologies, and we present at least five clinical scenarios demonstrating application of these tests in practice.

Finally, summary tables are provided to consolidate definitions, typical nystagmus characteristics (including localization patterns for different labyrinthine structures), distinguishing features between peripheral and central causes, and potential pitfalls in interpretation.

Neurophysiology and Anatomical Substrates

A solid understanding of vestibular neurophysiology and inner ear anatomy is fundamental to interpreting provoked nystagmus. The peripheral vestibular apparatus consists of the semicircular canals (anterior/superior, posterior, and horizontal/lateral canals) and the otolith organs (utricle and saccule). Each semicircular canal is oriented in an orthogonal plane and is maximally sensitive to angular acceleration in its plane. Movement of endolymph within a canal deflects the cupula in the ampulla, leading to excitation or inhibition of the hair cells depending on the direction of deflection. Notably, Ewald's first law states that stimulation of a semicircular canal causes eye movements (slow phase) in the plane of that canal [10]. The direction of the induced slow-phase eye movement depends on whether the flow of endolymph is toward or away from the utricle (ampullopetal vs. ampullofugal), which in turn depends on hair cell polarity in that canal. In the horizontal canals, endolymph movement toward the utricle (ampullopetal) excites the hair cells, whereas in the vertical canals (anterior and posterior) the opposite is true – ampullofugal flow excites those canals [10, 6]. Thus, each canal has a characteristic pattern of eye movement when stimulated: e.g. excitatory stimulation of a horizontal canal causes horizontal eye deviation toward the opposite side; excitatory stimulation of a superior (anterior) canal causes upward and torsional eye movement (torsion with the upper poles of the eyes toward the side of the stimulated canal), whereas a posterior canal excitation causes downward and torsional eye movement (upper poles toward the stimulated side) [6]. Figure 1 schematically illustrates these canal-induced eye movement vectors.

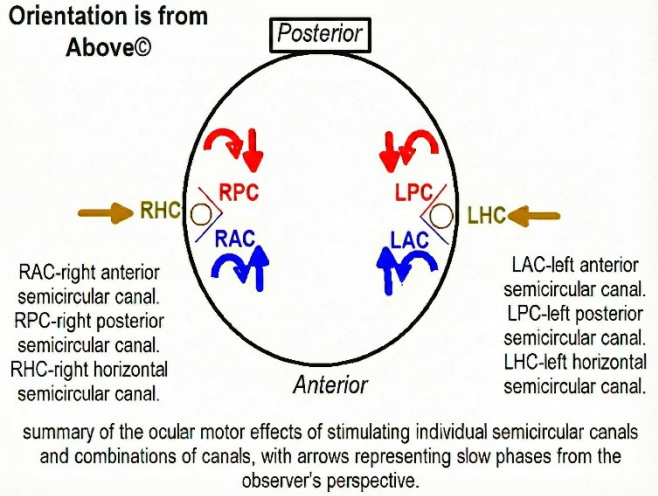


Figure 1: Schematic representation of eye movement directions induced by stimulation of each semicircular canal (viewed from above). In this diagram, arrows indicate the direction of the slow-phase eye movement when a given canal is excited. For example, stimulation of a horizontal canal produces slow-phase movement toward the contralateral side, whereas stimulation of the right anterior canal (RAC) produces slow upward movement with torsion (intorsion of the right eye, extorsion of the left eye). The fast phase of nystagmus is directed opposite to the slow phase (by convention, nystagmus direction is named after the fast phase). Knowledge of these patterns allows clinicians to infer which canal or end-organ is being stimulated in various provocation tests [6].

The vestibular end-organs are innervated by the vestibular division of the eighth cranial nerve (vestibulocochlear nerve). The superior vestibular nerve supplies the horizontal and superior canals and the utricle, while the inferior vestibular nerve innervates the posterior canal and saccule. A unilateral vestibular lesion (whether at the end-organ, nerve, or root entry zone level) creates an asymmetry in baseline vestibular input that the brain may partially compensate for over time. However, under certain conditions (e.g. physiological stress or specific stimuli), this latent asymmetry can manifest as nystagmus. Provocation manoeuvres often work by temporarily altering the firing rate on one side (or altering central compensation), effectively "unmasking" the imbalance between the two vestibular nuclei. The resulting nystagmus follows Alexander's law and other vestibular principles – typically a jerk nystagmus with the fast phase directed toward the more active side (or away from the relatively inhibited side).

Central vestibular pathways are also critical in understanding these tests. All primary vestibular afferents project to the vestibular nuclei in the brainstem (superior, medial, lateral, and inferior vestibular nuclei). From there, secondary neurons project via the medial longitudinal fasciculus (MLF) to the ocular motor nuclei (III, IV, VI) to mediate the vestibulo-ocular reflex (VOR). The cerebellum, particularly the flocculus and nodulus, plays a modulatory role, helping to calibrate and suppress inappropriate nystagmus. For instance, cerebellar clamp mechanisms normally dampen spontaneous nystagmus after an acute vestibular lesion; if these compensatory mechanisms are disrupted (as can happen with certain stimuli like hyperventilation or under specific central lesions), nystagmus can reappear. The ocular motor output that we observe as nystagmus is thus the end result of complex interactions between peripheral signals and central processing. By analysing the direction, phase, and other qualities of provoked nystagmus, one can often deduce the anatomical origin of the response (e.g. a particular semicircular canal or otolith organ, or a specific side of vestibular nerve involvement) [9, 10].

In summary, the key neurophysiological underpinnings relevant to provocation tests are: (1) the orientation and response polarity of each semicircular canal (and to a lesser extent the otolith organs), (2) the baseline asymmetry created by peripheral lesions and how central compensation may mask or unmask this, (3) the role of specific neural substrates (like partially demyelinated axons or “third windows” in the labyrinth) that can be influenced by targeted stimuli, and (4) the characteristic eye movement patterns that inform localization. With this foundation, we can better appreciate how each provocative manoeuvre works and what an observed nystagmus signifies about the site of lesion or dysfunction.

Mechanisms and Classification

Provoked nystagmus can be classified by the type of stimulus used to elicit it, each tapping into different mechanisms of labyrinthine or neural activation. Below we categorize the provocation tests into five stimulus groups and explain the underlying mechanisms of each:

3.1 External Mechanical Stimuli (Vibration and Head Shaking)

External mechanical stimulation involves imparting physical motion or vibration to the head to perturb the vestibular organs. In the head-shaking test, the patient vigorously shakes the head side-to-side (typically ~20 cycles) and then stops; any ensuing nystagmus (observed after the shaking stops) is considered head-shaking nystagmus. The mechanism relies on transiently overwhelming the vestibular system: in a normal symmetric vestibular apparatus, post-headshake nystagmus is minimal or short-lived, but with a unilateral vestibular hypofunction, the high-frequency stimulation saturates both sides and upon cessation a bias emerges favouring the healthier side (resulting in nystagmus beating toward the healthy ear). Head-shaking nystagmus is thus a sign of dynamic asymmetry; for example, in a right vestibular neuritis, head shaking often produces a few beats of left-beating nystagmus as the uninjured side (left) drives the fast phase. This test is well established to detect unilateral vestibular deficits. Similarly, skull vibration-induced nystagmus (SVIN) uses a high-frequency vibrator (e.g. 100 Hz) applied to the mastoid or forehead. Vibration stimulates both labyrinths but often preferentially activates the side with better residual function (likely via bone-conducted sound and utricular responses), causing a nystagmus beating toward the healthier ear in unilateral peripheral lesions [17]. SVIN is highly sensitive to even mild asymmetries: studies show that 55–90% of patients with superior canal dehiscence or unilateral vestibulopathy have a vibration-induced nystagmus [18]. It is thought that vibration causes a nonspecific excitatory bias of labyrinthine receptors, unmasking

