Oculomotor Assessment:
A Comprehensive Clinical Review

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Section 3A — Oculomotor Assessment | Vestibular Function Testing Series

How to Use This Review

This document is the companion clinical literature review to the oculomotor testing and 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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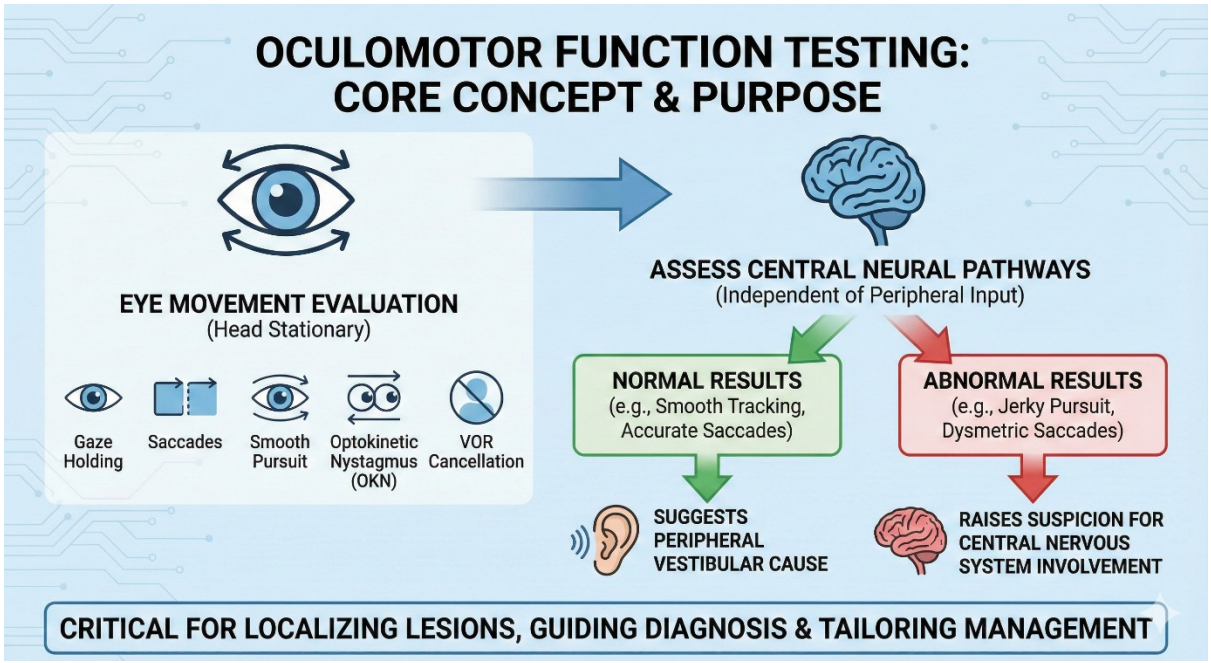
Oculomotor Function Testing in the Vestibular Test Battery

Introduction

Oculomotor function testing refers to the evaluation of a patient's voluntary and reflex eye movements as part of the comprehensive vestibular test battery. It typically includes a series of eye movement tests – such as **gaze holding, saccades, smooth pursuit, optokinetic nystagmus (OKN), and vestibulo-ocular reflex (VOR) cancellation** – conducted with the head stationary. These tests primarily assess the integrity of central neural pathways that control eye movements (the oculomotor system) independent of peripheral vestibular input [1]. By measuring eye movement behaviour relative to visual targets, clinicians can discern whether a patient's dizziness or imbalance arises from a central (brain/brainstem) lesion or a peripheral vestibular disorder [3, 1]. In essence, normal oculomotor test results (e.g. smooth, accurate tracking and normal saccades) in a dizzy patient suggest that the ocular motor control pathways are intact, increasing the likelihood of a peripheral vestibular cause, whereas abnormalities (e.g. jerky pursuit or dysmetric saccades) raise suspicion for central nervous system involvement [3, 5]. Oculomotor testing is therefore a critical component of vestibular diagnostics, offering a non-invasive window into central vestibulo-ocular function and providing context for interpreting other vestibular exams [5, 6]. Modern videonystagmography (VNG) systems with infrared eye-tracking goggles allow quantification of eye movements with high precision, enhancing the sensitivity of oculomotor tests and enabling objective baseline measurements for comparison [5]. Overall, oculomotor function testing significantly improves diagnostic accuracy by helping vestibular physicians localize lesions (central vs. peripheral), tailor further investigations, and guide management of patients with vertigo and balance disorders [1, 3].

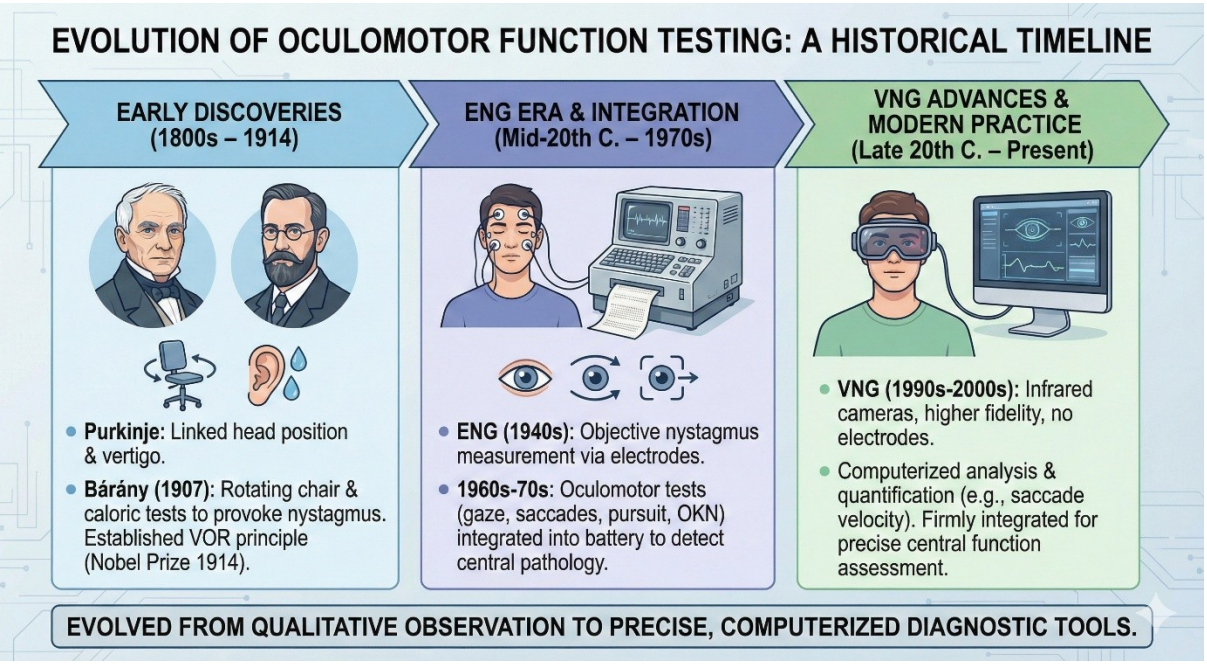


Historical Background

Early Discoveries: The link between eye movements and the vestibular system has been recognized for over a century. In the 1800s, pioneers like Jan Evangelista Purkinje observed that head position changes could induce vertigo and eye movement changes, presaging the concept of positional nystagmus [11, 11]. In 1907, Robert Bárány – often dubbed the father of vestibular science – first employed rotating chair tests and caloric ear irrigations to provoke nystagmus, using the resultant eye movements to evaluate inner ear function [11, 11]. Bárány's techniques

established the principle of assessing vestibular reflexes (especially the vestibulo-ocular reflex) via eye movement observation, for which he was awarded the Nobel Prize in 1914 [11, 11]. His work laid the groundwork for modern vestibular function testing.

Development of ENG and Integration of Oculomotor Tests: By the mid-20th century, technology enabled more quantitative eye movement assessment. **Electronystagmography (ENG),** introduced in the 1940s, used surface electrodes to record corneo-retinal potentials as the eyes moved [3]. ENG allowed objective measurement of nystagmus and other eye movements and quickly became a standard clinical tool for dizzy patients. Early ENG test batteries focused on the vestibulo-ocular reflex (e.g. caloric testing of semicircular canal function) but soon incorporated dedicated oculomotor sub-tests for a more complete evaluation [3]. By the 1960s–1970s, a typical ENG battery included three parts: (1) an oculomotor evaluation (voluntary eye movement tests), (2) positional/positioning tests (looking for nystagmus with head/body position changes), and (3) caloric stimulation of each ear [3]. The oculomotor test portion comprised tasks similar to today's exams: gaze stability (fixation), saccades, pendular smooth pursuit tracking, and optokinetic nystagmus, performed before vestibular-provoked tests [3, 3]. Incorporating these oculomotor assessments was driven by the recognition that certain eye movement abnormalities indicated central pathology (e.g. cerebellar or brainstem lesions), thus helping clinicians differentiate central vestibular.



Advances in Video-Oculography: In the late 20th century, video-based eye tracking (VNG or video-oculography) gradually supplanted ENG. Video goggles with infrared cameras provided higher fidelity recordings of eye movements without the need for electrodes, and could capture vertical and torsional eye motions that ENG electrodes could not. By the 1990s and 2000s, VNG systems became widely adopted, offering improved accuracy and efficiency [7]. The fundamental oculomotor test battery, however, remained a cornerstone – now enhanced by computer-generated visual targets and automated analysis of metrics like saccade velocity and pursuit gain. In current practice, oculomotor function tests are firmly integrated into standard vestibular assessment protocols, performed in nearly every comprehensive dizziness evaluation. They are valued for being low-provocation (causing little discomfort compared to caloric or rotational stimuli) and for yielding clues to central neurologic deficits that might otherwise be missed [7, 7].

