VOR Suppression Testing:
A Comprehensive Clinical Review

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How to Use This Review

This document is the companion clinical literature review to the VOR suppression and cerebellar floccular 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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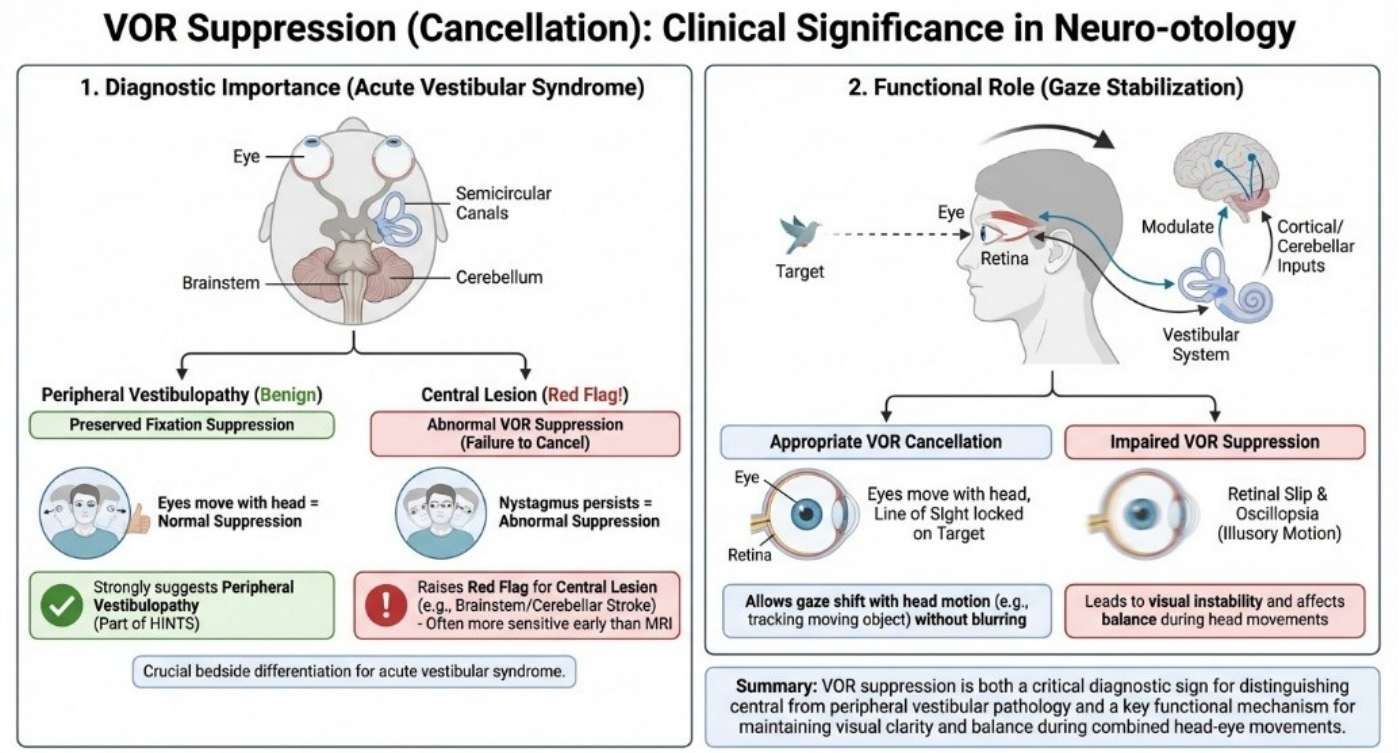
Vestibulo-Ocular Reflex (VOR) Suppression

A Deep Dive

1. Introduction

The ability to suppress the vestibulo-ocular reflex (VOR) – sometimes termed VOR cancellation – is a critical clinical sign in neuro-otology. In the context of an acute vestibular syndrome, a patient who cannot suppress vestibular nystagmus with visual fixation (i.e. who shows persistent nystagmus despite looking at a target) raises an immediate red flag for a central lesion, whereas preserved fixation suppression strongly suggests a peripheral vestibulopathy. This principle is incorporated in acute bedside algorithms like HINTS (Head-Impulse, Nystagmus, Test-of-Skew). In fact, an abnormal VOR suppression (failure to cancel the reflex with fixation) is often more sensitive in the first 1–2 days of a brainstem/cerebellar stroke than early MRI, underscoring its clinical importance. Thus, evaluating VOR suppression – for example, by asking the patient to fixate on their thumb while rotating en bloc (the classic "thumbs test") – is a crucial step in differentiating central vs. peripheral vestibular disorders at the



bedside.

Beyond diagnosis, VOR suppression plays a role in functional gaze stabilization. Many daily activities require one to shift gaze with head motion – for instance, tracking a moving object while turning one's head – without blurring vision. The ability to appropriately cancel the VOR in these contexts allows the eyes to move with the head (instead of reflexively opposite) so that the line of sight remains locked on the target [1]. Impairment of this mechanism can lead to retinal slip and oscillopsia (illusory motion of the visual scene) during head movements. In summary, VOR suppression is not only a diagnostic sign distinguishing central vestibular pathology, but also a key component of gaze stability that, when deficient, can profoundly affect a patient's visual clarity and balance.

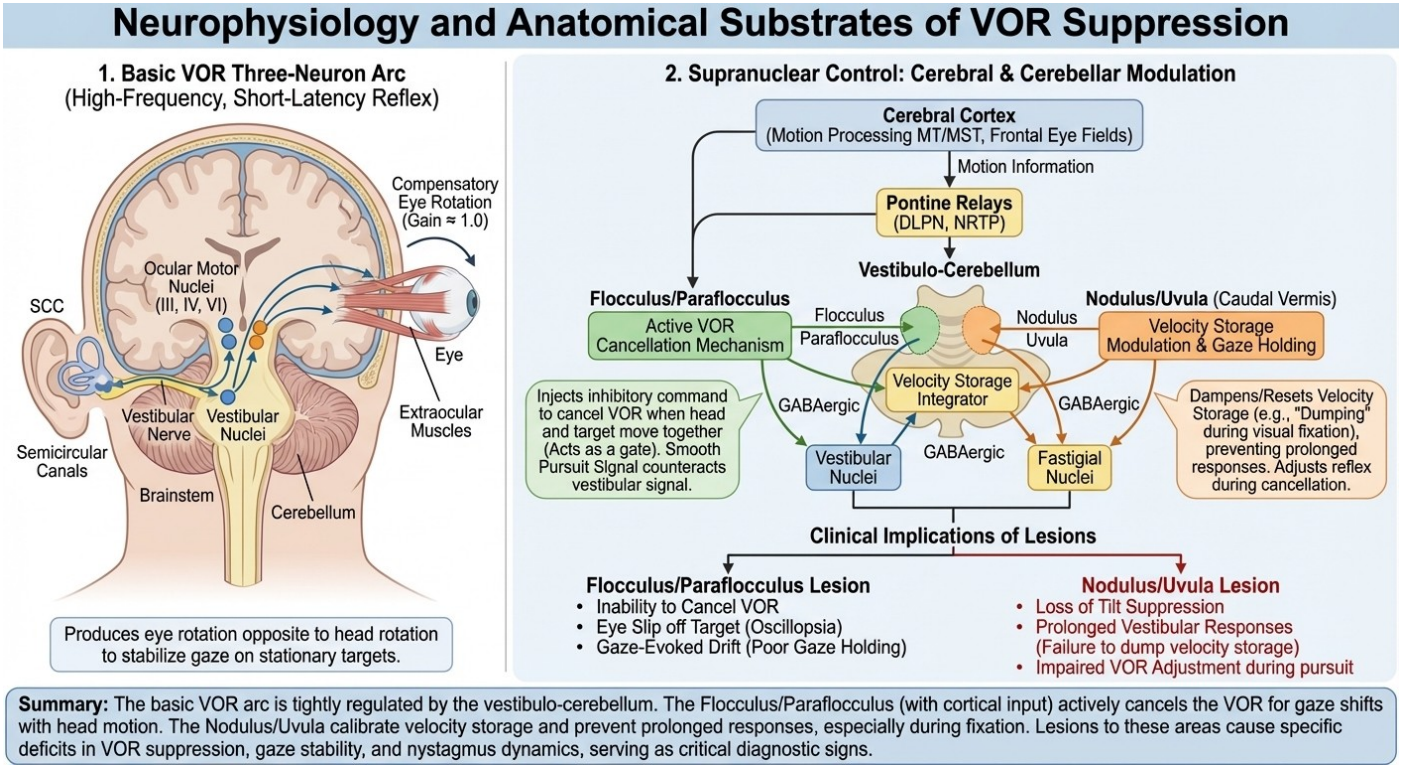
2. Neurophysiology and Anatomical Substrates

The basic VOR pathway is a short three-neuron arc: primary afferents from the semicircular canals (in Scarpa's ganglion) project via the vestibular nerve to the vestibular nuclei in the brainstem, and secondary neurons from the vestibular nuclei relay to the ocular motor nuclei (III, IV, VI) to drive the extraocular muscles [2]. This circuitry produces compensatory eye rotation

opposite to head rotation, with a velocity gain ideally near 1.0 (eye speed equals head speed, in opposite direction). In isolation, the primitive VOR is a high-frequency, short-latency reflex optimized to stabilize gaze on stationary targets [2].