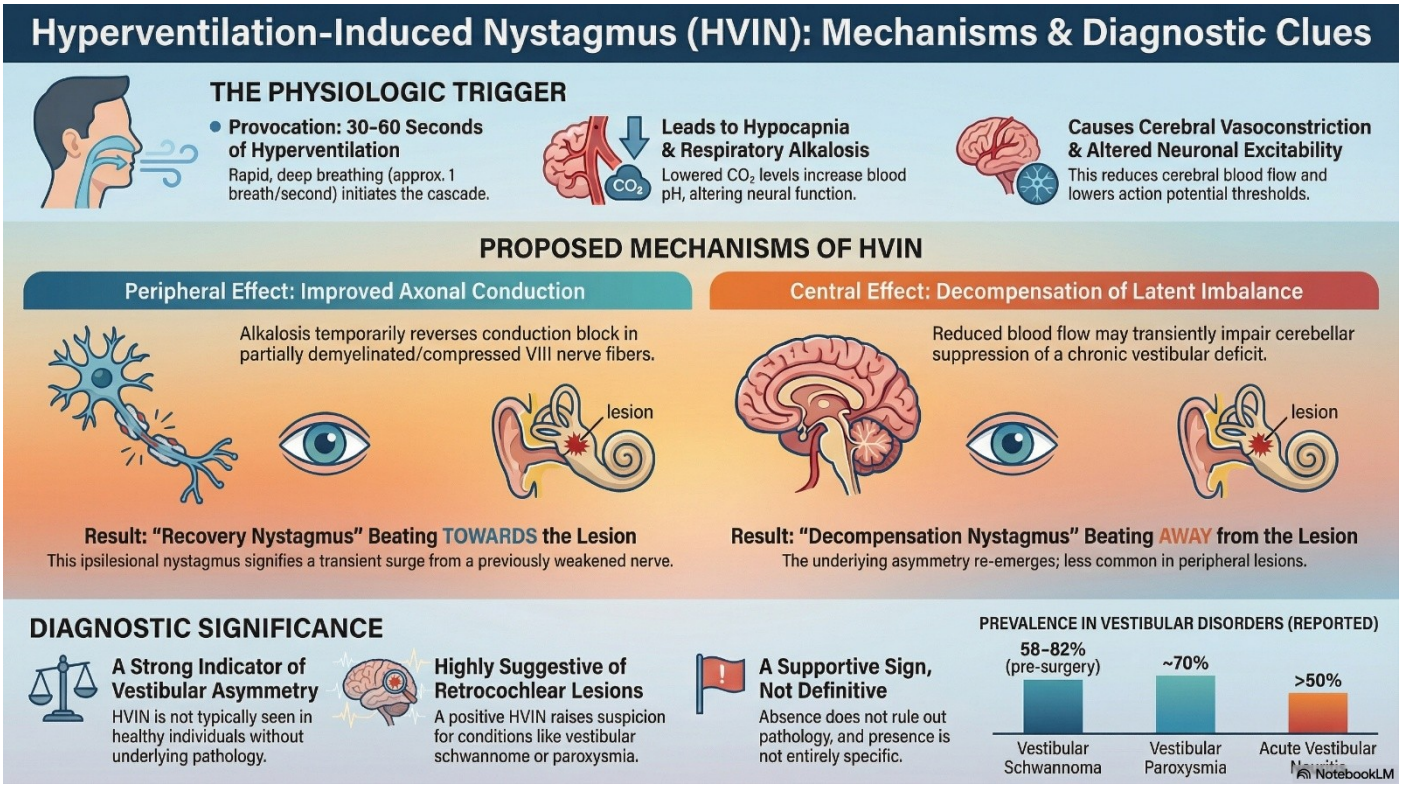
the asymmetry akin to a caloric or headshake test. Both head shaking and vibration primarily assess vestibular imbalance; their presence indicates a peripheral vestibular lesion but does not by itself localize which canal or specific pathology, and their nystagmus patterns have limited specificity for central vs. peripheral causes (vertical or torsional components can occur in either) [17]. For completeness, we acknowledge these external mechanical tests as part of the provocation family, though detailed coverage is available elsewhere.

3.2 Respiratory/Physiologic Stimuli (Hyperventilation)

Hyperventilation-induced nystagmus (HVIN) is triggered by rapid, deep breathing – typically one breath per second for 30–60 seconds. Hyperventilation produces systemic physiologic changes: notably, hypocapnia (low CO_2) leading to respiratory alkalosis. This alkalosis has several effects on the nervous system: it causes cerebral vasoconstriction (reducing cerebral blood flow by up to ~50% transiently) and alters neuronal excitability by increasing blood pH and reducing ionized calcium [2]. These changes can affect vestibular function in both peripheral and central pathways. The exact mechanism of HVIN remains partially controversial, but two major hypotheses exist:

1. **Direct Peripheral Nerve Effect:** In patients with demyelinating or compressive lesions of the vestibular nerve (e.g. vestibular schwannoma or neurovascular compression as in vestibular paroxysmia), hyperventilation is thought to improve axonal conduction temporarily in partially demyelinated fibres [3]. Alkalosis reduces the extracellular calcium concentration, which can lower the threshold for action potential generation and partly reverse conduction block in demyelinated axons. The result is a transient surge of activity in the affected vestibular nerve, mimicking a sudden recovery of function. Clinically, this appears as a “recovery nystagmus,” with the fast phase beating toward the side of the previously weakened nerve [1, 3]. In other words, if a patient has a unilateral vestibular deficit (e.g. right vestibular schwannoma compressing the nerve), hyperventilation may momentarily increase right-sided vestibular firing, producing a leftward slow phase (as the brain perceives a right-sided excitation) and a right-beating nystagmus (fast phase toward the lesion side) [1]. This ipsilesional HVIN has been reported in a significant fraction of vestibular schwannoma cases and is considered a supportive sign of a retrocochlear lesion [16]. It is also observed in other demyelinating vestibulopathies (e.g. patients with multiple sclerosis plaques affecting the root entry zone of VIII nerve, or in vestibular paroxysmia due to nerve compression) [3].
2. **Central Decompensation:** An alternate or additional mechanism involves central vestibular compensation. Hyperventilation-induced cerebral vasoconstriction can cause a brief reduction in cortical and cerebellar activity. It has been proposed that this “cortical suppression” may diminish the normal compensatory mechanisms that keep a chronic vestibular imbalance latent [2]. In patients with a compensated unilateral lesion, the cerebellum (especially nodulus) actively suppresses any spontaneous nystagmus at rest. If hyperventilation transiently impairs this suppression (via decreased blood flow), a previously compensated asymmetry might re-emerge as nystagmus. This could explain cases of HVIN in which the nystagmus beats away from the lesion (interpreted as a “decompensation nystagmus” or partial relapse). However, most clinical data indicates HVIN in peripheral lesions tends to be ipsilesional (a “recovery” type), supporting the improved conduction hypothesis for vestibular nerve lesions [3, 16]. In central lesions, the pattern may be less predictable and the incidence lower [2].

Regardless of mechanism, HVIN strongly suggests an underlying vestibular asymmetry. It is not typically seen in healthy subjects (hyperventilation might cause dizziness or faintness in anyone, but not usually nystagmus without pathology) [25]. Among dizzy patients, studies have found HVIN in roughly 20–30% overall [2, 5]. The prevalence is higher in certain conditions: for example, in one series HVIN appeared in over 50% of acute unilateral vestibulopathies (vestibular neuritis) during the acute phase, then dropped to ~20% in the chronic compensated phase [2]. In vestibular schwannoma, older reports showed HVIN in 58–82% of cases pre-surgery and even 100% post-surgically (perhaps reflecting the universal acute imbalance after neurectomy) [2]. These figures indicate that HVIN is particularly sensitive to retrocochlear lesions and acute unilateral vestibular loss [16]. It can also be induced in certain central disorders like vestibular paroxysmia: one study found 70% of patients with vestibular paroxysmia (neurovascular compression of VIII nerve) had transient nystagmus with 3 minutes of hyperventilation [3]. Thus, HVIN straddles the boundary of peripheral and central – it is classically associated with peripheral nerve lesions (demyelination/compression) but can also manifest in central demyelinating disease or even anxious hyperventilation in those with underlying vestibular instability.



In summary, hyperventilation is a physiologic stress test for the vestibular system: it transiently alters blood gas composition and neural excitability. If a latent asymmetry exists, hyperventilation may drive a temporary nystagmus. Typically, this is a horizontal nystagmus (often with a slight torsional component) with the fast phase toward the previously compromised side (ipsilesional) in peripheral disorders [1]. Vertical components are uncommon but may appear if vertical canal fibres are involved (Minor et al. noted horizontal canal afferents are most affected, superior canal less so, and posterior canal minimally by hyperventilation [1]). HVIN is an important diagnostic sign – for example, a positive HVIN in a patient with unilateral hearing loss or asymmetric vestibular function could raise suspicion for a vestibular schwannoma or other retrocochlear pathology [16]. However, its absence does not rule out such pathology (many vestibular

schwannomas do not produce HVIN), and its presence is not entirely specific (it can occur in central lesions or even in some normal-caloric patients without a firm diagnosis [2, 5]). Therefore, HVIN serves as one piece of the puzzle, best interpreted alongside other vestibular tests.