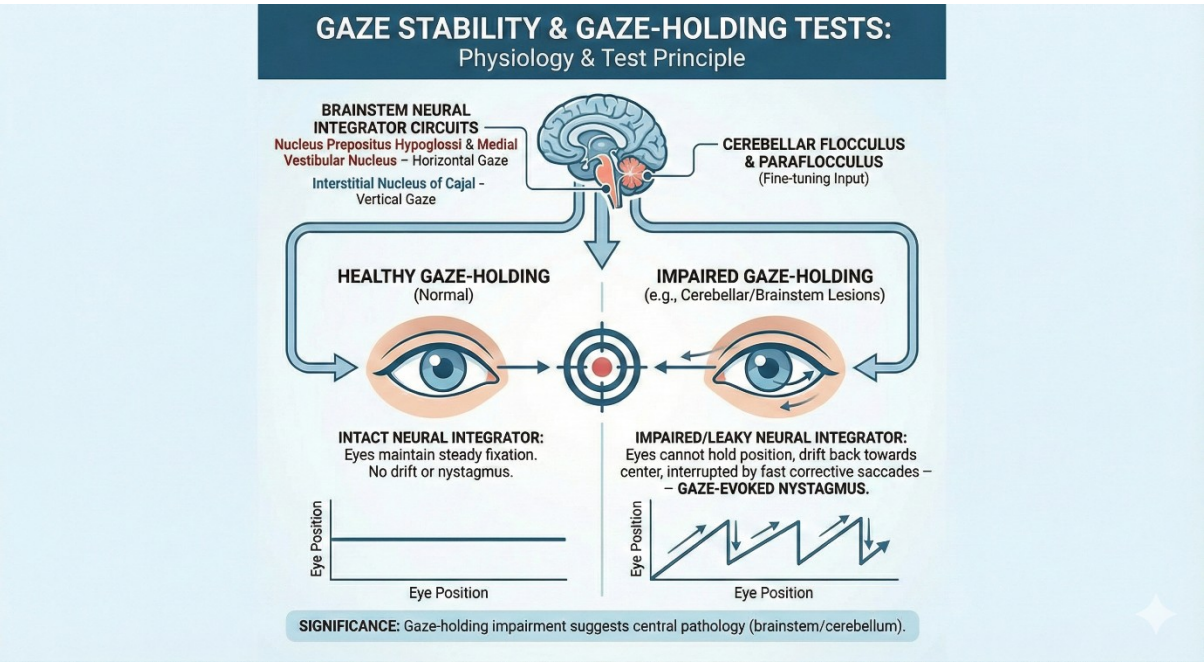
Thus, from Bárány's qualitative observations of nystagmus to today's computerized eye-tracking, oculomotor testing has evolved into a precise and indispensable tool in vestibular diagnostics.

Oculomotor Test Battery: Components and Procedures

Modern oculomotor function testing involves several specific sub-tests, each examining a different aspect of eye movement control. Importantly, these tests are performed with the head stationary (to minimize direct vestibular input), allowing assessment of central ocular motor pathways in isolation [1]. The following are the key oculomotor tests, with an overview of the neuroanatomy/physiology involved, how to perform each test, and guidelines for interpreting results:

Gaze Stability and Gaze-Holding Tests

Physiology: Gaze-holding tests evaluate the ability to maintain steady fixation on a target without developing involuntary drift or nystagmus. This function relies on **brainstem neural integrator circuits (primarily the nucleus prepositus hypoglossi and medial vestibular nucleus for horizontal gaze, and the interstitial nucleus of Cajal for vertical gaze)** to hold the eyes at an eccentric position, as well as **cerebellar flocculus and paraflocculus** input to fine-tune this holding reflex [12, 12]. A healthy gaze-holding mechanism keeps the eyes still when looking steadily at a target. If the neural integrator is leaky or impaired (as in cerebellar or brainstem lesions), the eyes cannot hold position and will drift back toward centre, interrupted by fast corrective saccades – i.e. gaze-evoked nystagmus.

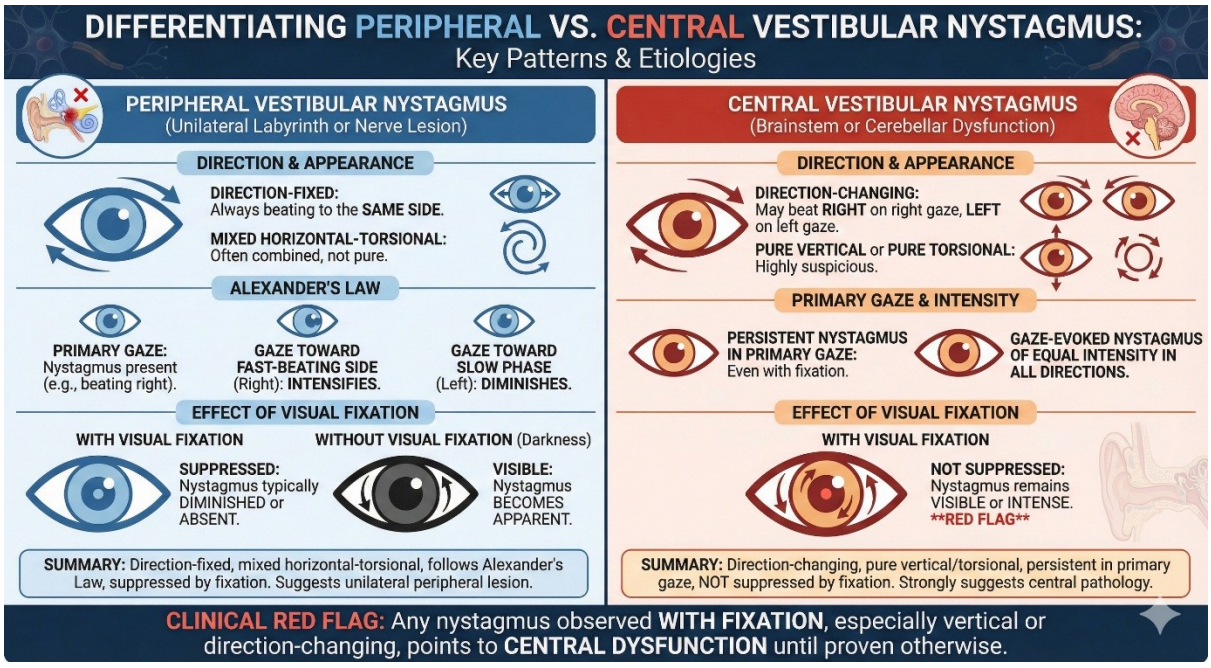


Procedure: The patient is asked to fixate on a stationary target positioned at primary gaze (straight ahead) and at **about 30°** eccentric gaze to the left, right, up, and down. Typically, the test is done twice: with the patient fixating on a visible target (to evaluate gaze holding under normal visual feedback), and again without fixation (in darkness or with the goggles' vision blocked) to uncover any nystagmus that might be suppressed by vision [5, 5]. The clinician or computerized system observes the eyes for any nystagmus (rhythmic jerk movement) or other abnormal eye motion in each gaze position. It is important during lateral gaze tests to remind the

patient not to turn their head – even subtle head movements can introduce false eye drifts [5]. Each gaze is held for several seconds while recording eye position. Any noted nystagmus should be characterized by direction and intensity (slow-phase velocity).

Interpretation: In a normal result, the eyes remain steady (no nystagmus or only minimal "endpoint" nystagmus at far eccentric gaze). Any nystagmus present during gaze fixation on a target is considered abnormal [2]. Nystagmus that appears or worsens when fixation is removed (in darkness) may indicate a peripheral vestibular imbalance or uncompensated lesion, although certain central nystagmus can also increase without fixation. Key patterns help differentiate aetiologies:

- **Peripheral vestibular nystagmus** (from a unilateral labyrinth or nerve lesion) is usually **direction-fixed** (always beating to the same side) and has a **mixed horizontal-torsional appearance.** It often follows **Alexander's law,** meaning it intensifies when gazing toward the fast-beating side and diminishes with gaze toward the slow phase [1]. Crucially, peripheral nystagmus is typically **suppressed by visual fixation**, so it may be visible in darkness but not with the patient fixating on a target [1]. The presence of a spontaneous nystagmus only with fixation removed (and not during fixation) must be interpreted in context of other test results and history [2] – it often signifies a recovering or compensated peripheral lesion.