Cerebral and cerebellar modulation overlays this reflex arc to allow more nuanced control. The vestibulo-cerebellum, particularly the flocculus and ventral paraflocculus, plays a pivotal role in tuning the VOR's gain and enabling its suppression [1, 2]. The flocculus receives motion information from visual cortex via pontine relays (e.g. DLPN (dorsolateral pontine nucleus) and NRTP (nucleus reticularis tegmenti pontis)) and can act as a gate: it injects an inhibitory command to cancel the VOR when head and target move together [1]. Lesions of the flocculus/paraflocculus cause an inability to cancel the VOR and result in "eye slip" off the target during head movement [3]. Indeed, floccular Purkinje cells normally fire in relation to head velocity and visual feedback, effectively superimposing a smooth pursuit signal that counteracts the vestibular signal during VOR suppression tasks [3, 4].

Another crucial cerebellar region is the nodulus and uvula (caudal vermis). These midline structures govern the so-called velocity storage integrator – a network in the brainstem (vestibular nuclei) that prolongs the VOR response at low frequencies [2, 4]. The nodulus/uvula act to dampen or reset velocity storage, particularly during visual fixation. For example, when a person stops rotating, an intact nodulus helps quickly extinguish any residual nystagmus (a process known as "dumping" velocity storage). Lesions here abolish the normal decay of nystagmus with fixation (so-called loss of tilt suppression of the VOR) and lead to abnormally prolonged vestibular responses [5]. In essence, the cerebellar vermis acts as a brake on the vestibular integrator, preventing excessive or sustained eye drift when it's not needed. Notably, animal studies indicate the uvula (nodulus) also sends inhibitory signals to vestibular and fastigial nuclei specifically during VOR cancellation, adjusting the reflex as part of the pursuit system [4].

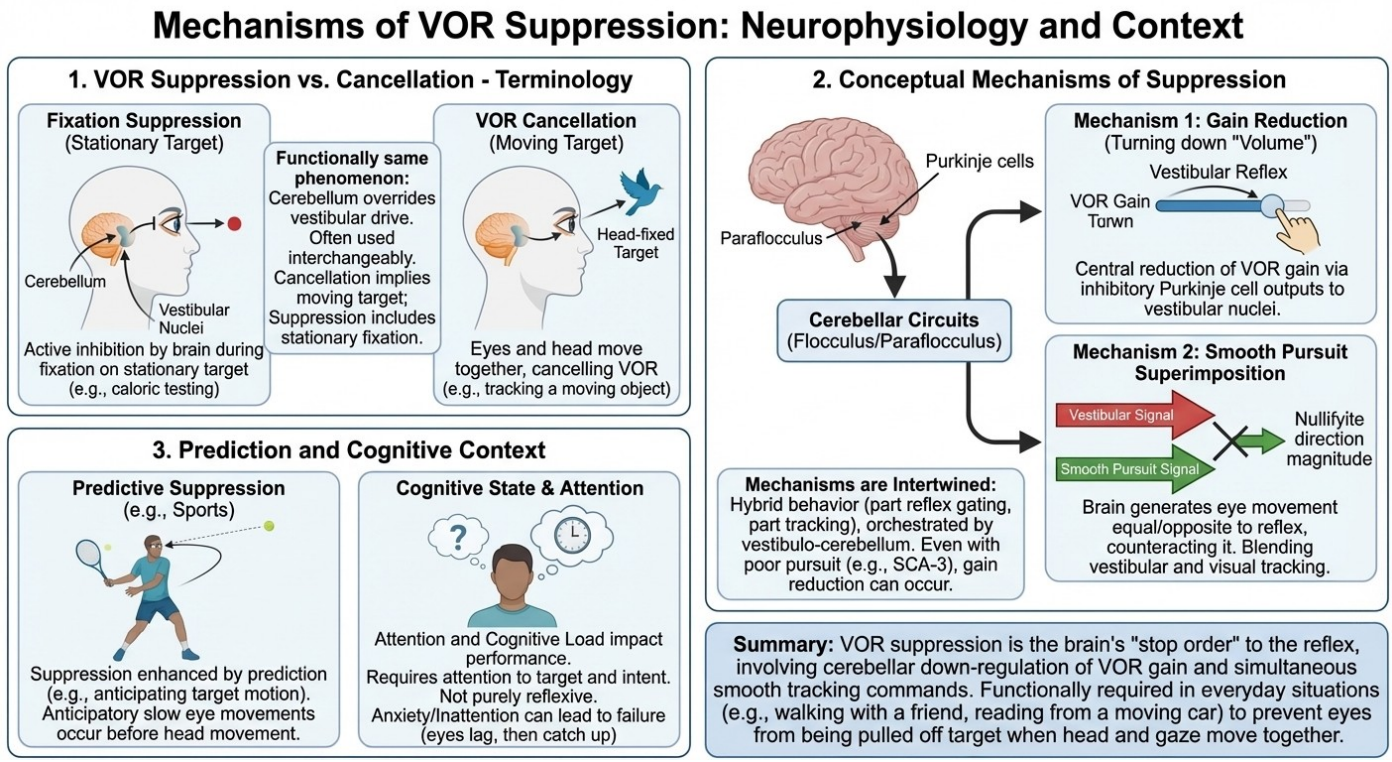


In summary, the core VOR neural substrate (semicircular canals → vestibular nuclei → ocular motoneurons) is under strong supranuclear control. The flocculus/paraflocculus (with input from cortical motion-processing areas MT/MST and frontal eye fields) provides the mechanism to actively suppress or cancel the reflex [1]. The nodulus/uvula calibrate the persistence of vestibular responses (velocity storage) and interact with the flocculus to ensure that when we fixate a target, any ongoing vestibular nystagmus is promptly terminated. Additionally, the flocculus is part of the neural integrator for gaze holding [1] – linking VOR suppression with gaze stability: damage to this system results not only in poor VOR cancellation but also gaze-evoked drift (because the eye can't hold steady when the reflex is disengaged).

3. Mechanisms of VOR Suppression

VOR Suppression vs. Cancellation – Terminology: In practice, "VOR suppression" and "VOR cancellation" are often used interchangeably, both referring to the active inhibition of the reflex by the brain. Some clinicians use "fixation suppression" to specifically denote the reduction of nystagmus when a patient fixates a stationary target (for example, during caloric or post-rotational testing) [6]. In contrast, "VOR cancellation" usually describes the scenario of a patient tracking a head-fixed target during head movement – that is, the eyes and head move together, cancelling out the VOR response [7]. Functionally these are the same phenomenon (both require the cerebellum to override the vestibular drive), but it's useful to remember that even a stationary target can suppress vestibular nystagmus via fixation, whereas the term cancellation implies a moving target that the patient follows with head and eyes. In this review, we will use the terms interchangeably, with context, since the neurophysiology underlying both is shared.

How is the VOR suppressed? Two conceptual mechanisms have been proposed: (1) the VOR's gain can be centrally reduced (i.e. turning down the reflex "volume"); and (2) a smooth pursuit eye movement can be superimposed in the opposite direction to counteract the reflex [4]. In reality, these mechanisms are intertwined – both mediated by the cerebellar circuits. To cancel the VOR, the brain must generate an eye movement equal in magnitude and opposite in direction to the reflex-driven eye movement. The flocculus accomplishes this by blending vestibular signals with visual tracking commands. Studies in cerebellar ataxia patients illustrate the balance of these mechanisms: patients with spinocerebellar ataxia type 3 (SCA-3) or episodic ataxia type 2 (EA-2) often have severely impaired smooth pursuit, yet they can still perform VOR cancellation reasonably well [4]. This suggests that the cerebellum can directly attenuate the VOR gain even when pursuit is poor, presumably via inhibitory Purkinje cell outputs that dial down vestibular nucleus activity. Conversely, in normal individuals, the smooth pursuit system augments this process by generating a predictive, continuous eye movement that matches head motion, effectively "erasing" any retinal slip [4]. Thus, VOR suppression is a hybrid behaviour – part reflex gating and part tracking eye movement – orchestrated by the vestibulo-cerebellum.