3.3 External Canal Pressure Stimuli (Fistula Test / Hennebert Sign)

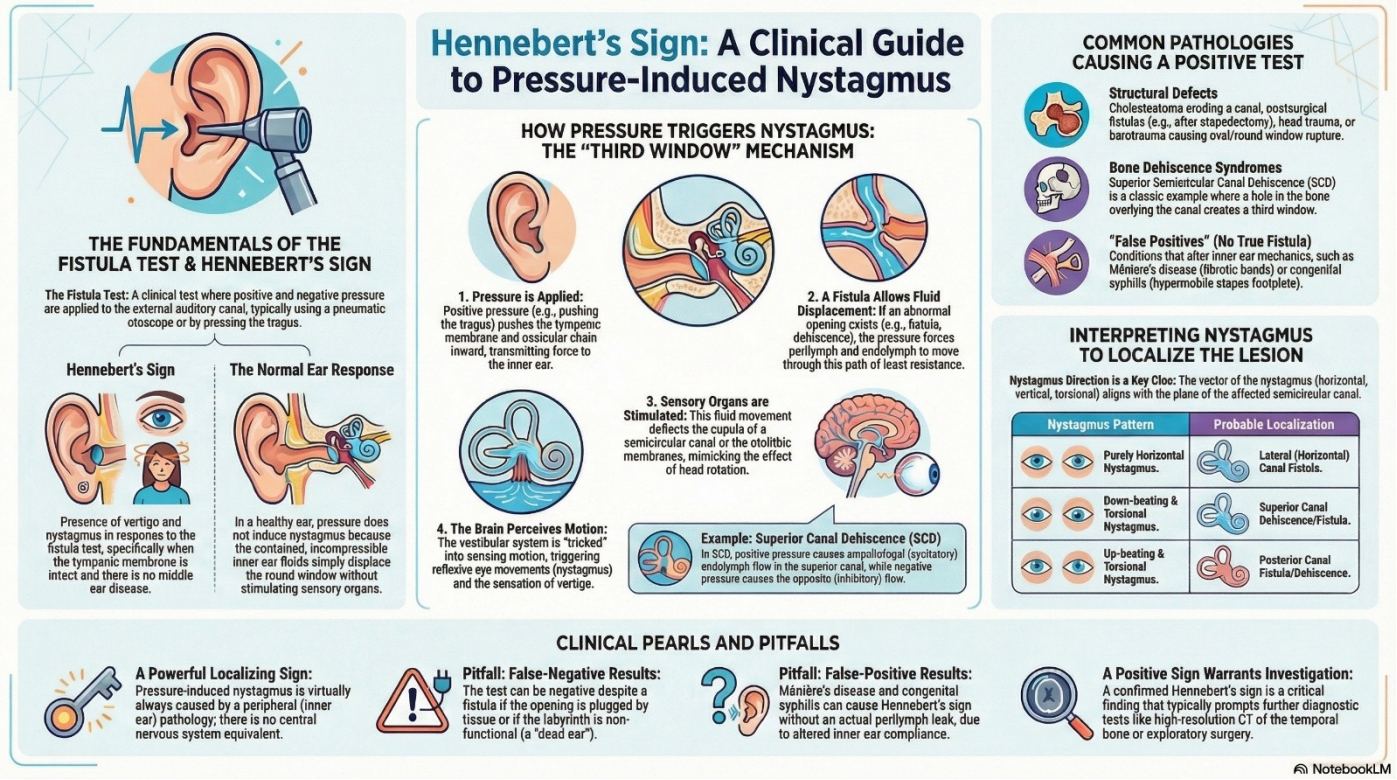
Applying pressure to the external auditory canal can induce nystagmus if there is an abnormal bony or membranous communication in the inner ear – a so-called "fistula" or third window phenomenon. The fistula test is classically performed by using a pneumatic otoscope or by repeatedly pressing and releasing the tragus to create positive and negative pressure transients in the external canal [4]. In a normal ear with intact bony labyrinth and appropriately stiff oval/round window membranes, moderate pressure changes do not cause nystagmus – the fluids in the inner ear are essentially incompressible and confined, so any pressure simply displaces the round window without significantly deflecting the sensory organs. However, if an abnormal opening exists (a perilymphatic fistula or a bone dehiscence), pressure can drive endolymph/perilymph movement within the vestibular organs, leading to pressure-induced nystagmus. A positive fistula test (also known as Hennebert's sign when occurring without middle ear disease) is defined by reproduction of vertigo and nystagmus upon pressure application [4].

Mechanistically, consider a perilymphatic fistula (PLF) at the oval or round window: pushing on the tragus raises pressure in the external canal, which pushes the tympanic membrane and ossicular chain inward (like a mini-Valsalva against the oval window) [10]. If the oval or round window membrane is torn (or bone eroded), this pressure is transmitted into the inner ear fluids and can cause a volume displacement through the fistula. The displacement of perilymph/endolymph can deflect the cupula of semicircular canals or the otolithic membranes, tricking the vestibular system into sensing motion. The direction of cupular deflection depends on whether the pressure is positive or negative and on the location of the fistula. For example, positive pressure (pushing inward) tends to push endolymph toward certain canals. In a superior semicircular canal dehiscence (an opening in the bone over the superior canal), positive pressure in the external canal will push the stapes inward and force the membranous canal to bulge outward at the dehiscence, causing endolymph flow away from the utricle (ampullofugal) in the superior canal [10]. Ampullofugal flow in the superior canal excites it, leading to an excitatory nystagmus (eyes move slowly in the plane of that canal). Conversely, negative pressure (pulling outward or release) causes the opposite (ampullopetal flow in that canal, inhibiting it) [10]. Each canal behaves similarly: a fistula involving the horizontal canal will experience ampullopetal endolymph movement with positive pressure (exciting the horizontal canal, since ampullopetal is excitatory for horizontal) and ampullofugal with negative (inhibitory), or vice versa if the fistula is effectively acting at the round window. The net result: pressure applied = nystagmus in one direction; pressure released = nystagmus in the opposite direction, often with associated vertiginous sensation [6, 4]. The patient may also exhibit Hennebert's symptom, which is dizziness or vertigo from pressure changes even if nystagmus is not observed [8].

Common pathologies that cause a positive fistula test include: cholesteatoma eroding the horizontal canal, postsurgical fistulas (e.g. after stapedectomy or inner ear surgery), head trauma barotrauma causing oval or round window rupture, and bone dehiscence syndromes like Superior Semicircular Canal Dehiscence (SCD) [10]. Interestingly, the fistula test can also be positive in congenital syphilis and in some cases of Ménière's disease despite no actual hole in the labyrinth – this is termed false-positive Hennebert's sign [8]. In congenital syphilis, it's thought

that syphilitic osteitis makes the stapes footplate hypermobile or alters inner ear compliance, so pressure changes excessively stimulate the vestibule [20]. In Meniere's disease, fibrosis bands or altered fluid dynamics might transmit pressure to the utricle (studies report ~25% of Meniere's patients can have a Hennebert's sign) [15]. These cases underscore that a "pressure-induced nystagmus" can occur without an actual leaking fistula – essentially a pathological third window effect due to increased inner ear compliance or abnormal connections (e.g. a mobile footplate acting like a window). Hence, the term Hennebert's sign is used for pressure nystagmus with an intact tympanic membrane and no obvious middle ear defect [4].

The nystagmus characteristics during a fistula test can help localize the affected ear and even the canal. Typically, nystagmus from an active fistula will have its fast phase directed toward the affected ear when pressure is applied (if the fistula causes excitatory stimulation) [8]. For instance, in a right ear lateral canal fistula, pressing the tragus often causes right-beating horizontal nystagmus (fast phase toward the lesion/pressure side), and releasing pressure causes transient left-beating nystagmus [8]. If the fistula involves a vertical canal, the nystagmus will have vertical and torsional components aligning with that canal's plane. Clinical references note: horizontal nystagmus alone on fistula test indicates a lateral (horizontal) canal fistula; torsional nystagmus with an upward slow phase (i.e. primarily down-beating, torsion present) suggests a superior canal dehiscence, whereas torsional nystagmus with a downward slow phase (primarily up-beating fast phase with torsion) suggests a posterior canal fistula or dehiscence [9]. In SCD specifically, pressure typically causes a mixed down-beating torsional nystagmus (fast phase down, top poles of eyes toward the affected ear) because of superior canal excitation [4, 10]. (Later in Section 5 we detail how Valsalva vs. tragal pressure affect SCD).



In summary, the fistula test is a direct probe for third mobile window lesions. A positive result (Hennebert's sign) is strong evidence of a peripheral vestibular abnormality – virtually all causes

are peripheral (inner ear) in origin [4]. There is no true central equivalent of a fistula sign because the mechanism requires an abnormal communication with labyrinthine fluid. Pressure-induced nystagmus is therefore highly localizing: if present, one should search for perilymphatic fistula, SCD, or similar inner ear pathology. Important caveats (pitfalls) include: (a) false-negatives – e.g. if the labyrinth is completely obliterated ("dead ear") or the fistula is plugged by tissue (a cholesteatoma may cover a canal fistula, preventing pressure transmission) the test may be negative despite a fistula [8]; and (b) false-positives – syphilis or Meniere's disease causing Hennebert's sign without a true fistula [8, 4]. Despite these, a Hennebert-positive nystagmus remains a critical bedside finding that often prompts confirmatory tests (like high-resolution temporal bone CT or exploratory surgery) [4].