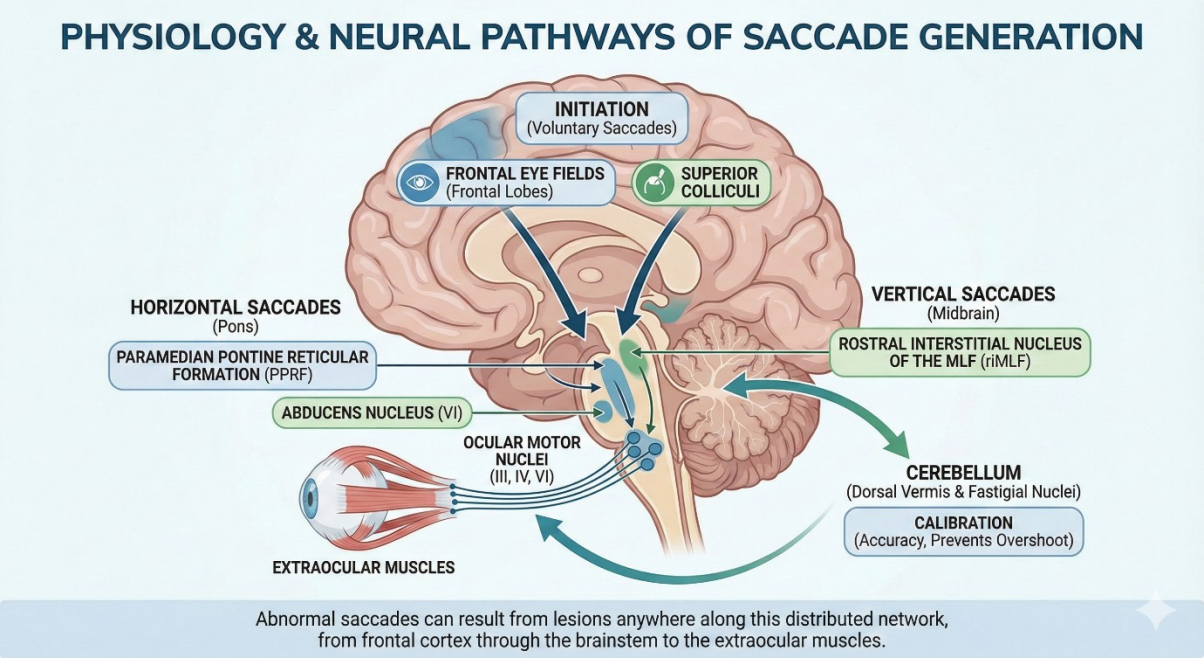
- **Central nystagmus** (due to brainstem or cerebellar dysfunction) often has distinctive features: it can be **purely vertical or purely torsional**, or it may **change direction with gaze** (beating rightward on right gaze, leftward on left gaze), none of which a peripheral lesion can typically produce [1, 1]. A gaze-evoked nystagmus of equal intensity in all directions, or persistent nystagmus in primary gaze despite fixation, strongly suggests a central pathology (e.g. cerebellar degeneration, brainstem stroke, multiple sclerosis). **Failure of visual fixation to suppress nystagmus** is another central red flag – for example, if the patient continues to have obvious nystagmus while trying to fixate on a target, a central vestibular pathway deficit (especially in the cerebellum) should be

suspected [6]. In practice, any nystagmus observed with fixation (especially vertical or direction-changing nystagmus) points to central dysfunction until proven otherwise [1, 1].

In summary, the gaze stability test establishes a baseline: if spontaneous or gaze-evoked nystagmus is present, it will influence other tests. A significant spontaneous nystagmus (particularly with fixation) may contaminate pursuit or saccade testing, so its characteristics must be noted first [5]. Normal gaze holding in all directions, on the other hand, implies intact neural integrators and no overt asymmetry in vestibular tone.

Saccade Testing

Physiology: Saccades are rapid, conjugate eye movements that redirect gaze between targets. They are generated by a network of cortical and brainstem centres. The frontal eye fields (in the frontal lobes) and superior colliculi initiate voluntary saccades, sending signals down to the brainstem saccade generators. Horizontal saccades are driven by burst neuron networks in the paramedian pontine reticular formation (PPRF) and abducens nucleus (pons), while vertical saccades originate from the rostral interstitial nucleus of the medial longitudinal fasciculus (riMLF) in the midbrain. These pathways synapse on the ocular motor nuclei (III, IV, VI) to move the eyes. The cerebellum (especially the dorsal vermis and fastigial nuclei) helps calibrate saccade accuracy (preventing overshoot or sustained oscillations). Because of this distributed circuitry, abnormal saccadic eye movements can result from lesions in a variety of locations from the frontal cortex, through the brainstem, to the extraocular muscles [1]. Key metrics assessed in saccade testing are latency (reaction time from target appearance to movement onset), velocity (peak speed of the eye movement), accuracy (how close the eye lands to the target; undershoot vs overshoot), and conjugacy (coordination between the two eyes) [2, 2].



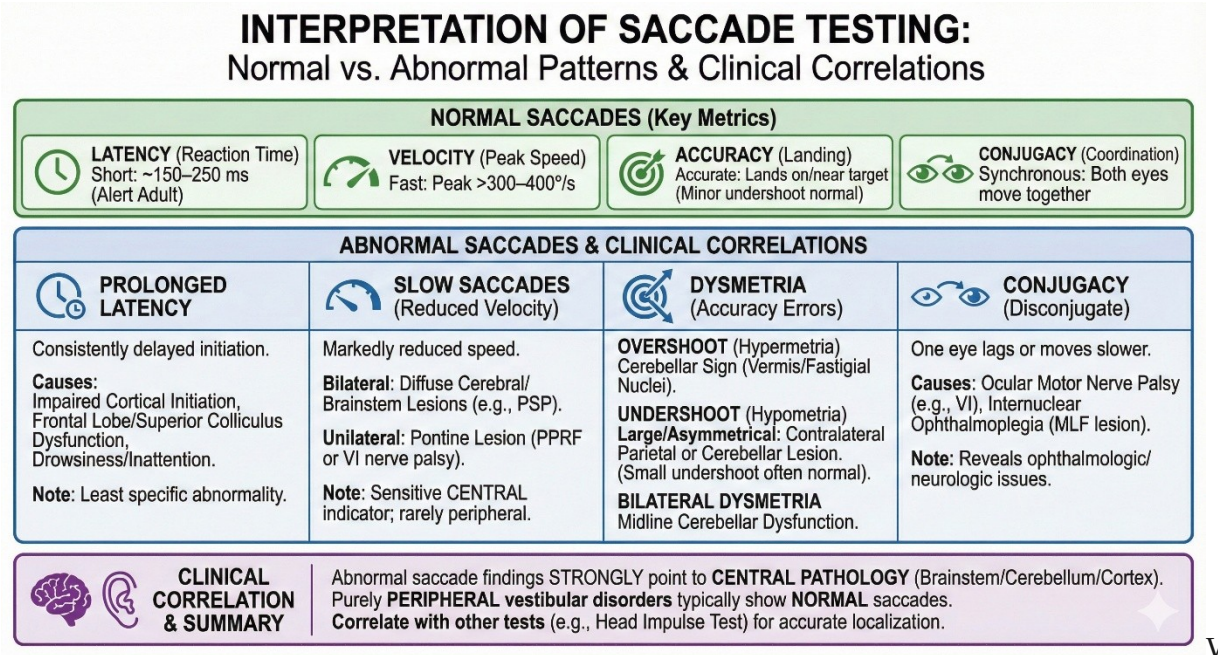
Procedure: The patient is instructed to look back-and-forth between targets that jump to new positions. In a typical computerized saccade test, a dot will randomly jump to positions e.g. 20° left or right of centre at irregular intervals. The patient is told to “follow the jumping dot with your eyes as quickly and accurately as possible” (while keeping the head still). About 20 or more saccades are recorded in each direction to ensure a reliable sample [5]. Some protocols also

test vertical saccades (up/down) if equipment and normative data allow. During the test, the system will record eye movement waveforms and calculate latency, peak velocities, and end-point accuracy for each saccade, often averaging them for analysis. The clinician observes whether the eyes reach the target in one clean motion or if they under- or overshoot and require corrective movements.

Interpretation: Normal saccades are fast (peak velocity often >300–400°/s for large amplitude targets), accurate (the eye lands on or very near the target with perhaps a minor undershoot), and have a short reaction time (latency ~150–250 ms in an alert adult) [5, 5]. Both eyes move together synchronously. Mild undershoot (a small corrective saccade) is not uncommon even in healthy individuals, especially in older age, but systematic overshooting (hypermetria) or large, inconsistent undershoots (hypometria) are abnormal [2, 2]. The presence of significant saccadic dysmetria should be noted as either bilateral (in both directions) or unilateral (only on saccades to one side), as asymmetric findings are more localizing for focal lesions [2].

Specific saccadic abnormalities and their correlations include:

- **Prolonged latency:** A consistently delayed initiation of saccades (far beyond normal range) can indicate **impaired cortical initiation or attention**. While latency can be affected by drowsiness or poor cooperation, pathologic latency prolongation may reflect frontal lobe or superior colliculus dysfunction. It is considered the least specific saccade abnormality, as it can also be due to medications or inattention [2].
- **Slow saccades (reduced velocity):** Markedly slow saccades are a very sensitive indicator of neurological dysfunction. **Diffuse cerebral or brainstem lesions, such as progressive supranuclear palsy (PSP) or neurodegenerative diseases, often cause bilaterally slow saccades. A unilateral decrease** in saccade velocity (one direction markedly slower) may point to a **pontine lesion** affecting the PPRF or abducens nerve/nerve palsy on that side [1, 2]. Importantly, saccadic slowing is rarely caused by a peripheral vestibular lesion; it almost always signals central pathology (or a severe peripheral ocular motor nerve palsy or myopathy), so this finding is “a very powerful indicator” of central neurologic disease [2].
- **Dysmetria (accuracy errors): Overshoot** (eyes go past the target and then come back) is typically a cerebellar sign (particularly implicating the cerebellar vermis or fastigial nuclei) [1]. **Undershoot** is more common and less specific – small undershoots can be normal, but large hypometria, especially if asymmetrical, may indicate a contralateral parietal lobe or cerebellar lesion. Consistent dysmetria in both directions (and vertically) often points to midline cerebellar dysfunction. If saccades overshoot to the right and undershoot to the left, for example, a lesion in the right cerebellar hemisphere (which overcorrects rightward movements and under-drives leftward movements) might be suspected.
- **Conjugacy:** Normally both eyes move together. A **disconjugate saccade** (one eye lagging or moving slower) suggests an ocular motor nerve palsy or internuclear ophthalmoplegia (e.g. a lesion in the medial longitudinal fasciculus causing an adduction lag). The saccade test can thus incidentally reveal ophthalmologic or neurological issues such as an abducens (VI) palsy if one eye cannot move fully outward.