Importantly, VOR suppression is often context-dependent and can be enhanced by prediction. For example, if you are watching a tennis match, your head may follow the ball as your eyes stay on it; your VOR is largely suppressed because the brain predicts the ball's trajectory and actively nulls the reflex so your gaze can travel with the ball [2]. If the required head-eye movement is anticipated, the suppression kicks in almost pre-emptively. Experiments have shown that when subjects are cued and trained with repetitive head-eye tracking tasks, they develop anticipatory slow eye movements before head movement begins [4]. This predictive suppression is analogous to anticipatory smooth pursuit and relies on the brain's expectation of target motion. On the other hand, if a person's attention or cognitive load is diverted, VOR suppression performance degrades. It is not a purely reflexive act; it requires the patient to attend to the target and intend to keep eyes on it. An anxious or inattentive patient might fail a VOR cancellation test simply because they aren't fully engaged (their eyes will lag and then catch up, mimicking a central deficit) [7]. Therefore, the cognitive state of the patient (alertness, attention, anxiety) directly impacts this "reflex" – a pitfall we discuss later.

In summary, the mechanism of VOR suppression can be thought of as the brain's "stop order" to a reflex: it involves cerebellar down-regulation of the VOR gain and a simultaneous smooth tracking command that holds gaze on the moving target. Functionally, this is required in everyday situations whenever we move our heads in concert with something we're looking at. Classic examples include walking while fixating a friend who is walking beside you (your head and body turn with them) or reading street signs through a car window (your head follows the moving scenery). In all such cases, the brain must selectively suspend the otherwise automatic vestibulo-ocular response to prevent the eyes from being pulled off target [1].

4. Clinical Assessment

Bedside testing of VOR suppression is straightforward and revealing. The key is to have the patient rotate their head (or body) while fixating on a head-fixed target, so that the target moves

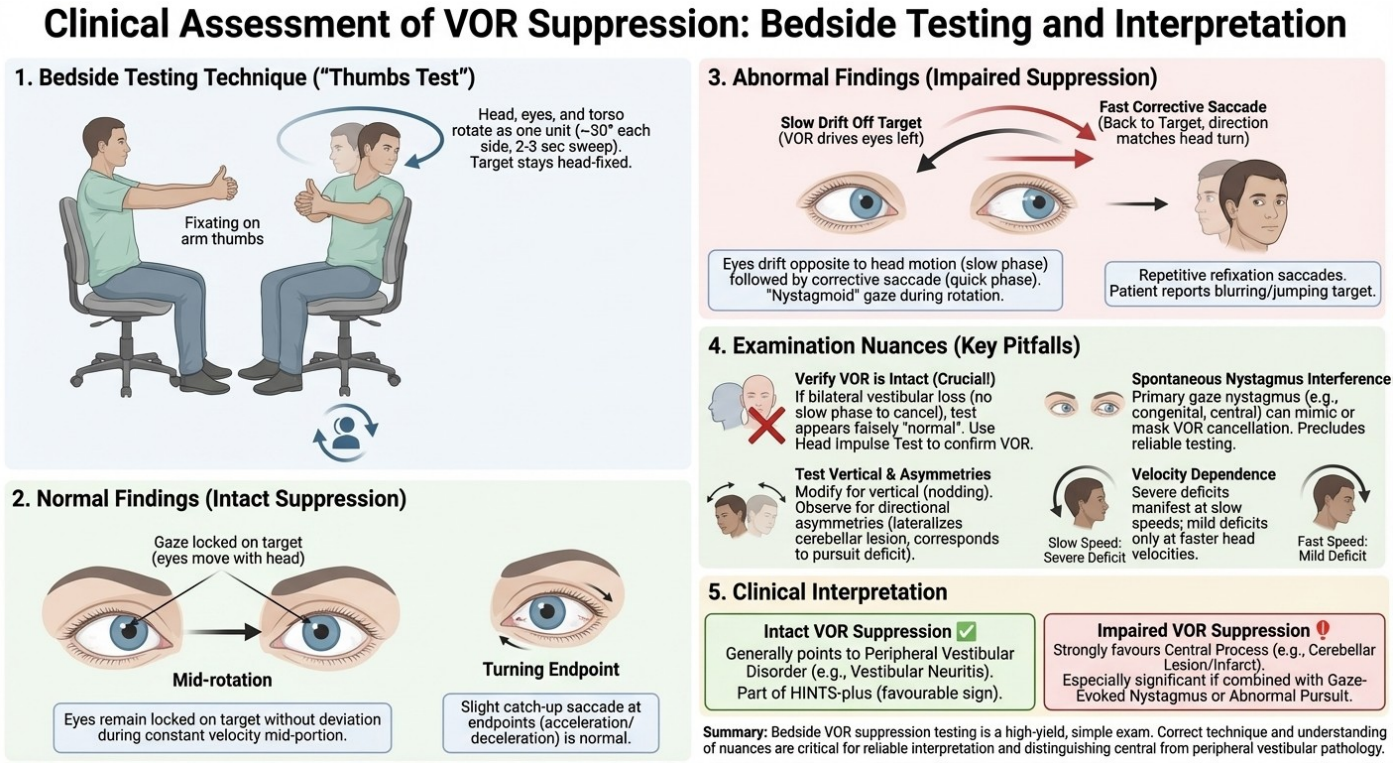
exactly with the head. The simplest method is the **"thumbs test."** Instruct the patient to clasp their hands together and extend both thumbs, focusing their gaze on their thumbs at arm's length. Then, while seated, the patient moves their head, eyes, and torso together in one unit – typically by rotating side-to-side in a swivel chair (or the examiner can guide their shoulders if no chair is available) [1]. The head turns approximately 30° to each side; importantly, the entire rotation should be done en bloc so that the visual target (thumbs) stays directly in front of the face [1]. A full side-to-side sweep can be done over ~2–3 seconds; increasing the speed gradually will stress the system and reveal the velocity limits of suppression [1]. The patient's eyes are observed closely: normally, if VOR suppression is intact, the eyes will remain locked on the thumbs without deviation throughout the mid-portion of the rotation [1]. It will appear as if the patient's eyes and head are moving together, because gaze is maintained on the moving target. Near the turning endpoints (when the rotation reverses direction), it's normal to see a slight catch-up saccade or two – due to the brief acceleration/deceleration – but during constant velocity mid-rotation, the eyes should not jerk off target [1].

If VOR suppression is impaired, the examiner will see the patient's eyes being dragged opposite to the head motion (i.e. following the VOR) and then fast corrective saccades back toward the target. In other words, the patient's gaze will appear "nystagmoid" during the rotation [1]. Specifically, as the head turns right, for example, the intact VOR (now inappropriately active) drives the eyes leftward off the target; the patient then makes a rightward quick phase to re-fixate the thumbs – producing a right-beating saccadic nystagmus (slow phase left with head-right, quick phase right) in the middle of the manoeuvre [1]. This pattern will repeat in each direction of motion if the cancellation failure is persistent. Notably, the direction of the corrective saccades is the same as head rotation (since the slow drift is opposite to head rotation). The presence of these repetitive refixation saccades during the manoeuvre is a positive finding for abnormal VOR suppression. Patients often describe it as an inability to keep their eyes on the target – "my thumb was getting blurry/jumping when I turned." Severe deficits will manifest even at slow rotation speeds, whereas mild deficits might only emerge at faster head velocities [1].