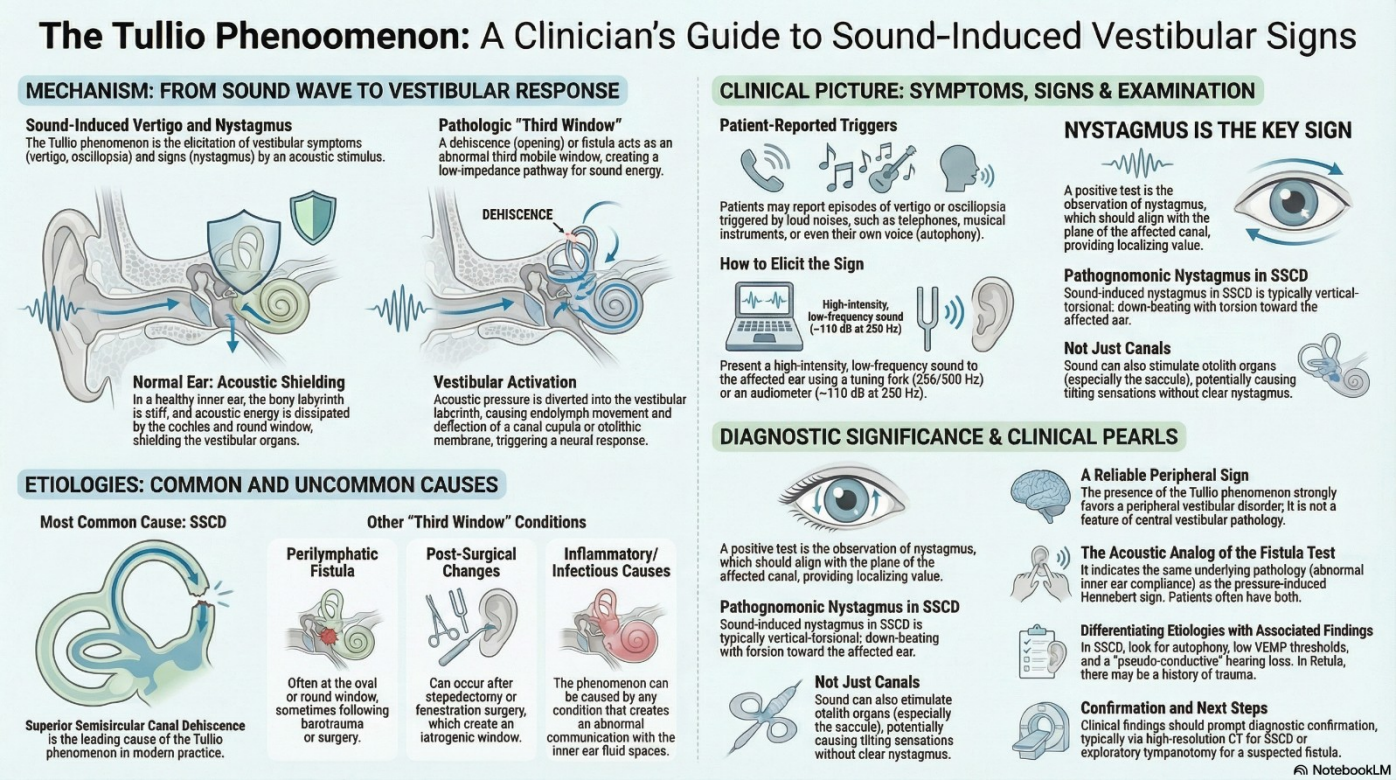
3.4 Acoustic (Sound) Stimuli (Tullio Phenomenon)

The Tullio phenomenon refers to nystagmus or vertigo provoked by sound. It was first described by Prof. Pietro Tullio in 1929 when he noted that creating small fenestrations in semicircular canals of pigeons caused them to exhibit head movements (and presumably nystagmus) in response to certain sounds [9]. In humans, pathological sound-induced vertigo was historically observed in patients with advanced syphilis (labyrinthine otosclerosis and fistulae) [9]. Today, the most common cause of Tullio's phenomenon is superior semicircular canal dehiscence (SSCD) syndrome [9, 10], though any condition that introduces an abnormal "third window" into the inner ear can cause it [9].

Mechanistically, intense sound (usually in the range of mid-to-low frequencies, delivered at high intensity) introduces an oscillatory pressure wave in the inner ear. In a normal ear, the round window and the cochlea dissipate most acoustic energy, and the vestibular organs are shielded by the stiffness of the bony labyrinth. But if there is a dehiscence (opening) in the bony capsule or a mobile "window" into the vestibule, some acoustic energy is diverted into the vestibular system, causing abnormal fluid motion in the canals or otoliths [9]. Essentially, the dehiscence acts as an alternative outlet for sound-induced pressure, leading to deflection of the cupula of a semicircular canal or displacement of otolithic membranes. For example, in superior canal dehiscence, loud sounds can cause endolymph movement in the superior canal (via the dehiscent area) as if the head were moving, thereby inducing nystagmus in the plane of that canal [10]. Similarly, in a large perilymph fistula at the oval window, acoustic energy might directly stimulate the vestibule (especially the saccule, which is sound-sensitive, as evidenced by vestibular-evoked myogenic potentials).

Clinically, a patient with Tullio phenomenon may report that loud noises (phones, musical instruments, even their own voice) trigger vertigo or oscillopsia. On examination, one can elicit sound-induced nystagmus by presenting a high-intensity sound stimulus to the affected ear – commonly done with a low-frequency tuning fork (e.g. 256 Hz or 500 Hz) placed near the ear or with an audiometric oscillator delivering a loud tone (e.g. ~110 dB at 250 Hz). A positive Tullio test is the observation of nystagmus coupled with patient's dizziness in response to the sound. The nystagmus characteristics will mirror those of the analogous pressure stimulation for that canal. In SSCD, sound typically causes a down-beating, torsional nystagmus (fast phase down, torsion toward affected ear) – essentially the same as the pressure-induced nystagmus for superior canal excitation [10]. This is considered pathognomonic for SCD when seen in the appropriate context [10]. Indeed, Minor's landmark 1998 paper described that in SCD patients, eye movements induced by sound or pressure align exactly with the plane of the dehiscent

superior canal [10]. If the sound instead induces a primarily horizontal nystagmus, one might suspect a lateral canal fistula or dehiscence.



It's worth noting that not all sound-induced vestibular responses are via canals – the otolith organs (particularly the saccule) have an intrinsic sound sensitivity (basis of the vestibular evoked potentials). In some conditions, a loud sound can cause predominantly translational or tilting sensations (otolithic symptoms) rather than a clear nystagmus. But in Tullio's phenomenon as clinically defined, we focus on observable nystagmus. Table 1 in Section 11 lists conditions known to cause Tullio phenomenon [9]. These include: SSCD, perilymphatic fistula, Ménière's disease (some Meniere patients with hydrops can have sound-induced dizziness, possibly from altered inner ear mechanics), congenital syphilis (due to labyrinthine bone erosion), post-stapedectomy or fenestration surgery (creating an iatrogenic window), and even rarely in otherwise "normal" individuals (perhaps at extremely high sound intensities) [9].

Importantly, Tullio's phenomenon is essentially the sound-frequency analog of the fistula test – both indicate an abnormal communication or compliance in the inner ear. A classic teaching is that when a patient presents with vertigo triggered by loud sounds, one should suspect superior canal dehiscence (especially if accompanied by conductive hearing loss that is actually "pseudo-conductive" from the third window) [10]. These patients often also have a positive Hennebert sign (pressure-induced nystagmus) [10] and other peculiar auditory symptoms like autophony (hearing one's own voice or heartbeat loudly) [10]. On the other hand, if a patient has had a recent stapes surgery or trauma, Tullio phenomenon could indicate a fistula at the oval or round window. The presence of Tullio phenomenon strongly favours a peripheral cause – it is not a feature of central vestibular disorders. A neurologic cause of sound-induced nystagmus is exceedingly rare (about the only central condition that can vaguely resemble it is a brainstem lesion causing hyperacusis with vestibular response, but this is not the classic Tullio). In differentiating aetiologies, the concurrent findings are helpful: for example, in SSCD you often

find a low-frequency conductive hearing loss and enhanced bone conduction (and low VEMP thresholds) [10]; in perilymph fistula, there might be a history of barotrauma or a fluctuating hearing loss; in syphilis or Meniere's, other signs of those diseases will be present.