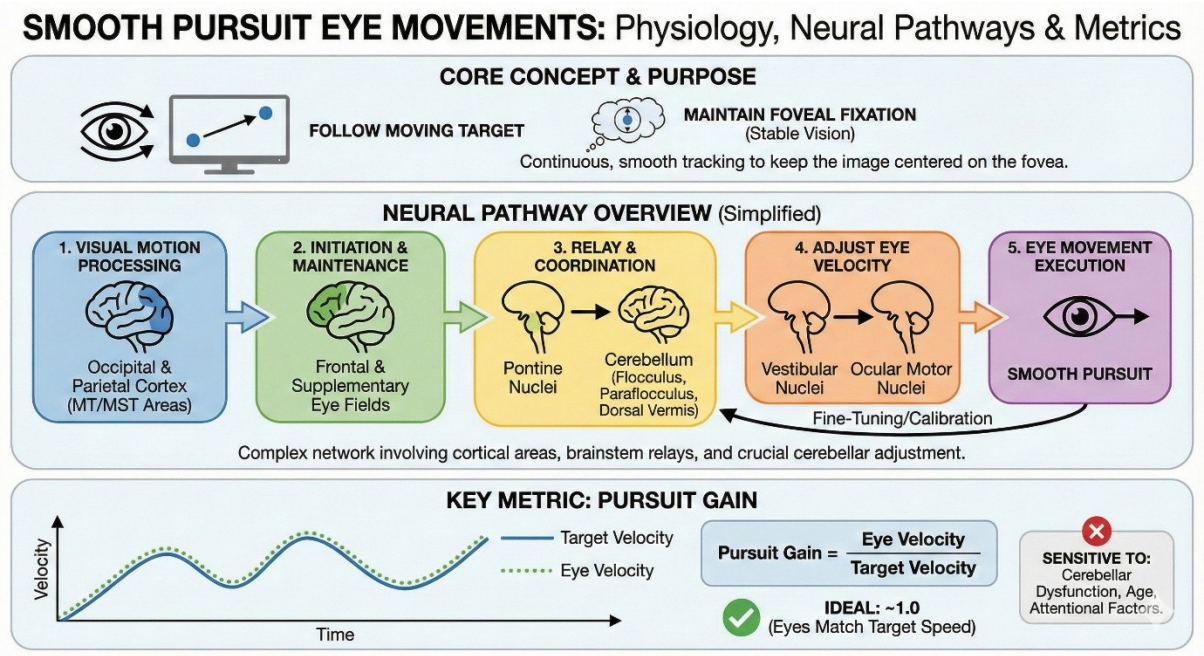
When interpreting saccades, the examiner should compare performance to age norms and look for consistent patterns. Central lesions in the brainstem or cerebellum commonly produce combinations of the above abnormalities (e.g. slow and inaccurate saccades) [5, 2]. For example, a patient with a pontine stroke affecting the PPRF might have slow, hypometric horizontal saccades toward the side of the lesion. A degenerative cerebellar disorder could cause saccadic intrusions (multiple catch-up jerks during what should be one saccade) or hypermetria. In contrast, peripheral vestibular disorders do not directly affect voluntary saccade generation, so saccade testing is usually normal in purely peripheral vestibulopathies (aside from possible small inaccuracies due to concurrent nystagmus or age). Thus, abnormal saccade findings strongly point to central pathology and often warrant further neurologic investigation. In practice, clinicians correlate saccade results with other tests – for instance, a unilateral vestibular neuritis patient would have normal saccades but an abnormal head impulse test, whereas a patient with central vertigo (stroke) might have conspicuously abnormal saccades and a normal head impulse test.

Smooth Pursuit (Visual Tracking)

Physiology: Smooth pursuit eye movements allow the eyes to follow a moving target smoothly to maintain the image on the fovea. This continuous tracking is a **complex function that involves multiple regions: the visual motion processing areas of the occipital and parietal cortex (especially middle temporal and medial superior temporal areas) detect target motion; the frontal eye field and supplementary eye field contribute to initiating and maintaining pursuit; the pontine nuclei relay these signals to the cerebellum (particularly the flocculus, paraflocculus, and dorsal vermis), which in turn adjusts eye velocity via connections to the vestibular nuclei and ocular motor nuclei.** The pursuit system is thus highly sensitive to cerebellar dysfunction and age-related degeneration, as well as to attentional factors [2, 5]. Pursuit gain (eye velocity divided by target velocity) should ideally be close to 1.0 (eyes matching target speed).

Procedure: The patient is instructed to visually track a target that moves smoothly back and forth. Typically, a dot moves sinusoidally or in a triangle wave horizontally across a screen (e.g. ±20°) at varying speeds. A standard protocol might test a range of frequencies (e.g. 0.2 Hz up to 0.4 Hz), or simply a slow and a faster condition. The instruction is “follow the moving dot with your eyes, keeping your head still, and try to stay directly on the target without lagging or jumping

ahead." The test usually lasts around 20–40 seconds, during which the target accelerates to a faster speed [5, 5]. Eye position is recorded continuously, and the software computes pursuit gain and sometimes phase lag. The examiner observes whether the eye movement is smooth or whether there are corrective saccades interrupting it (a phenomenon known as **saccadic or "cogwheel" pursuit).** Both horizontal pursuits (and sometimes vertical) are tested; note that vertical pursuit often is more variable and many clinics focus on horizontal tracking where normative data are strongest [5]. If vertical pursuit is tested, it should be interpreted cautiously due to lack of extensive normative values.

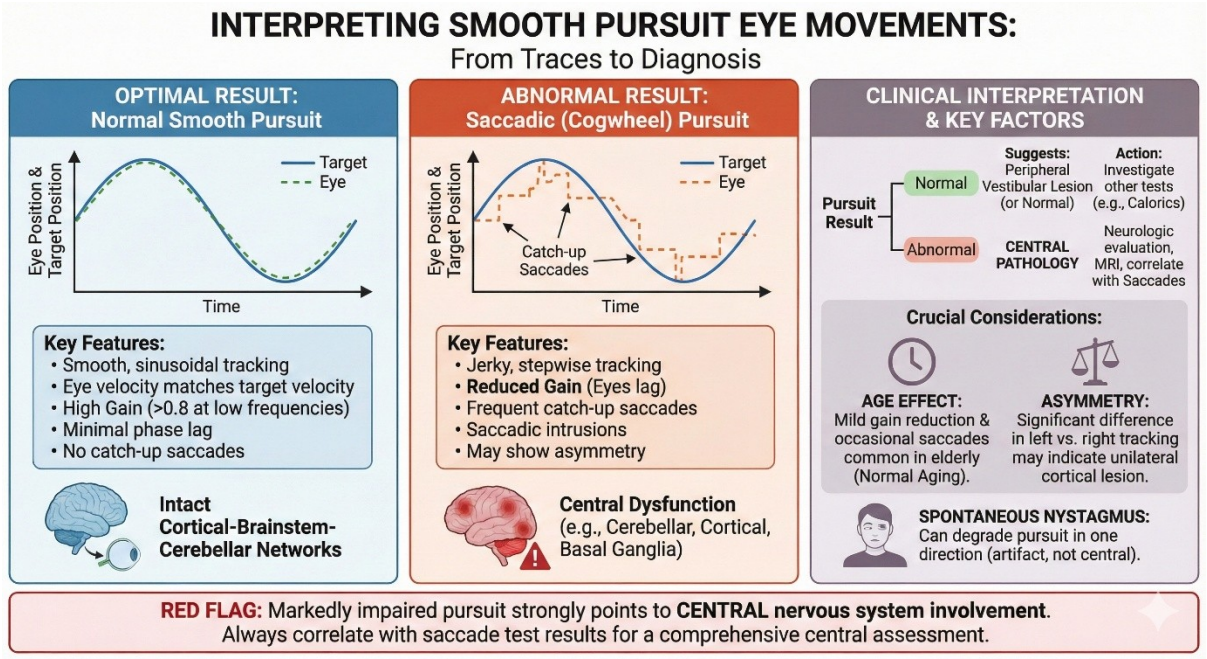


Interpretation: In an optimal result, the eyes smoothly follow the target with near-equal speed (normal gain typically >0.8 at low frequencies) and minimal phase lag. The eye trace will appear as a clean sinusoidal curve closely overlaying the target's motion curve. Abnormal smooth pursuit is characterized by reduced gain (the eyes lag behind the target, requiring catch-up saccades) or, less commonly, by an inability to decelerate (leading to jump-ahead or back-up saccades). On visual inspection, this appears as a jerky, stepwise tracking instead of fluid motion. Such "saccadic pursuit" (also called cogwheel pursuit) is a hallmark of central dysfunction in the pursuit pathways, often localized to the cerebellum or cortical visual motion areas. For example, degeneration in the cerebellar flocculus/paraflocculus (as in some spinocerebellar ataxias) can greatly impair pursuit gain. Likewise, aging has a well-documented effect on pursuit: elderly patients often show mildly reduced pursuit gain and occasional catch-up saccades even without overt disease [5, 2]. Thus, age must be taken into account – what is abnormal in a 30-year-old may be borderline normal in an 80-year-old. Most commercial systems provide age-stratified normative values for pursuit gain and phase [2, 2].

Critically, smooth pursuit is predominantly a central ocular motor function and is not significantly affected by peripheral vestibular lesions [5]. A unilateral vestibular loss, for instance, does not degrade smooth pursuit gain in a well-compensated patient (aside from interference by any spontaneous nystagmus). If a patient has a severe spontaneous nystagmus, pursuit to one side (opposite the nystagmus fast phase) can appear degraded simply because the baseline eye movement is disturbed; but in the absence of such nystagmus, a peripheral labyrinthine disorder yields normal smooth pursuit [5]. Therefore, markedly impaired pursuit strongly suggests a

central pathology. Common causes of pursuit impairment include cerebellar disorders, basal ganglia diseases, cortical lesions in parietal/temporal lobes, and diffuse encephalopathy. For example, an individual with a parietal stroke might have asymmetric pursuit (worse when tracking toward the side of the lesion). In degenerative conditions like progressive supranuclear palsy, pursuit can be profoundly disturbed (along with saccades).