Several nuances in examination technique:

- **Always verify the VOR is intact** before attributing a "normal" suppression test to the patient. If the patient has bilateral vestibular loss, they will have no slow-phase VOR to cancel – and thus will appear perfectly normal on the thumbs test despite profound pathology [1]. In such cases, the test is a false-negative (because there was nothing to suppress). A quick bedside head impulse test can ensure the patient has a functional VOR; if head impulse is bilaterally abnormal, the VOR suppression test is not informative (one cannot suppress what isn't there).
- **If the patient has spontaneous nystagmus or gaze-evoked nystagmus** in primary gaze, this can interfere with the test. For instance, congenital nystagmus or certain central nystagmus will cause continuous eye motion that can mimic or mask VOR cancellation ability [1]. In such cases, interpretation is difficult – the continuous "noise" of the baseline nystagmus can make even a normal individual look abnormal on the test. The rule of thumb is that primary position nystagmus precludes reliable VOR suppression testing [1].
- **Test both horizontal and vertical VOR suppression if possible.** The horizontal thumb rotation is most common, but one can modify the test for vertical VOR cancellation by having the patient fixate an outstretched thumb and then flex/extend the trunk (nodding motion) together with the target [1]. This is rarely done in routine exams, but it can uncover an asymmetric deficit (some patients, especially with cerebellar dysfunction, have worse downward vs. upward suppression, etc., correlating with their pursuit asymmetries).

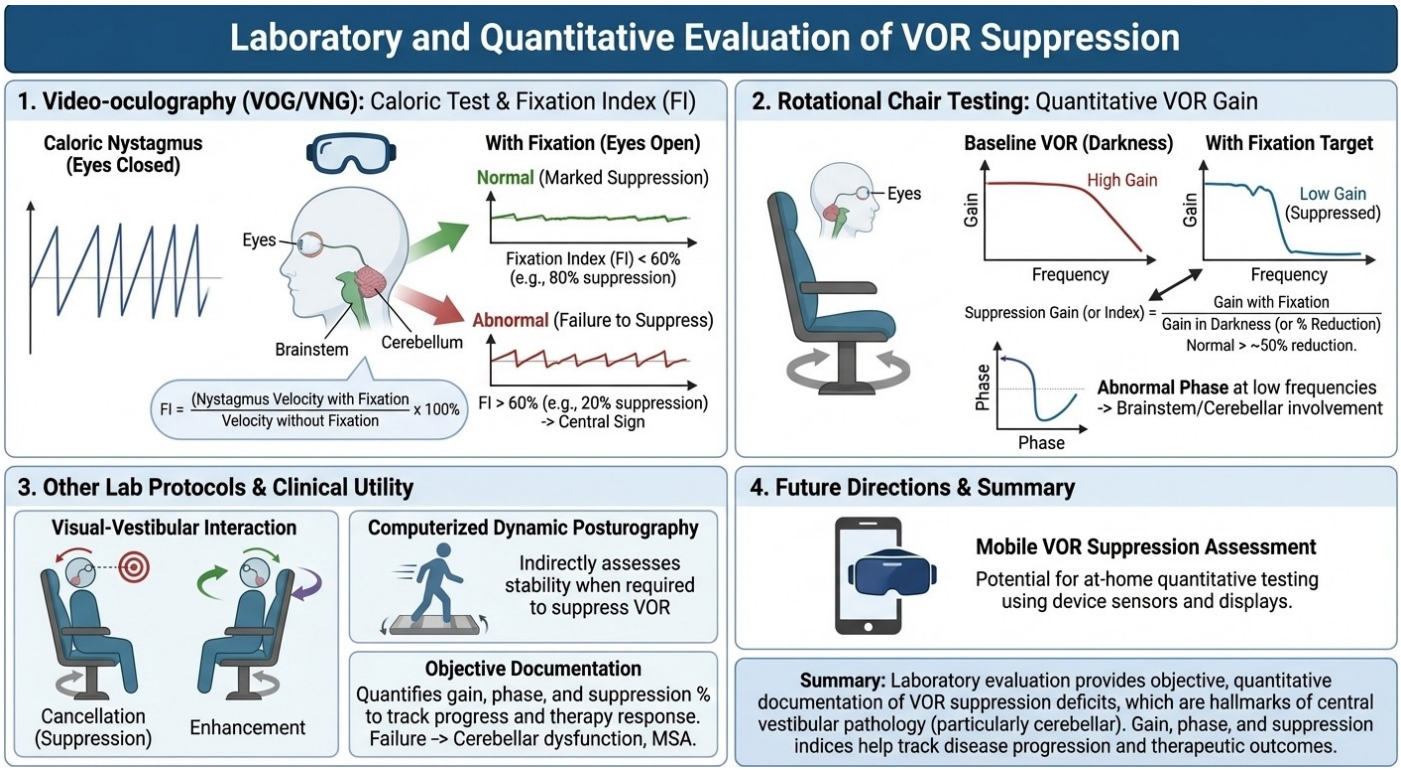
- **Observe for directional asymmetries.** A unilateral floccular lesion, for example, might predominantly affect VOR suppression toward the **ipsilesional side** [1]. Suppose a patient has a right flocculus infarct: when they rotate head-right (which requires cancelling a rightward head impulse), the right flocculus can't adequately suppress, so you see nystagmus; but head-left rotation might be near normal. This asymmetry can help lateralize a cerebellar lesion. In general, deficient VOR cancellation to one side corresponds to a low smooth pursuit gain to that same side on exam [1].



Clinically, a bedside VOR suppression test is often performed as part of the HINTS-plus battery or the ocular motor exam in any dizzy patient. For example, after assessing head impulse, nystagmus, and skew, the examiner might have the patient perform the thumb rotation test. The presence of saccadic intrusions (breakdown of fixation) strongly favours a central process (typically a cerebellar lesion) [1]. On the other hand, a patient with an acute peripheral vestibulopathy (like vestibular neuritis) will usually be able to keep their eyes on target with only minor difficulty (unless the head movement inadvertently increases their spontaneous nystagmus). The "functional" gaze stabilization capacity can also be roughly assessed by asking the patient to read or recognize objects while actively turning their head with a target. Some clinicians use a dynamic visual acuity test (reading an eye chart while oscillating the head with and without a head-fixed laser target) to gauge if the patient can effectively cancel VOR when needed. In summary, bedside VOR suppression testing – though simple – is a high-yield examination: intact VOR suppression in a dizzy patient generally points to peripheral vestibular disorder, whereas impaired VOR suppression (especially when combined with gaze-evoked nystagmus or abnormal pursuit) localizes to the central vestibular pathways (vestibulo-cerebellum) [1].

5. Laboratory and Quantitative Evaluation

Video-oculography (VOG/VNG) can quantify VOR suppression using dedicated paradigms. In the **caloric test**, for instance, after inducing vestibular nystagmus with warm or cold irrigation, the patient is asked to open their eyes and fixate on a target. The degree to which the nystagmus slow-phase velocity decreases with fixation is measured as the **Fixation Index (FI) or suppression percentage**. If nystagmus is fully wiped out, the FI approaches 0% (complete suppression). An FI of 100% would mean fixation had no effect (nystagmus just as strong with eyes open as eyes closed) [6]. In practice, normal individuals can suppress ~50–100% of caloric nystagmus; an **FI below about 60%** is often taken as a cutoff for abnormal (i.e. failure to suppress >40% of the nystagmus) [6]. A failure to suppress nystagmus with fixation is a clear sign of central pathology [6]. Because precise normative values can vary, many labs simply report present or absent fixation suppression. The key is that a normal peripheral vestibular system should show marked reduction or extinction of nystagmus once the patient can fixate (thanks to cerebellar clamp-down of the reflex), whereas a cerebellar lesion often yields little change – the nystagmus persists despite visual fixation.



Rotational chair testing provides a controlled way to assess VOR suppression across different frequencies. In a standard sinusoidal rotation (e.g. 0.1 Hz oscillation), the patient is tested in darkness to establish the baseline VOR (eye movements recorded via VOG). Then the test is repeated with the patient fixating on a dot moving with them (often a laser target projected inside the chair, so it is earth-stationary or chair-stationary depending on protocol). In a person with normal cerebellar function, the VOR gain will drop sharply when the fixation target is introduced – ideally approaching zero eye velocity (no nystagmus) [6]. The suppression gain (often called VOR cancellation gain) can be calculated as the ratio of eye velocity with fixation to eye velocity in darkness, or simply the percentage reduction in gain [6]. Modern rotary chair systems will compute a VOR suppression index. For example, if the VOR gain at 0.2 Hz is 0.5 in darkness and 0.1 with fixation, that's an 80% suppression, which is normal. An abnormal result might be, say, only a 20% reduction (gain 0.5 → 0.4 with fixation), indicating the patient cannot effectively cancel the reflex – strongly suggestive of a central lesion. Phase measurements can also be

examined: normally, when residual nystagmus is minimal, phase becomes moot, but in partial suppression one might see changes in the phase lag of the eye velocity relative to head velocity, reflecting the brain's imperfect cancellation timing. A markedly abnormal phase during low-frequency rotations often points to brainstem-cerebellar involvement in the VOR pathway [6].