In summary, sound-induced nystagmus (Tullio phenomenon) arises from pathologic acoustic activation of the vestibular system due to a third window or fistula. It underscores a peripheral pathology in essentially all cases. Its nystagmus aligns with the affected canal's orientation (e.g. vertical-torsional for superior canal) and thus provides localizing information similar to the fistula test [10]. Because sound and pressure often go hand-in-hand (patients with SCD or fistula often have both Tullio and Hennebert signs), they are sometimes collectively referred to as pressure/sound-induced vestibular symptoms. The diagnostic confirmation usually involves imaging (CT for SCD or exploratory tympanotomy for fistula) after these exam findings.

3.5 Internal Pressure Stimuli (Valsalva Manoeuvres)

Valsalva-induced nystagmus occurs when changes in internal pressure – either intrathoracic/intracranial or intranasal/middle ear pressure – provoke nystagmus. There are two classic forms of Valsalva manoeuvres relevant here, which must be distinguished:

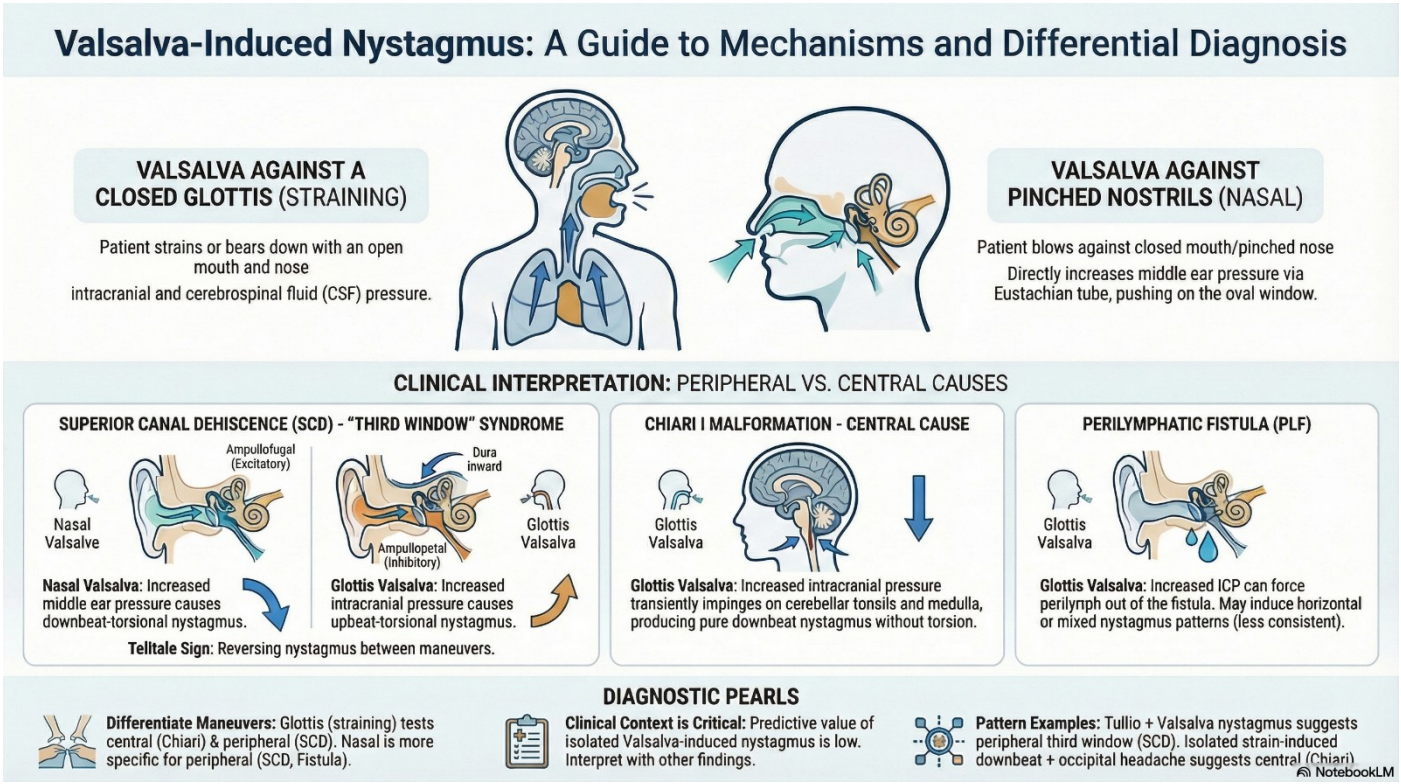
1. **Valsalva against a closed glottis:** The patient bears down or strains while keeping the mouth and nose open (essentially attempting exhalation against a closed airway). This dramatically increases intrathoracic pressure, which in turn impedes venous return and acutely raises intracranial pressure (ICP) by transmitted venous pressure. Importantly, in the context of vestibular effects, a closed-glottis Valsalva primarily increases intracranial and cerebrospinal fluid (CSF) pressure.
2. **Valsalva against pinched nostrils (closed nose):** The patient closes the nose and mouth and blows, as if trying to pop the ears. This manoeuvre directly increases middle ear pressure (and to a lesser degree intrathoracic pressure, but the emphasis is on middle ear via Eustachian tube). It forces air into the middle ear space, pushing the tympanic membrane and ossicles outward, elevating pressure on the oval window.

In patients with third window syndromes such as superior canal dehiscence, both types of Valsalva can induce nystagmus, but in opposite directions due to the different pressure vectors [10, 11]. Using SCD as the exemplar: A nasal Valsalva (increasing middle ear pressure) pushes the stapes footplate inward, analogous to a positive external pressure. As described earlier, for a dehiscent superior canal this causes ampullofugal endolymph movement and excitation of the superior canal, yielding nystagmus with slow phases upward and away from the affected ear (fast phase down toward the affected ear) [11]. By contrast, a glottis Valsalva (increasing intracranial pressure) causes the dural lining of the dehiscent canal to bulge inward, effectively pushing endolymph in the opposite direction (ampullopetal relative to the canal) [11]. This produces an inhibitory stimulus to that canal and a nystagmus in the same plane but opposite direction – in SCD, that means an up-beating torsional nystagmus (fast phase up, torsion opposite to what the excitatory was) [11]. In Minor's original report, patients with SCD exhibited downbeat-torsional nystagmus with nose-pinched Valsalva and upbeat-torsional nystagmus with glottis Valsalva [11]. This reversal with the two types of Valsalva is a telltale sign of a third window like SCD [11].

However, Valsalva-induced nystagmus is not exclusive to peripheral lesions. The notable central cause is the **Arnold-Chiari I malformation** (cerebellar tonsillar ectopia). Patients with Chiari often experience cough-induced or strain-induced vertigo, and on examination they may have

downbeat nystagmus elicited by Valsalva (particularly by straining) [19]. The mechanism in Chiari is different: increased ICP transiently impinges on the cerebellar tonsils and medulla, often affecting the caudal vestibular nuclei or their connections, leading to a transient downbeat nystagmus (fast phase downward) – a central ocular motor pattern. No third window is present; it's a direct central compression effect. Thus, Valsalva-induced downbeat nystagmus without torsion is a red flag for a central cause like Chiari (especially if accompanied by occipital headache) [19].

Other conditions to consider: A perilymphatic fistula can also respond to Valsalva manoeuvres. For example, a patient with a PLF might get dizzy with coughing, sneezing, or lifting (internal pressure events) – these are in fact recognized triggers for fistula symptoms [9]. What happens is that raised intracranial pressure via the cochlear aqueduct can push on the perilymph and force fluid out of the fistula (an "explosive" route of PLF formation per Goodhill's classification [9]). In such cases, a straining Valsalva might induce nystagmus similar to pressing on the tragus (if the fistula valve is open). In practice, one might see horizontal nystagmus or mixed patterns with Valsalva in a fistula patient, although it's less consistent than targeted tragal pressure.



From a classification perspective, we categorize Valsalva as "internal pressure" stimulus because it relies on pressure changes transmitted internally rather than externally. It is often considered alongside Tullio and Hennebert as part of pressure-induced vestibular phenomena. In fact, some sources lump them together when teaching "sound- and pressure-induced vertigo" signs [10]. But differentiating the type of Valsalva is crucial clinically: Valsalva (glottis) primarily tests for central pressure-sensitive causes (Chiari) and also contributes to identifying SCD (with its inverse response), whereas Valsalva (nasal) is more specific for peripheral causes like SCD or fistula (similar effect as direct external pressure) [10, 11].