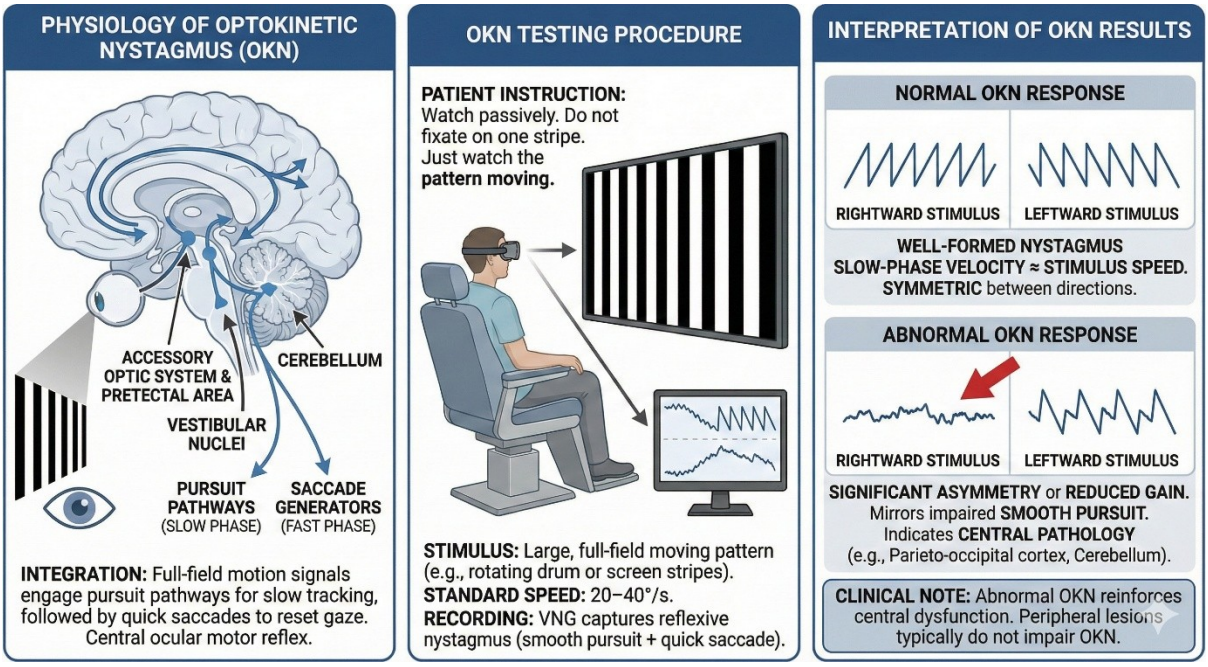
When interpreting pursuit, one also notes symmetry: Is pursuit gain equally reduced for leftward vs rightward tracking? Significant asymmetry might indicate a unilateral cortical lesion. Additionally, the presence of saccadic intrusions during pursuit (frequent catch-up saccades despite normal overall gain) can occur in early cerebellar disease – some devices quantify this as "percentage of saccadic intrusions" or a spectral purity measure [2]. In summary, normal smooth pursuit implies intact cortical-brainstem-cerebellar tracking networks, whereas saccadic or low-gain pursuit is a red flag for central nervous system involvement (keeping in mind that mild pursuit deficits in older patients may be age-related). Any pursuit abnormality should be interpreted alongside saccade test results: often both pursuit and saccades will be abnormal in a central disorder (e.g. cerebellar ataxia might show both pursuit breakdown and saccadic dysmetria), reinforcing the central lesion conclusion.



Optokinetic Nystagmus (OKN) Testing

Physiology: Optokinetic nystagmus is a reflexive eye movement that occurs when the entire visual field moves continuously, as when looking out of a moving train. It consists of a smooth pursuit movement in the direction of the visual field motion, followed by a quick saccade in the opposite direction to reset the eye – resulting in a repeating nystagmus. The OKN reflex serves to stabilize the image during sustained head rotation or when the environment moves around us. Neuroanatomically, OKN arises from integration of the pursuit pathways with subcortical optokinetic pathways. Visual motion signals (especially from the retinal periphery) project to the accessory optic system and nuclei in the pretectal area, which then engage the vestibular nuclei and cerebellar pathways to generate slow-phase eye movement. In essence, OKN tests similar circuitry as smooth pursuit (for the slow phase) and saccade generation (for the quick phase). It is therefore also a test of central ocular motor function.

Procedure: The patient is exposed to a large field moving visual stimulus and asked to watch it passively (or told to "look at the moving stripes"). Common methods include a rotating drum with vertical stripes or a projector/screen displaying moving vertical stripes or a series of dots. Typically, one tests OKN in both directions (e.g. stripes moving leftward, then rightward), at a standard speed (e.g. 20–40°/s). As the field moves, the patient's eyes should reflexively track some stripes then jump back repetitively, producing nystagmus. The examiner records this nystagmus, measuring its velocity and symmetry. Modern VNG systems generate full-field OKN stimuli on a screen. It's important that the stimulus fills most of the visual field to evoke a robust OKN (small stimuli would just be pursuit). The patient is usually instructed not to deliberately follow one target (to avoid turning it into a pursuit task), but rather to "just watch the pattern moving."

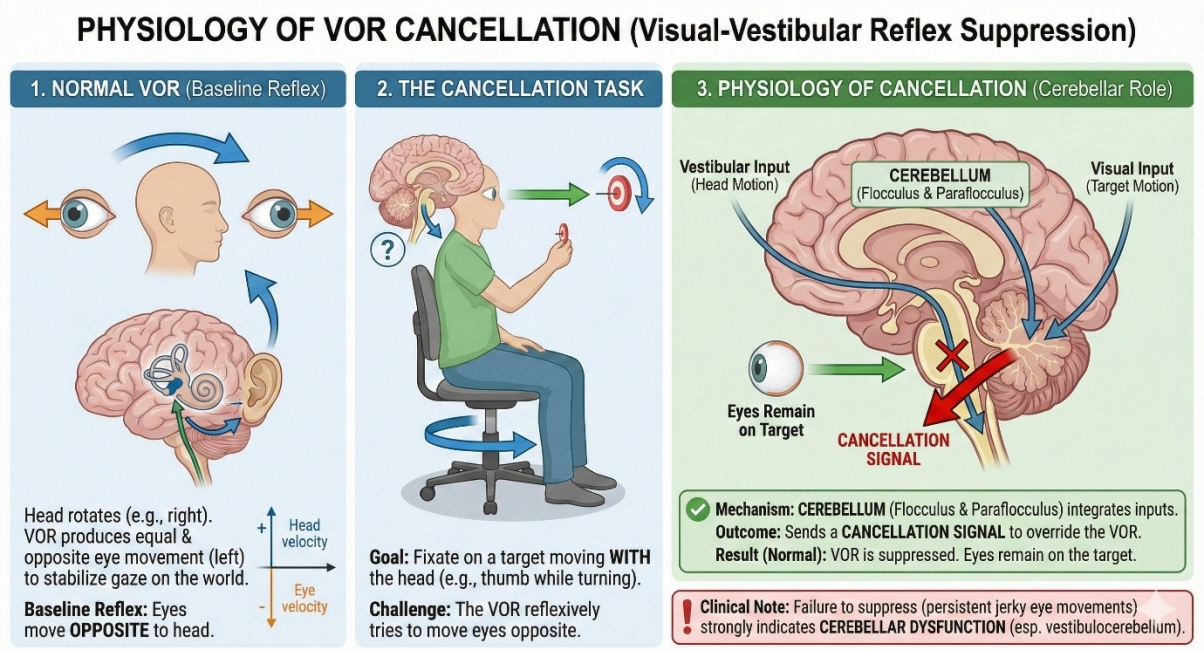


Interpretation: A normal OKN response consists of a well-formed nystagmus with slow-phase velocity that approaches the speed of the stimulus. The response should be fairly symmetric between clockwise vs counterclockwise drum rotation (or leftward vs rightward motion). OKN is essentially a culmination of smooth pursuit performance – if pursuit is impaired, the slow phase of OKN will likewise be impaired [18]. For example, if a patient cannot track smoothly to the right, then an OKN stimulus moving to the right will generate an irregular or low-gain slow phase to the right. Thus, OKN test results often mirror smooth pursuit ability. Significant asymmetry in OKN nystagmus (strong in one direction, weak in the other) is abnormal and could indicate a unilateral lesion in the neural pathways (for instance, a lesion in the left parieto-occipital region might reduce OKN when the stripes move toward the affected side). However, moderate OKN asymmetry can also be influenced by a pre-existing spontaneous nystagmus or poor cooperation. Clinically, optokinetic testing is somewhat less frequently emphasized than saccades and pursuit, since it does not usually yield unique information beyond what those tests show. Still, it can reinforce findings: for instance, a patient with cerebellar impairment may show both saccadic pursuit and a deficient OKN response. Conversely, a purely peripheral vestibular lesion should not affect OKN (aside from perhaps a slight asymmetry if spontaneous nystagmus is superimposed). Therefore, like pursuit, abnormal OKN responses indicate central pathology. One specific use of OKN is to ensure that both directions of cortical pursuit pathways are functional – if a patient has a unilateral cortical lesion, they may have selective difficulty with one

direction of OKN slow phase. In summary, an intact OKN (with good nystagmus generation in both directions) further supports normal central ocular motor function, whereas an absent or asymmetrically reduced OKN is a sign of central dysfunction (assuming adequate stimulus and patient alertness).

VOR Cancellation (VOR Suppression Test)

Physiology: VOR cancellation (also called visual-vestibular reflex cancellation or suppression) assesses the ability of the cerebellum to override the vestibulo-ocular reflex. Normally, the VOR produces eye movement equal and opposite to head movement to stabilize gaze. However, if one is moving the head and the object of interest is moving with the head (for example, if you fixate on your thumb and turn your head, or if you are on a rotating chair looking at a dot that rotates with you), a normal person can suppress the VOR to keep the eyes on the target. This ability depends on the cerebellar flocculus and paraflocculus, which receive both vestibular and visual inputs and can cancel out the VOR-generated eye movement by sending an equal and opposite signal. A failure to suppress the VOR (i.e. eyes continue to reflexively jerk during head motion despite a visible target) is a strong indicator of cerebellar dysfunction, particularly of the vestibulocerebellum.