Other lab protocols include the **visual-vestibular interaction tests** (sometimes called VOR enhancement and suppression). In one, a small optokinetic stimulus is rotated with the chair (testing cancellation), in another it's rotated opposite the chair (which enhances the VOR). These can further tease out deficits. Computerized dynamic posturography with head movements and visual tasks can indirectly assess if a patient becomes unstable when required to suppress VOR (a central integration issue). However, the most direct metric remains the VOR suppression test on rotational chair, which is essentially a quantitative analog of the bedside test. A "failure" of this test – defined as inability to drop gain appropriately – is reported in disorders like cerebellar degeneration, multiple system atrophy, etc., and correlates with clinical exam findings [4, 8].

Overall, laboratory evaluation provides objective documentation: one can literally see the trace of eye velocity not flattening out when it should. The gain, phase, and suppression percentage help track a patient's progress or response to therapy. It's worth noting that current research is looking to make VOR suppression testing more accessible using mobile devices. For instance, using a smartphone's VR or video capabilities to measure eye movement while the patient does an at-home VOR cancellation exercise [2]. This could in the future allow quantitative VOR suppression assessment without a full rotary chair, expanding our ability to detect subtle central vestibular signs.

6. Central vs. Peripheral Patterns

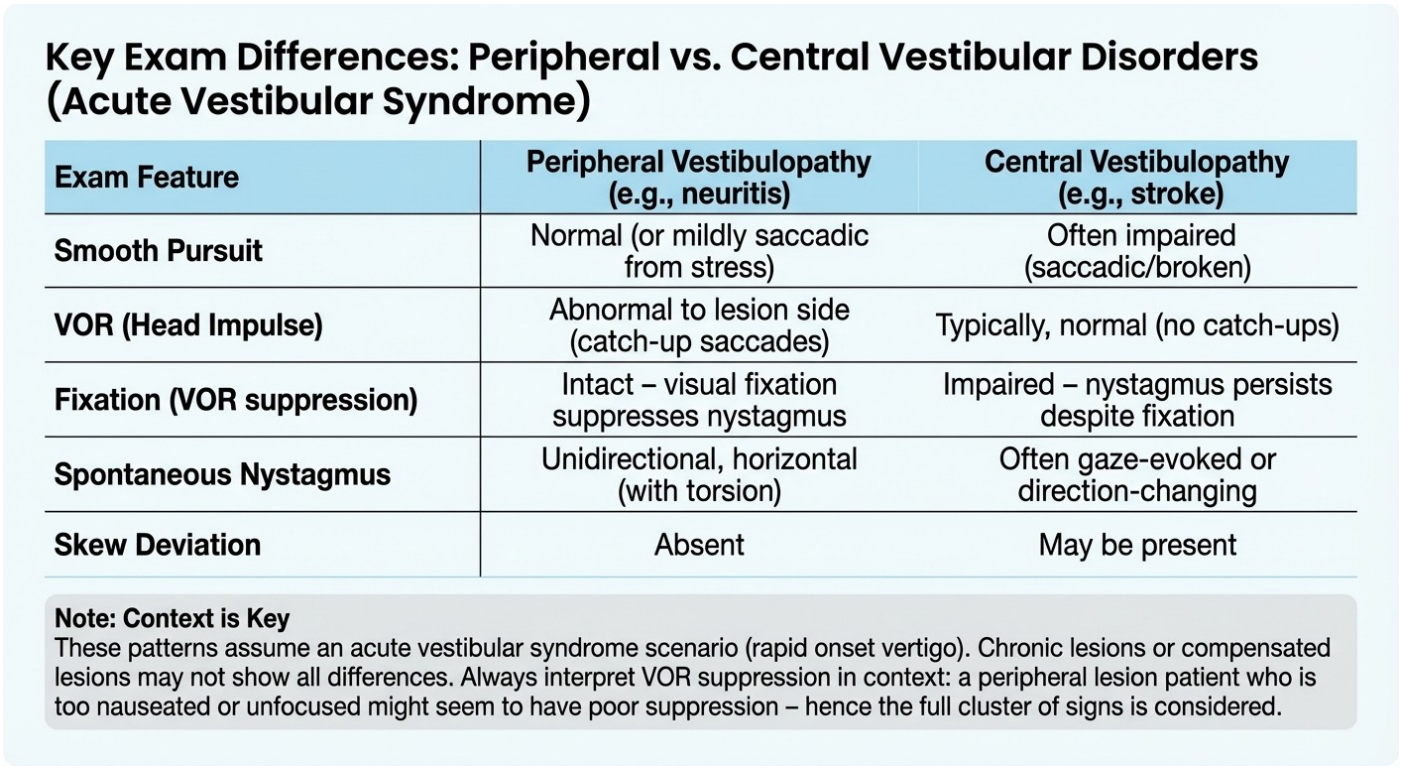
A recurring theme is that peripheral vestibular disorders (inner ear or vestibular nerve lesions) tend to spare VOR suppression, whereas central disorders (brainstem or cerebellar lesions) often impair it. In acute unilateral peripheral loss, the patient will have vestibular nystagmus, but the cerebellum is intact and thus able to suppress that nystagmus when the patient fixes gaze. By contrast, if the cerebellum (especially the vestibulocerebellum) is lesioned, fixation may not suppress nystagmus – in fact, central nystagmus can sometimes worsen with fixation (e.g. downbeat nystagmus amplitude increases on fixation).

Table 1 summarizes key differences between a typical peripheral vestibulopathy (e.g. vestibular neuritis) and a central vestibular lesion (e.g. cerebellar stroke) on examination:

Exam Feature	Peripheral Vestibulopathy (e.g. neuritis)	Central Vestibulopathy (e.g. stroke)
Smooth Pursuit	Normal (or mildly saccadic from stress)	Often impaired (saccadic/broken)
VOR (Head Impulse)	Abnormal to lesion side (catch-up saccades)	Typically, normal (no catch-ups)
Fixation (VOR suppression)	Intact – visual fixation suppresses nystagmus	Impaired – nystagmus persists despite fixation

Spontaneous Nystagmus	Unidirectional, horizontal (with torsion)	Often gaze-evoked or direction-changing
Skew Deviation	Absent	May be present

Table 1: Oculomotor and VOR findings in Peripheral vs. Central vestibular disorders. Note: These patterns assume an acute vestibular syndrome scenario (rapid onset vertigo). Chronic lesions or compensated lesions may not show all differences. Always interpret VOR suppression in context: a peripheral lesion patient who is too nauseated or unfocused might seem to have poor suppression – hence the full cluster of signs is considered.