The predictive value of Valsalva-induced nystagmus for central vs peripheral causes is considered low in isolation [25]. It must be interpreted in context. For example, if a patient has

both Tullio phenomenon and Valsalva-induced nystagmus, that combination strongly suggests a peripheral third window (like SSCD) [25]. If a patient only has Valsalva (strain)-induced downbeat nystagmus and no sound/pressure signs, a central cause (Chiari) is more likely [25].

In summary, internal pressure changes from Valsalva manoeuvres can provoke nystagmus by either transmitting pressure to an inner ear lesion (peripheral mechanism, as in SCD or PLF) or by compressing brainstem structures (central mechanism, as in Chiari). The direction of nystagmus and whether it appears with glottis or nasal Valsalva (or both) provides diagnostic clues. Valsalva-induced nystagmus is an important part of the test battery for patients with exertional or pressure-related dizziness, complementing the external pressure (fistula) and sound tests.

Clinical Examination Methodology

Performing these provocation tests properly is key to obtaining reliable results. Below we outline the methodology for each test, including patient instructions, optimal techniques, and precautions. It is assumed the patient has been informed about the manoeuvres and potential to induce dizziness, and appropriate safety measures (such as having the patient seated or supported) are in place.

General setup: Ideally, vision should be denied or minimized during these tests to accentuate nystagmus (prevent fixation suppression). Using Frenzel goggles or infrared video-oculography goggles is highly recommended [18]. These tools magnify the patient's eyes and prevent visual fixation, making even subtle nystagmus visible to the examiner [18]. If such equipment is not available, performing the manoeuvres in a dimly lit room and asking the patient to keep eyes open (looking straight ahead) or using an ophthalmoscope to observe one eye can also work.

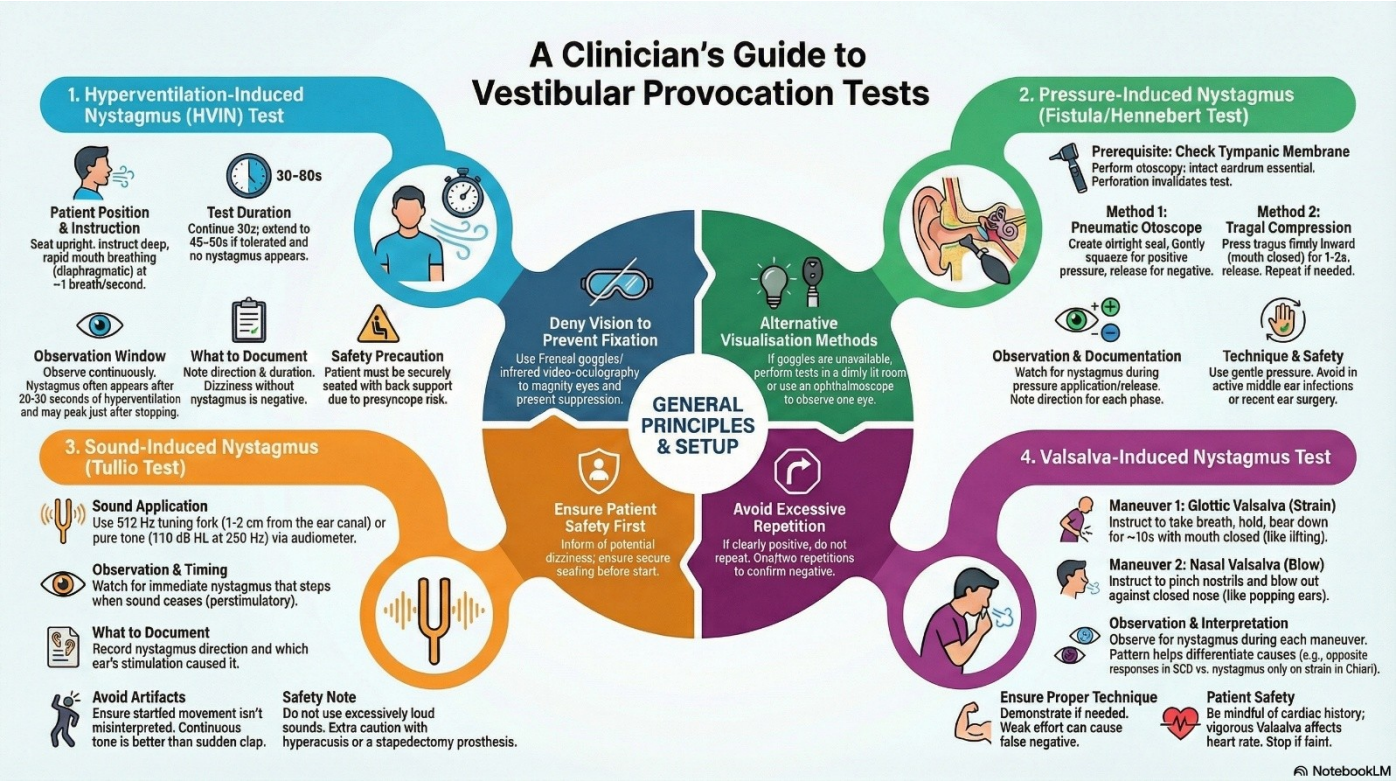
1. **Hyperventilation-Induced Nystagmus (HVIN) Test:** Have the patient sit upright (to avoid any orthostatic effect) and remove any vision (Frenzel goggles on). Instruct the patient to breathe deeply and rapidly through the mouth. A common protocol is **one breath per second for about 30 seconds;** some protocols go up to 60–90 seconds if tolerated [2]. It's important the patient uses diaphragmatic breathing to really exchange volumes; shallow panting is less effective than deep breaths. The examiner should observe the eyes continuously during and immediately after the hyperventilation period. Nystagmus usually does not begin instantly; often it appears after ~20–30 seconds of hyperventilation and may peak at the end of the stimulation or just after stopping. If no nystagmus is seen at 30 seconds, one may extend to 45–60 seconds but caution the patient to stop if significant light-headedness or tingling occurs. Safety: There is a risk of presyncope from hyperventilation, so ensure the patient is secure (seated with back support). Following the hyperventilation, allow the patient to rest and recover normal breathing. If a nystagmus is observed, note its direction (e.g. "right-beating horizontal with slight torsion") and duration. Often HVIN fades within 15–30 seconds after cessation of breathing, though it can sometimes last a minute or two. Also note any subjective symptoms (did the patient feel vertigo or just light-headed?). Sometimes patients with anxiety or no vestibular lesion will feel dizzy during hyperventilation, but they will not exhibit nystagmus – this would be a negative HVIN test, indicating the hyperventilation did not unmask a vestibular imbalance [2].
2. **Pressure-Induced Nystagmus (Fistula/Hennebert test):** Two methods can be used: (1) using a pneumatic otoscope (Siegle's speculum) to deliver controlled puffs of positive

and negative pressure to the sealed ear canal, or (2) using the examiner's thumb or finger on the tragus to alternately press and release (the tragal compression technique). Start with the patient seated and wearing Frenzel goggles. First, examine the ears otoscopically to ensure the tympanic membrane is intact – a perforation would invalidate the typical fistula test and can itself cause thermal nystagmus if air is flushed across (so avoid strong air pressure if perforation is present). To use the pneumatic otoscope, insert it in the ear canal to get an airtight seal; then gently increase pressure (squeeze bulb) while watching the patient's eyes for nystagmus. After a second or two of pressure, release or even apply slight negative pressure (sucking action) and watch for nystagmus reversing. Tragal method: Ask the patient to keep mouth closed (to avoid venting air through the Eustachian tube). Press the tragus inward firmly for 1–2 seconds (this increases canal pressure), then let go. Repeat this a few times if no immediate nystagmus, as sometimes a few pulses are needed [4]. Observe for any nystagmus during each press and release. If the patient reports dizziness or you see nystagmus, that's a positive sign. Note the direction of nystagmus for each phase: for example, you might record "pressure-in induced a torsional nystagmus beating toward the right ear, pressure release caused left-beating reversal." Also inquire if it reproduces the patient's symptom (often they'll say "yes, that made me dizzy like my spells"). Safety and technique tips: Be gentle; you don't need excessive force – a moderate pressure is sufficient. Avoid performing this if the patient has an active middle ear infection or recent middle ear surgery unless necessary, as it could induce pain or inner ear fluid shifts. If both ears are to be tested, do the symptomatic ear first (to avoid confusion or fatigue). A common pitfall is a faint endpoint nystagmus from the patient's gaze– ensure the eyes are in neutral primary position during the test.