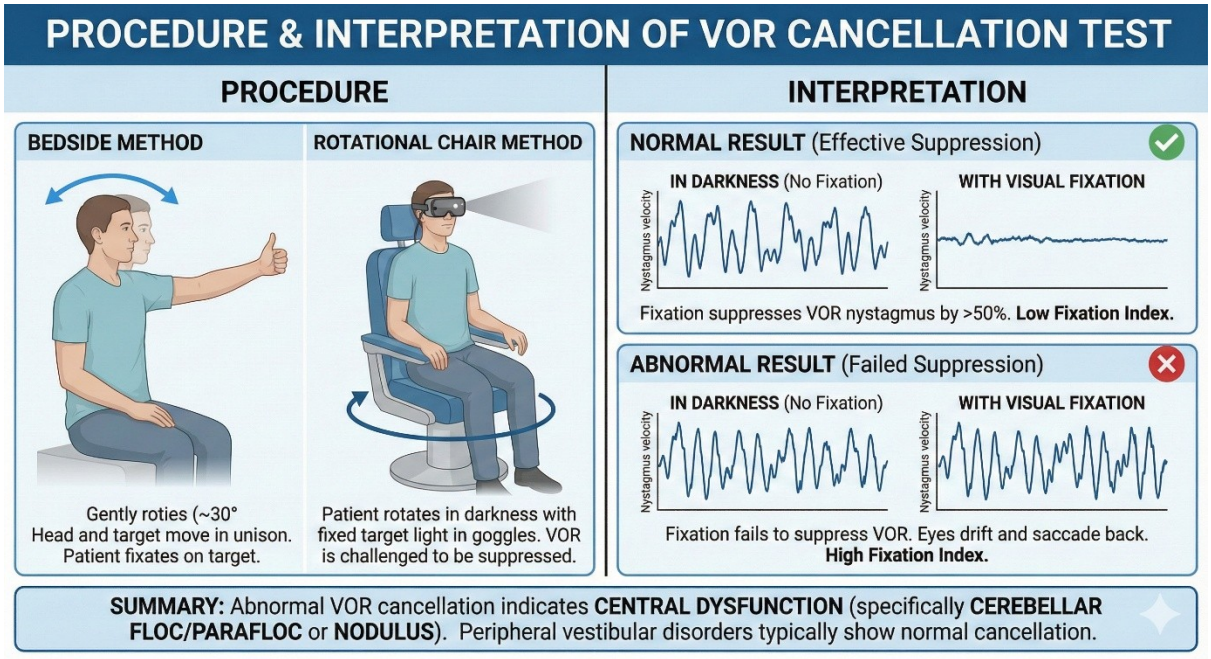


Procedure: There are a couple of ways to test VOR cancellation:

- **Bedside method:** The patient extends their arm and fixates on their thumb (or the examiner holds a small target in front of the patient at arm's length). The patient and target are moved together – for example, the examiner can gently rotate the patient's torso or head back and forth ~30° while the patient keeps staring at the target (which moves with them). Another method is the patient holds a visual target and actively rotates their trunk or head side to side, keeping eyes on the target. In both cases, the head and target move in unison, requiring cancellation of the VOR.
- **Rotational chair method:** If available, computerized rotary chair systems include a VOR suppression test. The patient is rotated in darkness to elicit VOR nystagmus, then a fixed target light is turned on inside the goggles or a head-mounted laser – the patient fixates

on this target that rotates with them. Normally, fixation on the moving target will greatly reduce the nystagmus (VOR gain drops). The eye movement is recorded to quantify how much the nystagmus is suppressed in light versus darkness [6, 6].

Interpretation: In a normal individual, fixation on a moving target should significantly suppress or eliminate nystagmus caused by head rotation. For example, after standard caloric irrigations (which induce nystagmus), allowing the patient to fixate on a visual target typically reduces the nystagmus intensity by >50%; failure of this fixation suppression indicates a central problem [6]. Thus, an abnormal VOR cancellation test is when the patient cannot keep their eyes locked on the moving target during head movement – instead, their eyes continue to be driven off target by the VOR, requiring catch-up saccades. Clinically, one might see nystagmus (e.g. if rotating to the right, the eyes show a left-beating nystagmus even though the patient is trying to look at a target moving with them). This suggests the cerebellum is not appropriately cancelling the reflex. The most common cause for VOR cancellation failure is a lesion in the cerebellar flocculus/paraflocculus or nodulus (as seen in cerebellar degeneration or vestibular cerebellar syndromes). It is a classic finding in disorders like cerebellar ataxia and some degenerative conditions (e.g. Arnold-Chiari malformation, spinocerebellar ataxias) – patients will have difficulty with this task and often report that they cannot keep eyes on target. In contrast, patients with purely peripheral vestibular lesions (normal cerebellum) can usually perform VOR cancellation normally, because their issue is with the VOR input itself, not the ability to suppress it.



In quantitative terms, rotational chair systems calculate a fixation index – the ratio of nystagmus slow-phase velocity with fixation to that without fixation. A high fixation index (i.e. little change with fixation) is abnormal, indicating central vestibular pathway involvement [6]. At the bedside, one simply notes if the patient's eyes stay on the target or if they drift. Any observed nystagmus or inability to maintain fixation during the head movement is considered a failure of VOR suppression and a central sign. This test nicely complements the gaze holding test: if a patient demonstrates, say, gaze-evoked nystagmus and also cannot cancel the VOR, a cerebellar lesion becomes a very strong possibility.

In summary, normal VOR cancellation requires an intact cerebellum; an abnormal result is almost always due to a central dysfunction, since peripheral vestibular disorders do not affect this suppression mechanism (in fact, with a reduced VOR from a peripheral lesion, there may be less need for cancellation). VOR cancellation is sometimes tested as part of a full rotational chair battery (often termed a Visual-Vestibular Fixation test) and is interpreted alongside other ocular motor tests for a comprehensive central assessment [6, 6].

*(Note: Additional oculomotor tests sometimes included are **fixation index measurement during caloric nystagmus, vergence eye movement testing, and ocular alignment tests (skew deviation)**, but these are beyond the core scope of standard oculomotor function testing. The primary tests listed above – gaze, saccade, pursuit, OKN, and VOR suppression – form the core oculomotor battery in vestibular assessments.)*

Common Abnormalities and Clinical Correlations

Oculomotor testing yields characteristic abnormal patterns that help distinguish central vestibular system disorders from peripheral inner-ear pathologies. Below are common abnormalities observed and their clinical significance:

- **Jerky or "cogwheel" smooth pursuit:** The presence of saccadic interruptions during pursuit (low pursuit gain) strongly indicates a central lesion, often in the cerebellum or cortical visual pathways. This is commonly seen in patients with **degenerative cerebellar diseases, advanced age (in milder form), or parietal lobe lesions.** In a patient with vertigo, saccadic pursuit suggests a central vestibular disorder rather than an isolated inner ear problem [5]. For example, an elderly patient with **small vessel ischemia in the pons or cerebellum may have bilateral pursuit impairment.** By contrast, pursuit is typically normal in peripheral vestibular disorders (like vestibular neuritis or Ménière's disease) [5]. Thus, pursuit abnormality shifts diagnostic consideration toward central causes such as stroke, multiple sclerosis, or drug effects (e.g. sedatives).
- **Saccadic dysmetria or slowing:** Overshooting or notably slow saccades are telltale signs of central involvement. **Cerebellar lesions cause hypermetric saccades** (eyes overshoot) or even **square-wave jerks (small saccadic intrusions on fixation)** [1, 1]. **Pontine or midbrain lesions can cause slow saccades or selective loss of saccades** in one plane (for instance, a paramedian pontine reticular formation lesion can nearly abolish horizontal saccades toward the lesion side). Slow saccades may also be seen in certain peripheral neuropathies or ocular muscle disorders (affecting the effector mechanism), but in vestibular practice they more often signify central pathology such as a brainstem degenerative condition [2]. Patients with **progressive supranuclear palsy (PSP),** for example, exhibit slow vertical saccades and often abnormal pursuit, pointing to a central neurodegenerative process rather than an ear disorder [5, 5].
- **Gaze-evoked nystagmus and direction-changing nystagmus:** As described, if a patient cannot hold eccentric gaze and develops gaze-evoked nystagmus (especially if it beats in the direction of gaze and changes direction when gaze changes), that usually indicates a central lesion – often in the **cerebellum** (e.g. a lesion of the vestibulocerebellum) or **brainstem nuclei** [1, 1]. One common example is **drug-induced nystagmus** (certain anticonvulsants or sedatives can cause gaze-evoked nystagmus by affecting cerebellar function). **Downbeat nystagmus** (persistent downward drifting of eyes, often present in primary gaze) is a classic central sign found in craniocervical

junction lesions (e.g. Chiari malformation) or cerebellar degenerations – it would appear during gaze tests and is not attributable to any peripheral vestibular lesion. Pure torsional or pure vertical nystagmus are virtually always central in origin [1] (the only exception being certain rare peripheral conditions like bilateral superior canal dehiscence causing transient downbeat nystagmus, which is an outlier [1]). In contrast, peripheral nystagmus has a fixed direction (usually horizontal with slight torsion) and obeys Alexander's law; it will not suddenly change direction when the patient looks in different directions (except slight intensification to one side) [1]. So, observing direction-changing nystagmus on gaze tests immediately raises concern for a central lesion (e.g. a brainstem stroke or drug effect).