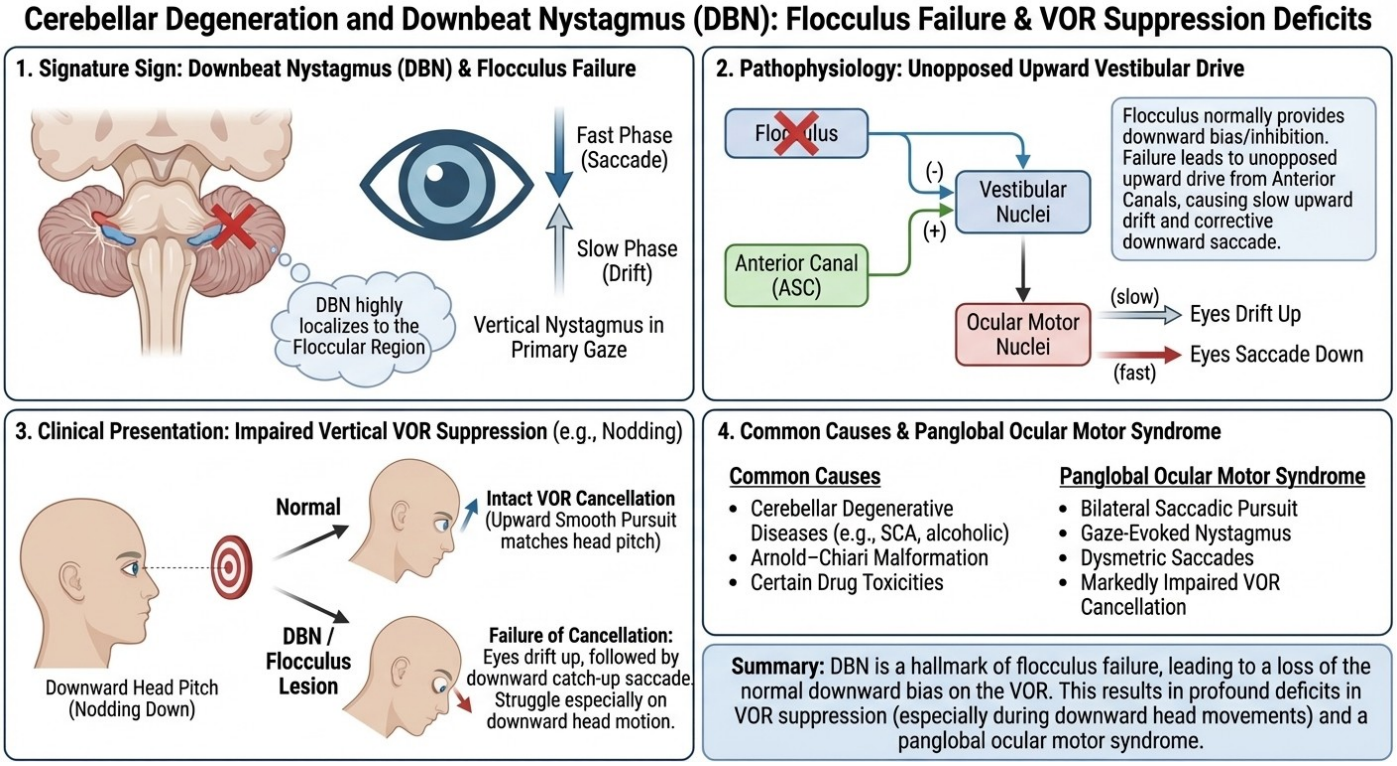


In essence, a peripheral lesion (labyrinth or nerve) causes a deficit in the afferent input, which yields nystagmus and a positive head-impulse test, but the brain's central apparatus (flocculus, etc.) is intact, so any vestibular nystagmus can be tamed by fixation. Conversely, a central lesion in the vestibular circuitry means the normal "brakes" on the VOR are gone – so even if the peripheral labyrinth is fine (head impulse normal), the patient cannot centrally adjust or suppress the vestibular signals. They often have other ocular motor signs (abnormal pursuit, gaze-evoked nystagmus, skew deviation) that accompany the VOR cancellation failure [1].

A classic example is the HINTS exam for stroke vs neuritis: a patient with vertigo and nystagmus who has a normal head impulse, direction-changing nystagmus, or skew deviation is likely central. One could add that impaired fixation suppression of the nystagmus is an additional central clue. In practice, when a dizzy patient can't keep their eyes on a head-fixed target (thumbs test shows breakdown), the index of suspicion for a cerebellar stroke or brainstem lesion skyrockets. On the flip side, in a patient with acute peripheral vestibular loss (e.g. neuritis), one expects that after head thrusts reveal the deficit, allowing the patient to visually fixate will significantly calm the nystagmus (unless they are in darkness or wearing Frenzel goggles). This principle also guides management: in peripheral lesions we encourage fixation (e.g. allow light) to help the patient stabilize gaze, whereas in central vestibular nystagmus, even with fixation the patient may continue to have oscillopsia, sometimes necessitating medication.

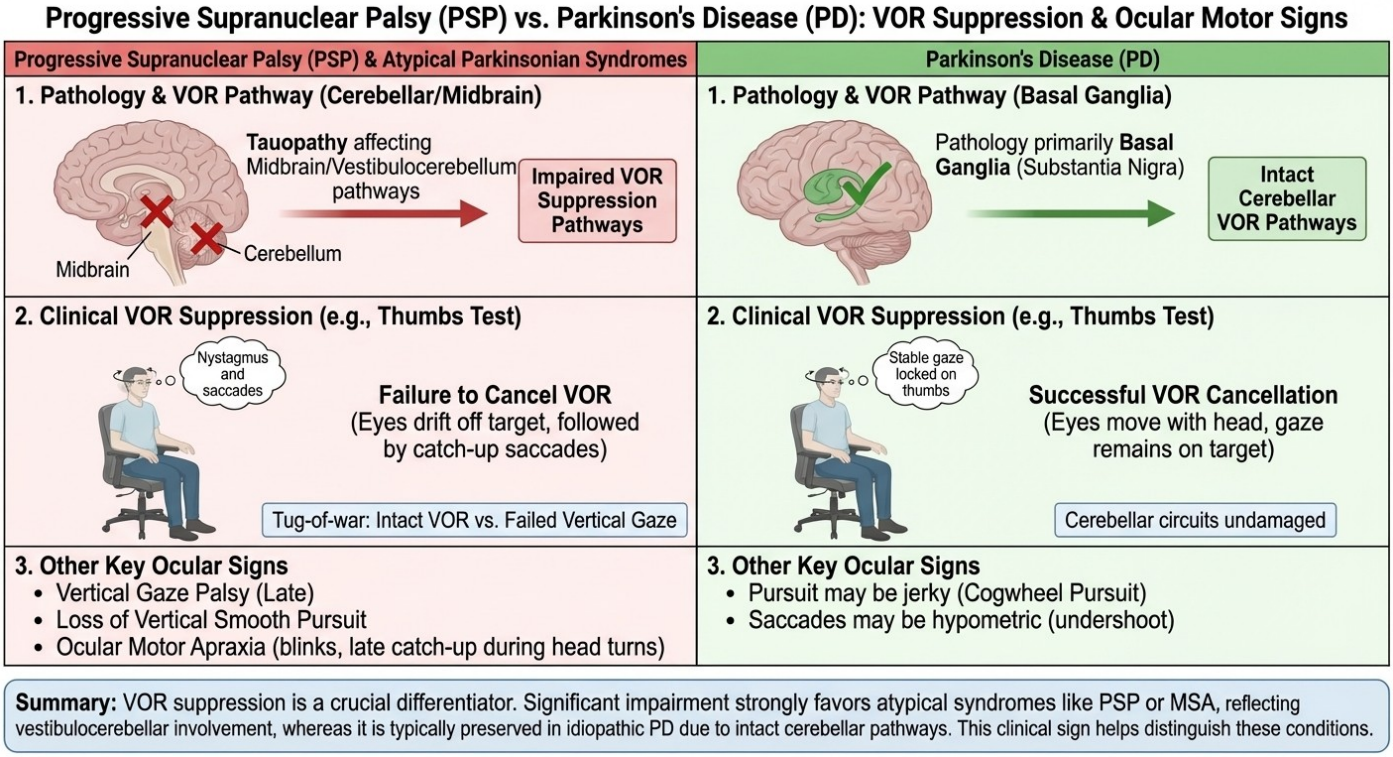
7. VOR Suppression in Specific Clinical Conditions

Cerebellar Degeneration and Downbeat Nystagmus (DBN): Deficits in VOR suppression are most dramatically seen in disorders that affect the **flocculus and paraflocculus.** In diffuse cerebellar degeneration (alcoholic cerebellar degeneration, spinocerebellar ataxias, etc.), patients often have a panglobal ocular motor syndrome: bilateral saccadic pursuit, gaze-evoked nystagmus, dysmetric saccades, and markedly impaired VOR cancellation. These individuals simply cannot hold their eyes on target when their head moves, reflecting the loss of vestibulo-cerebellar function. A signature finding in such patients is Downbeat Nystagmus (DBN) – a vertical nystagmus (fast phases down) present in primary gaze. DBN is highly localizing to the floccular region. The pathophysiology involves the flocculus's normal bias for downward eye movement; when the flocculus fails, upward vestibular drive from the anterior canals goes unopposed, and the eyes drift up, requiring corrective downward saccades. Functionally, these patients have profound VOR suppression deficits, especially for downward head motion. For example, if a DBN patient tries to cancel the VOR while pitching their head up and down, they will particularly struggle when the head is moving downward (which normally requires the flocculus to generate upward smooth pursuit to cancel the downward VOR). The flocculus lesion means they cannot produce that upward cancellation, so an upward drift (and downward catch-up saccade) ensues. Clinically, one may observe that DBN patients cannot hold their eyes on a target when nodding or when attempting vertical VOR cancellation – the eyes will drift upward on downward head movement. In short, DBN = flocculus failure, and with it comes failure of VOR suppression (often in both horizontal and vertical planes). Classic causes include cerebellar degenerative diseases, Arnold–Chiari malformation, and certain drug toxicities (see below).