3. **Sound-Induced Nystagmus (Tullio test):** Traditionally, this can be done with a high-output tuning fork (512 Hz or 256 Hz) or an electronic audio source. One simple bedside way: take a 512 Hz tuning fork, strike it, and hold the vibrating tines about 1–2 cm from the patient's ear canal. An even simpler trigger (though less frequency-specific) is to have the patient hum or speak loudly, or to clap hands near the ear – but these are less controlled. Ideally, if an audiometer is available, present a pure tone at e.g. 110 dB HL at 250 Hz via headphone to the suspected ear. Under Frenzel goggles observation, watch for nystagmus onset coincident with the sound. True Tullio phenomenon will often occur almost immediately with the sound and stop when the sound stops (perstimulatory nystagmus). Document the direction of nystagmus and which ear's stimulation caused it. For instance, "Loud 500 Hz tone to the right ear produced down-beating, right-torsional nystagmus lasting for the duration of the sound." Ensure the patient is not startled or blinking – those can cause movement artifacts misinterpreted as nystagmus; that's why a continuous tone or a prolonged tuning fork is better than a sudden loud clap. Precaution: Do not use extremely loud noises that could damage hearing; if using a tuning fork, do not press it against the skull (that would also introduce bone vibration and complicate interpretation). The intensity should be enough to be uncomfortable but not painful. If the patient already has hyperacusis or a stapedectomy prosthesis, be extra cautious. Usually, one or two trials per ear suffice.

4. **Valsalva-Induced Nystagmus Test:** This requires patient participation. It's done in two parts to differentiate the two types of Valsalva. First, instruct the patient how to do a strain: "Take a breath, hold it, and bear down as if you're lifting a heavy weight or having a bowel movement – keep your mouth closed and don't let air out." Some examiners have the

patient plug their nose with their fingers for reinforcement. Observe the eyes while the patient strains for ~10 seconds (or less if nystagmus appears sooner or patient gets symptomatic). Often, if positive, nystagmus will start a few seconds into the strain. Then have the patient relax. Next, test the "nasal Valsalva": have them pinch their nostrils closed, close their mouth, and "blow out" (like trying to pop ears) for a few seconds. Again, watch the eyes during this. (If the patient cannot do these on their own, the examiner can simulate an intracranial pressure increase by doing jugular vein compression – pressing on the neck veins – which is rarely done in practice but mentioned in literature for SCD testing [11].) Note any nystagmus with either manoeuvre and its direction. Typically, you might see something like: "On glottic Valsalva (strain), patient developed down-beating nystagmus; on pinched-nose Valsalva, no nystagmus." That pattern would point to a central cause (Chiari). Or in SCD: "Strain Valsalva caused up-beating right-torsional nystagmus, whereas pinched-nose Valsalva caused down-beating right-torsional nystagmus" – an opposite response indicative of superior canal dehiscence [11]. Precautions: Make sure the patient doesn't hyperventilate while doing it; sometimes they take a big breath and blow off CO_2 (could confound with HVIN). Also, not every patient can execute a Valsalva effectively; if they have weak effort, the test might be falsely negative. It's helpful to demonstrate if needed. As always, be mindful if the patient has any cardiac history – a vigorous Valsalva can affect heart rate; usually not an issue in these short tests, but if the patient feels faint, have them stop.



For all tests, avoid excessive repetitions. If a manoeuvre is clearly positive, you usually do not need to repeat it multiple times (doing so could risk inducing more symptoms or fatigue the response). On the other hand, if negative, one might repeat once or twice to be sure, especially HVIN (since sometimes a subtle nystagmus might be missed on the first try). Always compare sides when applicable: HVIN inherently affects the side of lesion (direction of nystagmus tells side), whereas fistula and sound tests should be done on each ear individually to compare.

Throughout these examinations, maintain a systematic approach: observe baseline eye movements first (ensure no spontaneous nystagmus or other confounder), then apply the stimulus, then observe post-stimulus. Good documentation is important – describe the nystagmus in all three components (horizontal, vertical, torsional), the latency (immediate or delayed), duration, and whether the patient's symptoms correlated. Many of these tests can be augmented by quantitative recording (see Instrumented Assessment section), but at bedside, it's largely an observational art.

In summary, the methodology is straightforward but requires practice to recognize the often-subtle induced nystagmus. Proper technique and patient cooperation are essential. Table 4 (Pitfalls) later will highlight common mistakes and confounding factors (for example, Valsalva can induce a mild horizontal gaze nystagmus in normal individuals due to strain – one should differentiate that from a true pathologic response). Mastering these manoeuvres adds a powerful dimension to the vestibular examination, often yielding immediate clues to diagnoses that would otherwise need more expensive testing.

Interpretation Framework

Interpreting provocation test results involves linking the observed nystagmus pattern with the underlying vestibular anatomy and pathology. Here we outline a framework to systematically interpret each test's outcome, focusing on key questions: Is the test positive or negative? If positive, what is the direction and characteristics of the nystagmus? What does that imply about localization (which ear, which canal or structure)? And how do we integrate that with the clinical context?

1. **Determine if the response is truly positive:** A "positive" test means nystagmus (and usually vertigo) has been elicited by the manoeuvre. Because these stimuli can sometimes cause non-specific effects (e.g. hyperventilation causing lightheadedness, or a loud sound causing a startle blink), it's important to verify that the eye movements observed are consistent and repeatable with the stimulus. Typically, a few beats of nystagmus in the appropriate direction, especially if accompanied by the patient's typical dizziness, constitutes a positive result. If the patient just reports a sensation without any ocular movement, it may be a subjective response but not a full "sign" (e.g. Hennebert's symptom vs sign). The presence of nystagmus is more objective.
2. **Characterize the nystagmus three-dimensionally:** Note the direction of the fast phase (horizontal right/left, up, down, torsional clockwise or counterclockwise from the examiner's perspective). Also note intensity (e.g. slow phase velocity) if possible – a very robust nystagmus (clearly visible) vs. a faint one. A very delicate nystagmus might suggest a minor asymmetry or early lesion, whereas a robust one often indicates a larger lesion or acute issue.
3. **Assign the nystagmus to a likely anatomical origin:** Use known patterns:
    - **Horizontal (horizontal-torsional) nystagmus** provoked by these tests usually implicates the horizontal semicircular canal or lateral vestibular nerve asymmetry. For example, HVIN that is purely horizontal suggests primarily a lateral canal or nerve imbalance (which aligns with Minor's finding that horizontal canal fibres dominate HVIN [1]). Pressure or sound-induced horizontal nystagmus suggests a lateral canal fistula or horizontal canal dehiscence. The fast phase direction will be toward the excited side (e.g. pressure-induced horizontal nystagmus beating

toward the affected ear means ampullopetal flow in that horizontal canal, an excitatory response) [6, 4].

- **Vertical-torsional nystagmus** indicates involvement of a vertical canal (anterior or posterior) or the central vestibular pathways controlling vertical eye movements. Down-beating torsional (with torsional component pointing toward one ear) strongly points to the superior (anterior) canal on that side being excited – classic for SCD or superior canal fistula [10]. Up-beating torsional (often torsion to the opposite side) could mean a posterior canal excitation on the side of torsion. One can use Ewald's second law: ampullopetal flow is excitatory in horizontal canals but ampullofugal is excitatory in vertical canals. In a superior canal dehiscence, positive pressure causes ampullofugal (excitation) – nystagmus indicates excitation (downbeat torsional), whereas negative pressure causes ampullopetal (inhibition) – nystagmus flips (upbeat opposite torsion) [10, 11]. If the pattern is pure vertical without torsion, consider a central cause (downbeat on Valsalva suggests central).

4. **Duration and latency:** A provoked nystagmus usually starts within seconds of the stimulus and is transient. If nystagmus persists longer than the stimulus (except a few seconds after, which is normal), one should be cautious – e.g. a head-shaking nystagmus should fade within ~30 sec, a hyperventilation nystagmus usually fades in <1 minute.
5. **Determine the likely side (laterality):** In general, the fast phase of the evoked nystagmus beats toward the more active or affected side for peripheral lesions:
    - In HVIN for vestibular schwannoma or neuritis, the fast phase is toward the lesion side (ipsilesional) [1].
    - In the fistula test, pressure applied drives nystagmus toward the affected ear for an excitatory lesion [8]. Reversal on release confirms the same side.
    - For Tullio phenomenon, similarly, the ear exposed to sound that triggers nystagmus is the affected side.
    - For Valsalva: if both Valsalva types yield nystagmus but opposite directions, that implicates one side (the side toward which the torsional component is directed during excitatory phase) – i.e. SCD side.
6. **Integrate with clinical context:** Correlate with symptoms, other exam findings (like Head Impulse Test or calorics), hearing loss [16], and imaging [4, 9, 10].
7. **Differentiate peripheral vs. central patterns:** Peripheral nystagmus tends to be mixed horizontal-torsional and obey Alexander's law, while central might be pure vertical or direction-changing [25].

To streamline interpretation, some clinicians use a flowchart approach:

- If HVIN present, check if ipsilesional or contralesional. Ipsilesional HVIN + hearing loss = strong evidence of vestibular nerve lesion [16].
- If fistula test positive, localize side and canal [4]. Then look for cause (e.g., cholesteatoma).
- If Tullio positive, assume third window [9].