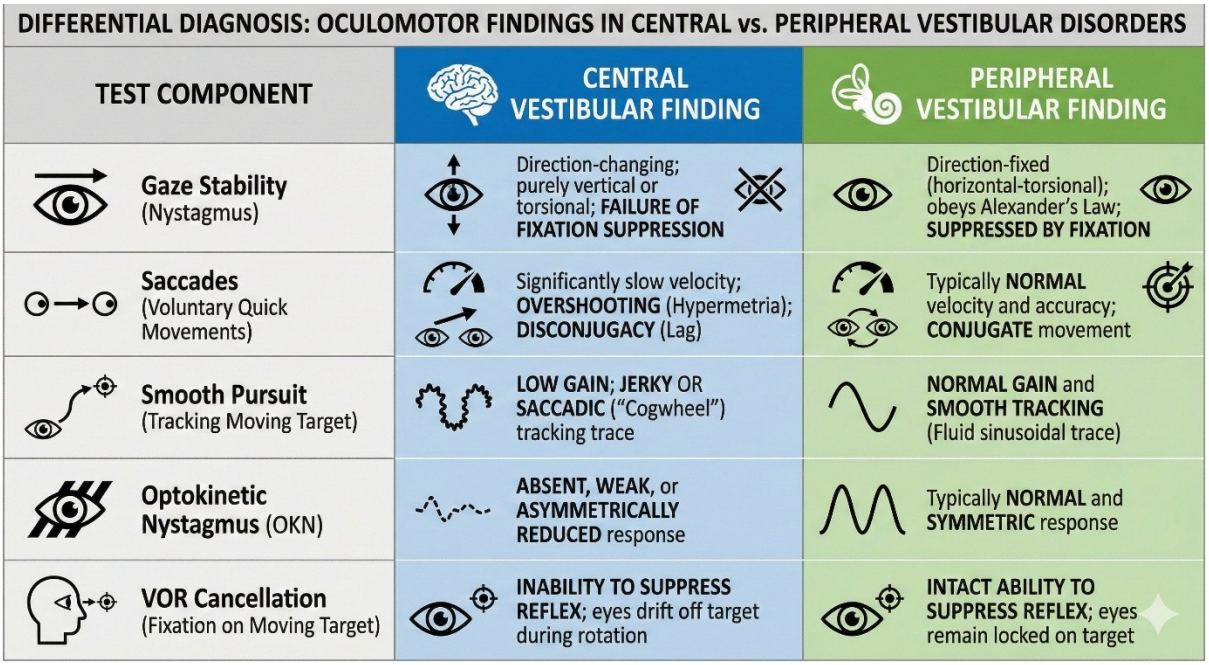
- **Impaired VOR cancellation (failure of fixation suppression):** If during the VOR cancellation test the patient's eyes cannot stay on the moving target (i.e. nystagmus persists), it implies a lesion in the pathways that integrate visual and vestibular information – specifically the cerebellum. This finding is commonly associated with cerebellar strokes or degeneration. Clinically, a patient with an acute cerebellar stroke (involving the flocculus) may have severe gait ataxia and also an inability to suppress vestibular nystagmus, whereas a patient with a peripheral vestibular neuritis would have no trouble with VOR cancellation once the acute nystagmus subsides. Thus, abnormal fixation suppression is a marker of central pathology and often correlates with other cerebellar signs (dysmetria on finger-nose testing, ataxia, etc.) [6].
- **Saccadic intrusions and oscillations:** Though not always formally tested, the presence of abnormal fast eye movement intrusions **(like square-wave jerks, ocular flutter, or macrosaccadic oscillations)** during any portion of oculomotor testing is a sign of central dysfunction (often cerebellar or brainstem). For example, square-wave jerks (spontaneous small saccades away from and back to target) might be seen when the patient is attempting to fixate; this is linked to **cerebellar or Parkinsonian disorders** [1]. These would be noted qualitatively in the examination and support a central cause of dizziness.

In summary, central vestibular disorders (e.g. ischemia in the posterior circulation, demyelinating disease, cerebellar atrophy, encephalopathy) often produce a combination of oculomotor abnormalities – such as gaze-evoked nystagmus, saccadic pursuit, dysmetric or slow saccades, failure of fixation suppression, etc. These tests thus help localize the "lesion site" to central pathways [1, 1]. Peripheral vestibular disorders (e.g. vestibular neuritis, unilateral labyrinthine loss, benign paroxysmal positional vertigo) characteristically do not cause pursuit or saccade abnormalities – their signature is a unidirectional nystagmus that increases with fixation removed and other vestibular-reflex deficits, while the oculomotor examination remains normal [5, 1]. Any deviation from that (for instance, a patient with what looks like peripheral vestibular nystagmus who also has broken pursuit or other central signs) should prompt reconsideration of a possible central lesion or a dual pathology.

Table 1: Comparison of Oculomotor Test Findings

Test	Central Vestibular Finding	Peripheral Vestibular Finding

Component		
Gaze Stability	Direction-changing; purely vertical or torsional; failure of fixation suppression.	Direction-fixed (horizontal-torsional); obeys Alexander's Law; suppressed by fixation.
Saccades	Significantly slow velocity; overshooting (hypermetria); disconjugacy (lag).	Typically, normal velocity and accuracy; conjugate movement.
Smooth Pursuit	Low gain; jerky or saccadic ("cogwheel") tracking trace.	Normal gain and smooth tracking (fluid sinusoidal trace).
Optokinetic Nystagmus	Absent, weak, or asymmetrically reduced response.	Typically, normal and symmetric response.
VOR Cancellation	Inability to suppress reflex; eyes drift off target during rotation.	Intact ability to suppress reflex; eyes remain locked on target.



It is important to consider that certain non-pathologic factors can affect oculomotor test results: advanced age (reducing pursuit gain and saccade accuracy), medications (e.g. tranquilizers, anti-seizure drugs can cause nystagmus or slow eye movements), fatigue and inattention (can prolong saccade latency or degrade pursuit) [2]. Experienced clinicians interpret test findings in the context of these factors and the overall clinical picture. Nonetheless, distinct abnormalities on oculomotor tests are highly informative. For example, if a patient with acute vertigo shows normal head impulse test (no catch-up saccades, implying central), direction-changing gaze nystagmus, and skew deviation, one strongly suspects a brainstem stroke – this HINTS paradigm (Head Impulse, Nystagmus, Test of Skew) is essentially leveraging bedside oculomotor signs to detect central emergencies. In routine vestibular clinic practice, the detailed oculomotor battery similarly helps categorize a dizziness case as likely peripheral or central, guiding whether to pursue central imaging studies or focus on inner ear therapies.

Integration of Oculomotor Findings with Other Vestibular Tests

Vestibular physicians synthesize oculomotor test results with other elements of the vestibular test battery (caloric irrigation, head impulse tests, vestibular evoked myogenic potentials, rotational chair, etc.) to arrive at a comprehensive diagnosis. Each test provides a piece of the puzzle:

- **Caloric Testing:** Calorics isolate low-frequency **(≈0.003 Hz)** horizontal semicircular canal function by inducing nystagmus with temperature changes. A unilateral caloric weakness indicates a peripheral vestibular loss on that side [1, 1]. Oculomotor tests complement this by indicating if central compensation is present. For instance, in a unilateral vestibular neuritis patient, calorics might show a reduced response on the affected side, while oculomotor tests are entirely normal (since the lesion is peripheral). This pattern (peripheral deficit + normal ocular motor) reinforces a diagnosis of peripheral vestibulopathy. If, however, the same patient also had an abnormal smooth pursuit or gaze nystagmus, one would question if there is a concurrent central issue delaying compensation or a separate central lesion. Oculomotor testing also assesses compensation status: a caloric weakness with a persistent spontaneous nystagmus on gaze tests implies incomplete central compensation, whereas if gaze tests show no spontaneous nystagmus (with fixation) months after an acute lesion, it suggests the brainstem and cerebellum have compensated for the imbalance [1]. Additionally, the **fixation suppression test** during calorics (asking the patient to fixate on a target after caloric-induced nystagmus) is an integration of oculomotor and vestibular responses – failure to suppress caloric nystagmus indicates a central lesion in the vestibulo-ocular pathways [6]. Thus, caloric results and oculomotor results together can differentiate a simple peripheral hypofunction from a combined peripheral+central disorder.
- **Video Head Impulse Test (vHIT):** vHIT evaluates high-frequency **(≈5 Hz)** VOR function of each semicircular canal by measuring eye responses to rapid, small-amplitude head impulses. It primarily detects peripheral vestibular deficits (reduced VOR gain with corrective saccades). In a classic peripheral vestibular loss, vHIT will be abnormal for the affected canals, but the patient's oculomotor (central) tests will be normal. This combination – abnormal vHIT with normal pursuit/saccades – is a strong indicator of an isolated peripheral lesion (e.g. superior vestibular neuritis affecting the horizontal and anterior canals) [11, 11]. Conversely, if vHIT is normal (suggesting intact peripheral VOR) but oculomotor tests show central signs, one should suspect a central vestibular pathway disorder (e.g. a vestibular migraine or brainstem lesion) that can cause dizziness without impairing the peripheral end-organs. In acute vestibular syndrome, the presence of normal head impulse test but direction-fixed nystagmus often suggests a stroke (central) – known in emergency medicine as an important dangerous sign. Moreover, vHIT can sometimes reveal catch-up saccades which themselves are eye movements assessed in context: if those corrective saccades are abnormally slow or disconjugate, it may hint at an overlapping central issue, although vHIT primarily focuses on peripheral function.
- **Vestibular Evoked Myogenic Potentials (VEMP):** VEMPs test otolith (saccule and utricle) function via reflexes to neck or eye muscles. These are largely peripheral tests. Integration comes in interpreting whether a patient's pattern of deficits is purely peripheral. For example, a patient with bilateral loss of caloric responses, bilaterally abnormal vHIT, and absent VEMPs likely has bilateral peripheral vestibulopathy. If that patient's ocular motor tests are normal, it further supports a peripheral aetiology (like ototoxic damage).