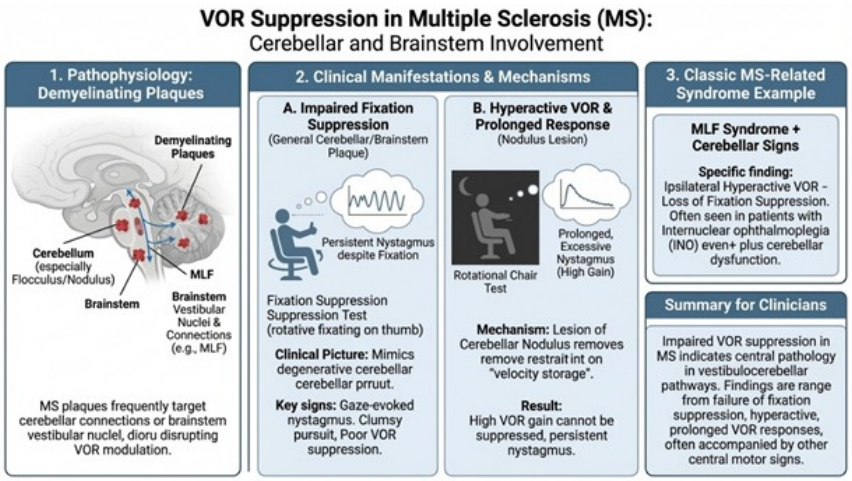


Progressive Supranuclear Palsy (PSP) and Parkinsonian Syndromes: PSP, a neurodegenerative tauopathy affecting midbrain and cerebellar structures, is known for vertical gaze palsy and loss of smooth vertical pursuit. Patients with PSP (and the related Multiple System Atrophy, MSA) frequently show an inability to suppress the VOR, especially as the disease progresses [4]. One study demonstrated that VOR cancellation was impaired in most PSP and MSA patients, but not in Parkinson's disease (PD) [4]. This aligns with pathology: PSP

and MSA often involve brainstem and cerebellar pathways (the vestibulocerebellum), whereas PD primarily affects the basal ganglia (leaving the cerebellar VOR pathways intact). Early in PSP, vertical pursuit may be relatively preserved, and accordingly VOR cancellation might be near-normal in straightforward head rotations. However, as vertical gaze fails, those patients also lose the ability to cancel vertical VOR (they develop a "tug-of-war" between a still-intact VOR and an eye that cannot move volitionally). Clinically, one may see "ocular motor apraxia" in PSP during head turns – the eyes don't move with the head when they should (due to poor suppression), leading to blinks or late catch-up saccades. In Parkinson's disease, by contrast, although pursuit can be jerky (cogwheel pursuit), patients can usually perform VOR cancellation because the cerebellar circuits are undamaged. This difference can help differentiate atypical Parkinsonian syndromes (PSP, MSA) from PD: if a patient with parkinsonism has clear VOR cancellation impairment, one leans toward a central degenerative syndrome rather than idiopathic PD [4, 8].



Multiple Sclerosis (MS) and Brainstem Stroke: Demyelinating plaques in MS often hit the cerebellar connections or brainstem vestibular nuclei. An MS patient with a plaque in the cerebellum may present exactly like a degenerative cerebellar disease: gaze-evoked nystagmus, clumsy pursuit, and poor VOR suppression. In fact, impaired fixation suppression of the VOR is one finding in MS patients with brainstem/cerebellar involvement [4]. Moreover, MS can cause hyperactive VOR responses – for instance, a lesion of the cerebellar nodulus will remove the restraint on velocity storage,



leading to prolonged and sometimes excessive nystagmus in rotational tests [9, 6]. Such patients may have a high VOR gain that they cannot suppress, manifesting as a combination of gaze-evoked and vestibular nystagmus that persists with fixation. One classic MS-related finding is ipsilateral hyperactive VOR with loss of fixation suppression, described in patients with **internuclear ophthalmoplegia plus cerebellar signs (the so-called "MLF syndrome")** [1].

In acute brainstem strokes, especially in the lateral medulla or pons, VOR suppression can be variably affected depending on whether the vestibular nuclei or cerebellar peduncles are involved. A stroke in the floccular region (e.g. lateral pontine/cerebellar infarct, such as an AICA stroke) often produces an acute vestibular syndrome where head impulse may be normal (central), nystagmus may be present (often direction-changing or gaze-evoked), and VOR cancellation is notably impaired. Such a patient is a set-up for being missed as "labyrinthitis" unless the physician checks pursuits and VOR suppression. Indeed, one study of small cerebellar strokes noted that these patients had normal initial MRIs but ocular motor signs (like impaired pursuit or VOR cancellation) that were more sensitive to the stroke. Thus, in MS and stroke affecting central vestibular pathways, preserved VOR suppression essentially rules out a significant lesion, whereas impaired VOR suppression is a key positive finding that supports central pathology (mandating MRI, etc.) [1].

Pharmacological influences: A variety of drugs can reversibly impair VOR suppression by their depressive effects on the cerebellum. Clinically, this is important: a patient on **high-dose barbiturates, anti-epileptics, or sedatives** can look cerebellar (with gaze nystagmus and poor VOR cancellation). For example, benzodiazepines (diazepam, lorazepam, etc.) are GABA-ergic and suppress cerebellar and brainstem function, leading to significantly reduced pursuit gain and troublesome VOR cancellation – the eyes will not stay on target, showing intrusions. Phenytoin toxicity famously causes nystagmus and ataxia; such patients have saccadic pursuit and likely could not cancel their VOR either. One particularly well-known drug effect involves alcohol: alcohol acutely impairs floccular function and thus VOR suppression, giving rise to gaze-evoked nystagmus and an inability to hold fixation during head movement. In fact, field sobriety tests (e.g. watching for eye jerks on head rotation) exploit this – an intoxicated person's eyes will be dragged off target when attempting to follow a moving object, a direct reflection of impaired cerebellar suppression of the VOR. Lithium, at toxic levels, can cause downbeat nystagmus by accumulating in the flocculus and similarly produce cancellation failure. Anticonvulsants, sedatives, some antidepressants, and even opioids (in high dose, via indirect effects) have all been reported to degrade smooth pursuit and fixation suppression [1]. The good news is that these effects are often dose-dependent and reversible – removing the offending agent will restore normal cerebellar function and thus normal VOR suppression. For the vestibular clinician, recognizing a drug-induced VOR suppression deficit is crucial to avoid misdiagnosing a degenerative cerebellar disorder. A patient on, say, carbamazepine who shows poor cancellation might just need a medication adjustment rather than an MRI – if all other signs align with a drug effect (symmetric bilateral findings, etc.).

Other specific conditions:

- **Vestibular Migraine:** Some migraine patients have brainstem aura or fluctuating cerebellar dysfunction during episodes. They might transiently have difficulty with VOR suppression (feeling that during head motion their vision slips).
- **Otosclerosis/Stapes surgery:** Interestingly, patients with stiff or surgically absent stapes may have an abnormal "otolith-VOR" interaction but their central suppression is typically intact.

- **Anxiety disorders:** Chronic anxiety can both mimic and worsen VOR suppression issues, as anxious patients often struggle to concentrate on targets and can exhibit subtle gaze instabilities that complicate the exam.

8. Interpretation Challenges and Pitfalls

Interpreting VOR suppression tests requires vigilance for confounding factors:

1. **Attention and compliance:** Perhaps the most common pitfall is a patient who simply isn't trying or isn't able to maintain attention. VOR suppression is a volitional task; an inattentive patient (distracted, anxious, somnolent, or cognitively impaired) may produce an abnormal result even with a perfectly intact cerebellum [7]. Their eyes might wander off the target because they aren't fixating well, leading the examiner to see saccades that mimic a failure of suppression. One distinguishing feature is variability: if one trial is terrible but after coaching ("Look at your thumb, keep it clear!") the next trial is normal, the issue was attention, not anatomy. Encouraging the patient and even using verbal cues during the manoeuvre can help – if performance dramatically improves, a true cerebellar deficit is less likely. Anxiety can also cause small, jerky eye movements (square-wave jerks) or over-responsiveness that are not true VOR reflexes. The clinician should ensure the patient is calm, understands the task, and is exerting full effort.
2. **Visual issues:** A basic but important point – if the patient cannot see the target clearly, they cannot fixate and suppress their VOR effectively. Poor visual acuity, uncorrected refractive error, or significant oscillopsia will impede fixation. For instance, an elderly patient who forgot their glasses might seem to have abnormal cancellation simply because the thumb or pen they're told to look at is a blur. Always use an adequate target (high contrast, maybe an 'X' on the thumb, etc.), ensure the patient's best correction is worn, and provide good lighting. Similarly, ocular alignment problems (strabismus) could reduce fusion and fixation ability, affecting the test.
3. **Baseline nystagmus or ocular motor disorders:** As mentioned, any ongoing nystagmus (especially central nystagmus in primary gaze) will interfere. For example, a patient with upbeat nystagmus at baseline will have a continual vertical drift that can be misinterpreted as VOR slip. If possible, such patients can be tested in the null position of their nystagmus (where it's minimal) or not tested at all for suppression. An internuclear ophthalmoplegia (INO) (common in MS) can also complicate things: if one eye lags on adduction, during a head rotation that requires that eye to move nasally, it may appear as a failure – but the issue is the MLF lesion, not the VOR suppression per se. In these cases, careful examination will note the adduction lag and dissociate it from a true bilateral cancellation problem. Likewise, a VIth nerve palsy could cause the patient to make catch-up saccades not because of VOR failure but because one eye simply can't maintain fixation toward an extreme. The examiner should be mindful of any ocular motor palsies or gaze limitations that could alter the test. Generally, VOR cancellation is best assessed in the mid-range of gaze (primary position) to avoid end-gaze nystagmus or limitations.
4. **Bilateral vestibular loss giving false "normal" result:** This is the converse of the usual central/peripheral expectation. As noted, a patient with absent vestibular function (e.g. bilateral vestibulopathy from gentamicin toxicity) will have no VOR to speak of. If you ask them to rotate with their thumb, their eyes might stay on target simply because the head movement doesn't generate any reflex eye movement that needs suppression. One might erroneously conclude "wow, great VOR cancellation!" – a dangerous mistake, since in fact the patient has lost VOR altogether. The clue is in the rest of the exam: such a patient

will have bilaterally abnormal head impulse tests and likely oscillopsia with head movement. Always pair interpretation of a normal suppression test with the results of head impulse and other vestibular function tests [1]. A normal suppression test means something only if the baseline VOR is present and generating eye movement to be suppressed. This is a critical pitfall in patients with bilateral vestibular failure.

5. **Age-related changes:** Older patients may have a degree of physiologic decline in all ocular motor systems, including smooth pursuit and perhaps fixation suppression. While VOR gain itself often remains high in aging, the integration with visual tracking can degrade. An elderly person might show a few small saccades on cancellation testing yet not have a frank pathology – this could be due to age-related cerebellar Purkinje cell loss or slight attentional lapses. The literature suggests VOR suppression (VORS) function worsens with age, and this correlates with balance issues and fall risk in the elderly **[2]**. So, mild imperfections in an octogenarian should be judged against an age norm (slightly saccadic cancellation might be their "normal"). That said, a truly abnormal response (clear inability to suppress, with robust nystagmus) is never normal at any age – it should prompt investigation.
6. **Technique and adaptation artifacts:** If the examiner does not rotate the patient's head and target exactly together (for instance, if the patient's eyes have to make tiny adjustments because the target isn't perfectly head-fixed), one might see small corrective saccades that aren't due to VOR – they're due to target/head misalignment. It's important to keep the target strictly head-fixed. Additionally, if the rotation is too slow or too fast, results can vary. Very slow rotations (<0.1 Hz) might not provoke much VOR to begin with, whereas very fast sudden rotations might engage other mechanisms (like cervico-ocular reflex or catch-up saccades even in normal subjects). The optimal speed is a gentle, continuous rotation that challenges the system but is not abrupt (e.g. 1–2 Hz oscillation is plenty). Another consideration is adaptation: if you repeat the test multiple times in the same session, patients (especially young ones) can "learn" to improve their suppression by anticipating the movement. They essentially enhance their predictive pursuit component with repetition [4]. Finally, Frenzel goggles or video goggles (used to observe nystagmus by removing fixation) obviously cannot be on during a fixation suppression test – it seems obvious, but if one forgets to allow the patient to see, the test is invalid (and will always appear abnormal, since you've prevented the very mechanism – vision – that does the suppression!) [1].

9. Advanced Insights and Future Directions

Rehabilitation and training: Interestingly, because VOR suppression is a learned, volitional skill, it can be trained and improved to some extent. Vestibular rehabilitation therapy often includes specific VOR cancellation exercises. For example, a common exercise is to have the patient hold a card with a letter at arm's length and practice moving their head and the card together side to side, keeping the letter in focus (essentially a formalized thumbs test) [10, 11]. By doing sets of these movements daily, patients with central vestibular dysfunction (or even those with residual symptoms after a peripheral insult) can potentially enhance their gaze stability. The idea is that repetitive practice helps engage any remaining cerebellar plasticity to maximize suppression and perhaps recruit cortical predictive mechanisms. In disorders like cerebellar ataxia, therapy won't cure the flocculus, but it might teach the patient compensatory strategies (e.g. initiating a head movement more slowly or using slight head thrusts with saccades). In vestibular migraine or mild TBI, where the issue may be more of a "tuning" problem than structural loss, such training can significantly reduce symptoms of visual motion sensitivity. In fact, the Visual Motion Sensitivity (VMS) test in concussion evaluations is essentially a VOR

suppression task (the patient rotates and rates symptoms) [12]. Therapists use habituation exercises based on that: by gradually exposing patients to head-body rotations with complex visual input, they aim to restore normal VOR modulation.

VOR suppression in sports and daily life: Beyond the clinic, many sports inherently demand good VOR suppression. Consider a basketball player doing a quick no-look pass – their head turns but their eyes are momentarily tracking a moving teammate; they need to suppress the VOR to keep their gaze on the moving target rather than reflexively staying on the original scene. Athletes in sports like skiing, snowboarding, or gymnastics, where head movements are rapid and vision must stay briefly on a moving reference (like spotting a landing), likely develop robust vestibulo-cerebellar function. Understanding VOR suppression could thus have implications for optimizing performance and preventing injuries (e.g. better gaze stabilization might reduce falls).

Falls and balance: As noted, impaired VOR suppression has been linked to imbalance in older adults [2]. If you think about walking through a crowded environment, your ability to navigate without dizziness requires that you sometimes suppress VOR (when walking with a friend and looking at them) and other times use VOR (looking at a sign while moving). This flexibility is part of an overall vestibulo-visual balance system. People with poor VOR suppression may avoid moving their head or become overly reliant on vestibular cues, which could contribute to instability.

Neurophysiology and modelling: VOR suppression continues to be a useful paradigm for neuroscientists to study sensorimotor integration. By examining neuronal firing during VOR cancellation versus VOR active conditions, researchers can identify which neurons carry vestibular signals, visual tracking signals, or both. For example, a neuron in the vestibular nucleus might decrease its firing during cancellation – indicating it's being inhibited by the cerebellar input. In fact, using VOR cancellation as a condition in experiments allows isolation of pure pursuit-related activity in certain cells (since the reflex component is nulled) [13]. Modern computational models seek to incorporate adaptive neural networks that can predict when to suppress the VOR based on context (visual scene, efference copy of head movement, etc.). The cerebellum is central to these models, acting as a dynamic gain controller and predictor [4].

Digital and virtual reality (VR) assessments: As highlighted in a recent review, digital technology is poised to revolutionize VOR suppression assessment [2]. For instance, using a VR headset, one can present a patient with a rich visual environment that moves with head motion and measure their eye responses accurately. This can standardize the test (controlling for attention with engaging tasks) and also probe different conditions (moving backgrounds, cognitive dual tasks, etc.). Smartphone-based eye trackers could allow at-home monitoring of VOR suppression – potentially useful for telemedicine or for long-term tracking in degenerative disease.

Pathophysiological research: VOR suppression is being examined in conditions like cerebellar ataxia genetic subtypes. Also, pharmacologic probes (like giving baclofen, a GABA-B agonist) have been used in research to see how velocity storage and VOR suppression change – baclofen is known to reduce velocity storage time constant, essentially mimicking nodulus effects [13]. Studying patients on such medications or with implanted stimulators can provide insights into the neural circuits of suppression.

In conclusion, the Vestibulo-Ocular Reflex suppression is a small window into the brain's larger ability to modulate reflexes with volition. For the clinician, it is a powerful bedside tool to detect

central vestibular dysfunction. For the scientist, it represents a model system for adaptive motor control, prediction, and sensorimotor integration. As technology enhances our ability to measure and train this reflex, we anticipate improved diagnostic accuracy, better rehabilitation strategies, and a deeper understanding of how our brains achieve the delicate balance between reflexive stability and flexible interaction with a moving world.

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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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