- If Valsalva positive: glottis strain only (downbeat) = Chiari; opposite directions for both types = SCD [11].

Finally, always interpret these tests in light of their sensitivity and specificity limitations. A negative test does not rule out pathology, and a positive test strongly suggests pathology but requires corroboration.

Central versus Peripheral Differentiation

A critical part of analysing provoked nystagmus is determining whether the findings point to a peripheral vestibular lesion or a central (brainstem/cerebellar) disorder. Differentiating central vs peripheral is important for patient management. Here we outline distinguishing features for each test:

Hyperventilation-Induced Nystagmus (HVIN): By default, HVIN is usually thought of as a peripheral sign. However, central lesions like multiple sclerosis plaques or vestibular paroxysmia [3, 21] can also cause HVIN.

- **Direction:** Peripheral HVIN typically yields a unidirectional horizontal nystagmus. Central nystagmus might be pure vertical or direction changing [25].
- **Associated signs:** If HVIN is peripheral, other peripheral signs often coexist. If it's central, the head impulse test might be normal.
- **Symmetry:** HVIN in central lesions could be bilateral or variable [2]. However, an “ipsilesional HVIN” is specifically noted as a red flag for vestibular schwannoma [16].

Pressure/Sound-Induced Nystagmus (Hennebert/Tullio): These are almost exclusively peripheral phenomena [25].

- **Exceptions:** Arnold-Chiari causes nystagmus with increased intracranial pressure (strain), not typically with pressing on the ear canal or loud sound [19].
- **Summation:** If pressure or sound induces nystagmus, think peripheral. The Tullio phenomenon is often neglected in neurological presentations because its cause is almost always peripheral [10].

Valsalva-Induced Nystagmus: Here the central vs peripheral question is very pertinent.

- **Distinguishing:** If only the straining Valsalva (glottis closed) yields downbeat nystagmus, consider central (Chiari) [19].
- **Peripheral pattern:** If the patient has both types of Valsalva positive with opposite directions, that fits peripheral third window (SCD) [11].

Generally, peripheral nystagmus suppresses with fixation and follows Alexander's law, while central nystagmus might be pure vertical and less effectively suppressed by fixation [25]. Combinations like Valsalva + Tullio strongly favour peripheral (SSCD) [25]. Lastly, consider other findings: a peripheral cause might have a head impulse deficit (vHIT), whereas a central cause like Chiari would have normal vHIT but abnormal downbeat on fixation removal [25].

Clinical Application Scenarios

To ground the above concepts in real-world practice, here we present several clinical scenarios:

- **Scenario 1: Acoustic Neuroma Suspected** – A 55-year-old with gradual hearing loss. Hyperventilation triggers a right-beating nystagmus. This ipsilesional HVIN strongly suggests a right vestibular nerve lesion [1, 16].
- **Scenario 2: Vestibular Neuritis Compensation Monitoring** – A 40-year-old recovering from neuritis. One month later, HVIN briefly appears, indicating latent imbalance and ongoing compensation [2].
- **Scenario 3: Superior Canal Dehiscence (SCD) Syndrome** – A 35-year-old with vertigo from loud sounds and straining. Tullio test is positive (down-beating torsional nystagmus), and Valsalva shows opposite responses for strain vs. nasal blow. Pathognomonic for SCD [10, 11].
- **Scenario 4: Perilymphatic Fistula (Oval Window Rupture)** – A 28-year-old diver with post-barotrauma vertigo. Tragal pressure induces nystagmus (Hennebert sign). Strong evidence of PLF [4, 6].
- **Scenario 5: Arnold-Chiari I Malformation** – A 30-year-old with oscillopsia on coughing. Glottis Valsalva reproduces pure down-beating nystagmus, while nasal Valsalva is negative. Strongly suggests Chiari [19, 25].
- **Scenario 6: Vestibular Paroxysmia (Neurovascular Compression)** – Short vertigo attacks. HVIN triggers a typical episode, supporting the diagnosis of a hyperexcitable nerve [3].
- **Scenario 7: False-Positive Hennebert Sign in Meniere's Disease** – Pressure induces mild nystagmus despite no dehiscence; likely due to fibrous bands [15].

Each of these scenarios illustrates how provocation tests are applied to tilt the diagnostic balance and direct appropriate investigations.

Instrumented and Laboratory Assessment

Modern vestibular assessment can augment bedside manoeuvres with instrumentation:

- **Video-oculography (VOG) / Electronystagmography (ENG):** Recording eye movements during HVIN or fistula tests allows for quantifying subtle nystagmus and measuring slow-phase velocity [2].
- **Skull Vibration:** Labs use dedicated devices (100 Hz) to monitor vibration-induced nystagmus via VOG [18].
- **Audiometric and Evoked Potential Correlates:** Vestibular Evoked Myogenic Potentials (VEMP) detect third-window syndromes by showing low thresholds [10]. Electrocochleography (ECoG) may show patterns in fistula or hydrops [15].
- **Imaging:** High-resolution CT is mandatory if sound/pressure tests are positive [10]. MRI is indicated if a central cause is suspected.
- **Specialized protocols:** Many labs use VNG for HVIN and Valsalva to ensure consistency and normative data [2, 11].
- **Integration:** If vHIT is normal but pressure tests are positive, suspect a third window [10, 13].

Summary of instrumented assessment: VOG/ENG improves sensitivity and allows for documentation, while VEMP and imaging serve as confirmatory tools for the findings discovered at the bedside.

Advanced Insights, Controversies, and Evolving Concepts

Our understanding of these tests continues to evolve:

- **HVIN Mechanism:** Debate remains whether HVIN is a direct peripheral nerve effect [3] or a central compensation issue [2]. Some literature suggests its sensitivity for schwannoma might be lower than previously thought [1, 16, 2].
- **Hennebert Sign Pathophysiology:** Recent work suggests pressure might predominantly activate specific regular vestibular afferent subsets [4, 10].
- **Perilymphatic Fistula (PLF) Diagnosis:** The fistula test is known to be neither perfectly sensitive nor specific [6, 15]. New assays like cochlin-tomoprotein or endoscopic visualization are being researched [4].
- **Skull Vibration for SCD:** SVIN is emerging as a consistent sign of third-window syndromes [18].
- **Magnetic Vestibular Stimulation:** Strong magnetic fields in MRI machines can induce nystagmus [7].
- **Vestibular Paroxysmia:** The fact that HVIN triggers episodes in VP reinforces the use of carbamazepine for treatment [3].

As technology provides new tools like VEMP or high-res imaging, we both validate and refine the interpretations of provoked nystagmus.

Summary of Clinical Utility

Special oculomotor provocation tests hold significant utility:

1. **Enhanced Diagnostic Sensitivity:** Unmask latent dysfunction missed by routine exam [2, 4].
2. **Localization and Differentiation:** Distinguish SCD from horizontal fistula [10] and peripheral nerve lesions [1].
3. **Quick and Non-Invasive:** Cost-free bedside manoeuvres.
4. **Risk Stratification:** Vertical head-shake nystagmus suggests central lesions [25].
5. **Monitoring and Prognosis:** Repeated tests monitor disease progression or surgical success.
6. **Guiding Management:** Targeted diagnosis leads to appropriate surgical or medical intervention.
7. **Educational and Psychological Value:** Demonstrates physiology for trainees and validates the patient's experience.

When performed and interpreted correctly, these tests provide the critical evidence needed to coalesce disparate symptoms into a clear vestibular syndrome.

Table 1: Definitions of Special Provocation Tests

Test & Stimulus Type	Procedure	Positive Response	Typical Use Cases
HVIN (Respiratory)	Rapid deep breathing (30–60 s) [2].	Nystagmus (usually horizontal) [1].	Schwannoma, Neuritis, MS [2, 16].
Fistula/Hennebert (Pressure)	Tragal or pneumatic pressure [4].	Nystagmus and vertigo [4].	PLF, SCD, Syphilis [4, 8, 15].
Tullio (Acoustic)	Loud sound exposure [9].	Sound-induced nystagmus/vertigo [9].	SCD, PLF, Syphilis [9, 10].
Valsalva (Internal Pressure)	Glottis strain or nasal blow [10].	Manoeuvre-provoked nystagmus [25].	SCD, Chiari, PLF [11, 19, 25].
Head-Shaking (Mechanical)	Horizontal shake ~20 times [4].	Post-shake transient nystagmus.	Unilateral loss, Central [25].
Vibration-Induced (Mechanical)	100 Hz vibration to skull [18].	Nystagmus during vibration [18].	Unilateral loss, SCD [18].

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Accuracy and Currency

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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