In contrast, if a patient has inconsistent vestibular test findings (say normal VEMPs but abnormal ocular motor signs and poor pursuit), one might suspect a central cause of imbalance (since central lesions won't affect VEMP which is a peripheral reflex). Oculomotor tests can also detect central adaptation to otolith disorders; for example, skew deviation (a vertical misalignment of the eyes often tested with alternate cover test) is a sign of an acute otolith imbalance or brainstem involvement, linking ocular motor exam with vestibular otolith function in conditions like lateral medullary (Wallenberg) syndrome.

- **Rotational Chair Testing:** Rotary chair assesses the VOR at mid-frequency ranges by spinning the patient and measuring eye nystagmus in darkness and with visual inputs. It often includes tests of visual-vestibular interaction, like measuring the VOR in the light (with a visual surround) and during fixation on a target that moves with the chair [6, 6]. Here, oculomotor integration is explicit: the visual-vestibular fixation test in the rotary chair is essentially a VOR cancellation test – a patient with a central deficit will show an abnormally high VOR gain when they should suppress it with a fixation target [6]. Rotary chair results are often interpreted together with oculomotor tests: for instance, a central pathology might present as normal calorics (or mild symmetric reduction), normal rotary chair VOR gain, but inability to suppress nystagmus with fixation in the rotary chair, aligning with the gaze test abnormalities. On the other hand, a peripheral vestibular loss will show reduced rotary chair VOR responses but normal visual enhancement and suppression, since the central mechanisms are intact [6, 6]. Additionally, rotary chair can measure velocity storage (central integrator of prolonged nystagmus); a central lesion can shorten velocity storage time, affecting rotary chair decay measures – again linking with gaze-holding function which is also a central integrator function.
- **Positional and Dix-Hallpike tests:** These are more directly vestibular (looking for BPPV or positional nystagmus). Oculomotor findings help ensure that any nystagmus seen is interpreted correctly. For example, in Dix-Hallpike maneuvers for BPPV, the presence of purely torsional or vertical nystagmus might actually indicate central positional nystagmus if it doesn't fit canalithiasis patterns – one would cross-check with the gaze exam (central gaze nystagmus, ocular coordination, etc.) to see if the patient possibly has a central positional nystagmus (from a lesion like a cerebellar tumor) rather than typical BPPV [1]. If oculomotor tests showed other central signs, an atypical positional nystagmus should be viewed with suspicion. Conversely, benign positional nystagmus in BPPV occurs in an otherwise normal oculomotor exam (no pursuit or saccade abnormalities). Thus, the absence of other ocular motor findings can increase confidence that a positional nystagmus is truly benign BPPV.

Overall, oculomotor test results provide context for other vestibular test outcomes. A useful approach is: if all ocular motor tests are normal, yet vestibular-provoked tests (caloric, vHIT, etc.) are abnormal, the lesion is likely confined to the peripheral vestibular system. If ocular motor tests are abnormal, especially in conjunction with modest or equivocal peripheral test findings, a central disorder should be strongly considered [1, 1]. Integration examples:

- In **vestibular migraine**, calorics and vHIT are often normal (no permanent peripheral loss), but oculomotor testing might show subtle central signs (e.g. slight pursuit impairments or positional nystagmus without ear signs), reflecting central vestibular network dysfunction.
- In **stroke** (e.g. lateral medullary infarction), there may be a mix: abnormal ocular motor findings (skew deviation, broken pursuit, gaze nystagmus) alongside some vestibular test

asymmetries (perhaps mild caloric weakness). The pattern of central signs with any peripheral-type nystagmus would prompt neuroimaging.

- In **bilateral vestibulopathy**, oculomotor tests are typically normal (unless the patient is also older or on medication), but rotational chair and head impulse will both be abnormal. Normal ocular motor function in this setting confirms that the issue is bilateral peripheral loss (and not a central integration problem).

Integration also extends to rehabilitation considerations: if a patient has both peripheral vestibular loss and central ocular motor deficits (for example, an elderly patient with vestibular loss and coexisting cerebellar small vessel disease), the vestibular rehabilitation therapy might need to be adjusted, as central ocular motor deficits (like impaired VOR cancellation or poor smooth pursuit) could make recovery slower or require special exercises. The test battery results guide these decisions by delineating all domains of dysfunction.

Emerging Developments in Oculomotor Testing

Oculomotor testing continues to evolve with technological advancements and research, further solidifying its role in vestibular diagnostics and beyond:

- **Digital Eye-Tracking and "Oculomotor Biomarkers":** The adoption of high-speed infrared eye-tracking has opened possibilities for automated, quantitative analysis of ocular motor performance. Recent research is focused on developing digital biomarkers from oculomotor tests using computerized platforms. For example, proprietary systems (e.g. RightEye or similar eye-tracking tools) can administer a battery of saccade and pursuit tests and automatically score metrics like velocities, reaction times, and errors. In a 2025 study, a six-test computerized oculomotor battery (including horizontal/vertical saccades and pursuits) was validated against clinician-administered exams, demonstrating high sensitivity (70–93%) and specificity (85–90%) in detecting abnormal eye movements [9]. The diagnostic accuracy of such automated tests was reported as "good to excellent," suggesting these tools can accurately flag central ocular motor deficits [9]. The ability to rapidly quantify subtle abnormalities (for instance, a mild saccadic delay or small asymmetry in pursuit gain) can aid in early detection of central disorders. Moreover, eye-tracking systems facilitate longitudinal tracking – clinicians can objectively measure if a patient's ocular motor function is improving or worsening over time (e.g. tracking disease progression in degenerative cerebellar disease or recovery after concussion). As this technology matures, we may see the integration of normative databases and machine learning algorithms that use oculomotor metrics as biomarkers for specific conditions (for example, differentiating Parkinsonian syndromes by their distinct eye movement signatures [5, 1]).
- **Concussion and VOMS:** In the past decade, there has been growing recognition of vestibular and oculomotor impairments after concussion (mild TBI). Tests that were once confined to vestibular clinics are now being adapted to sports medicine and neurology for concussion screening. **The Vestibular/Ocular Motor Screening (VOMS)** tool is one such development – a brief battery including smooth pursuit, saccades, convergence, VOR, and visual motion sensitivity, designed to provoke symptoms and identify concussion-related dysfunction [10, 10]. Research has shown that the vestibulo-oculomotor examination is among the most sensitive components for predicting prolonged recovery in sports concussions [10, 10]. Consensus guidelines now often recommend incorporating oculomotor function checks (eye movement tests) in the evaluation of concussion [10, 10]. For example, the presence of impaired convergence or saccadic pursuit in a

concussed athlete can corroborate injury to brain networks even when neuroimaging is normal. The crossover of oculomotor testing into concussion management underscores its broadening applicability. Ongoing research is refining these tests for youth athletes and exploring if early vestibular-ocular therapy post-concussion can speed recovery.

- **Refinement of Interpretation Criteria:** As evidenced by studies like Wu et al. (2022), there is an effort to improve how we interpret oculomotor tests for central lesions. Wu and colleagues applied principal component analysis to a large sample undergoing oculomotor tests and MRI, seeking an objective composite "oculomotor index" to predict central vestibular disorders [7, 7]. They found that a combination of metrics – **including vertical saccade performance, horizontal pursuit gain, and presence of gaze-evoked nystagmus – best predicted central lesions on MRI** [7, 7]. Such research can lead to scoring systems or algorithms that give an overall probability of central pathology based on the oculomotor battery, increasing diagnostic confidence. Additionally, age and comorbidities were noted to affect interpretations, prompting recommendations for age-adjusted norms and caution in older patients [7, 7]. This kind of data-driven approach will likely inform future guidelines, making oculomotor test interpretation more quantitative.
- **Expanded Neuro-Ophthalmologic Insights:** Oculomotor testing in the vestibular context is converging with neuro-ophthalmology. With detailed eye tracking, clinicians are now observing subtle signs like microsaccades, ocular drift, or small disconjugacies that might serve as early indicators of central diseases (e.g. mild cognitive impairment or early Alzheimer's may manifest subtle pursuit or saccadic changes [9, 9]). There is also interest in eye movement testing for neurodegenerative disease differentiation – for instance, using saccadic velocity and accuracy to help distinguish Parkinson's disease from atypical parkinsonian syndromes, or using pursuit metrics to quantify traumatic brain injury effects [1, 10]. While these applications are still being researched, they represent a frontier where vestibular oculomotor tests provide more general neurologic information.
- **Improved Portable and Home Diagnostics:** Traditionally, oculomotor tests require an in-clinic setup. Emerging devices, however, such as mobile headsets or even webcam-based eye trackers, are being tested for remote or bedside use. These could allow screening of patients in emergency departments (for stroke vs vestibular neuritis) or even home monitoring of chronic vestibular conditions. For example, some research prototypes use virtual reality goggles to test pursuit and VOR in any setting, possibly enabling telehealth vestibular assessments.
- **Integration with Other Modalities:** Future vestibular assessments may integrate oculomotor testing with imaging or electrophysiology. For example, functional MRI studies during pursuit or saccades are identifying precise brain regions engaged in these tasks, potentially correlating specific dysfunction patterns with lesion locations. Additionally, novel tests like smooth pursuit neck torsion test (which compares pursuit with head straight vs head turned to implicate cervical vs central causes of dizziness) are being explored in specialized contexts [10].

In conclusion, oculomotor function testing remains a cornerstone of vestibular assessment and continues to advance. Its value in diagnosing vestibular disorders is well established – by delineating central vs peripheral dysfunction – and ongoing innovations promise even greater precision. For the vestibular specialist, mastery of oculomotor tests is essential: it enables accurate localization of pathology, informs safer triage of acute vertigo (identifying strokes that mimic ear disorders), and guides appropriate therapy. As technology enhances our ability to measure eye movements, oculomotor testing will likely expand in use, providing critical biomarkers not only for vestibular disorders but for a spectrum of neurological conditions. This

synergy of tradition and technology ensures that the oculomotor examination will remain an invaluable tool in the vestibular physician's armamentarium for years to come.

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