Optokinetic Nystagmus (OKN):
A Comprehensive Clinical Review

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How to Use This Review

This document is the companion clinical literature review to the optokinetic nystagmus and visual motion processing 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.*

Table of Contents

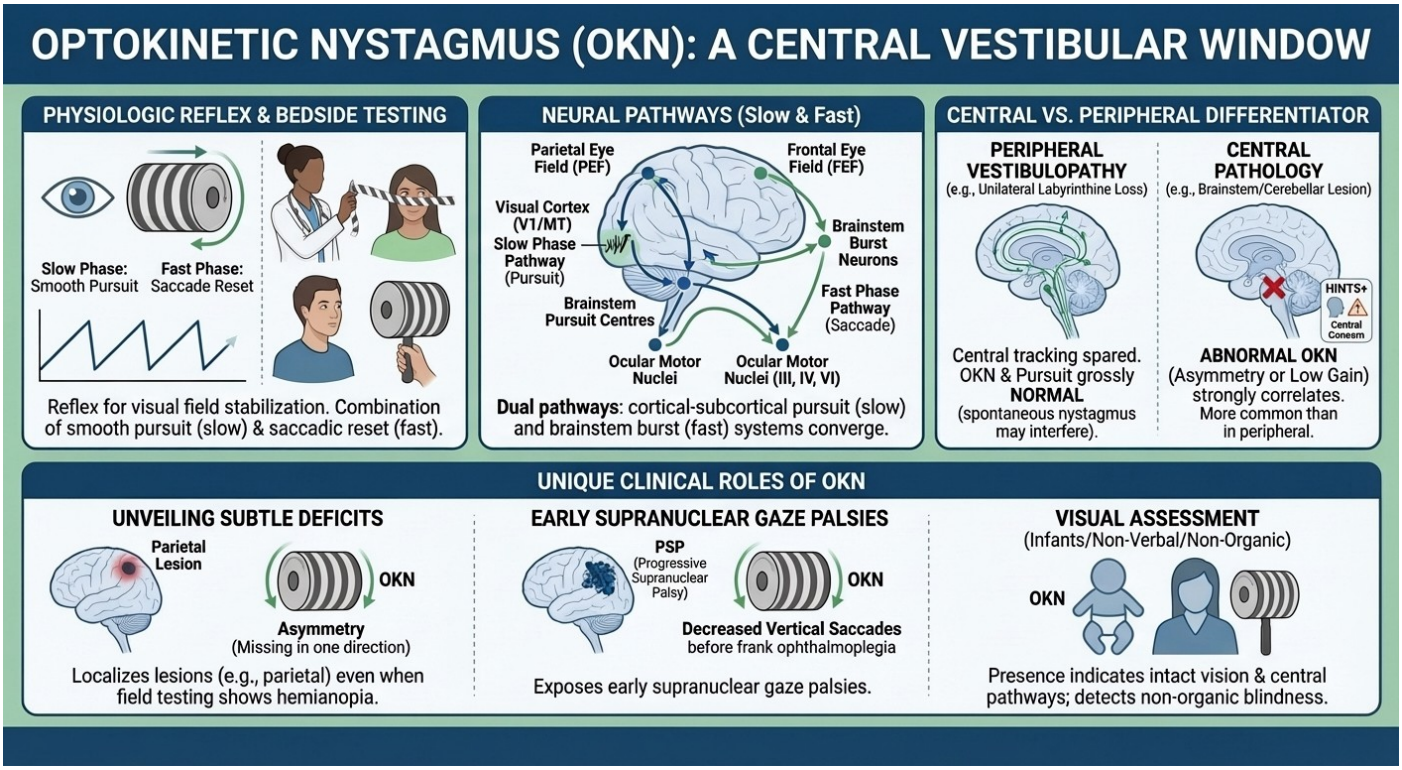
Clinical Neuro-Otology of Optokinetic Nystagmus

An Exhaustive Review

1. Introduction: Optokinetic Nystagmus in Oculomotor Evaluation

For the vestibular physician, optokinetic nystagmus (OKN) is a vital component of the ocular motor examination, serving as a complementary window into central vestibular function alongside smooth pursuit and saccadic testing [1, 2]. OKN is a physiologic reflex eye movement elicited by a full field moving visual scene, classically exemplified by the eyes tracking passing stripes on a rotating drum (slow phase), then rapidly resetting back (fast phase) [3]. This reflex helps stabilize the entire visual field during motion and is fundamentally a combination of smooth pursuit (slow-phase tracking) and saccade-like quick resets (fast-phase saccades). Bedside assessment of OKN – using a striped tape, rotating drum, or even the examiner's moving fingers – is quick, inexpensive, and highly informative [4].

Clinically, the presence or absence of normal OKN responses can distinguish central from peripheral vestibular lesions. Peripheral vestibulopathies (e.g. acute unilateral labyrinthine loss) generally spare central visual tracking mechanisms, so OKN (and pursuit) remain grossly normal aside from interference by spontaneous nystagmus. In contrast, abnormal OKN (especially asymmetry or low gain) strongly correlates with central nervous system pathology [1]. Indeed, one study found that combined abnormalities of saccades, pursuit, and OKN were significantly more common in central vertigo than peripheral vertigo [1]. An inability to generate symmetric, robust optokinetic nystagmus implies a dysfunction of central pathways [2]. OKN testing thus complements the head impulse and nystagmus examinations (as in HINTS+) by providing a direct look at cortical-subcortical visual motion processing: a patient with acute vertigo who shows normal head impulse (suggesting central) but also has clearly impaired OKN slow phases would raise immediate concern for a central lesion.



OKN also serves unique roles in clinical neurology beyond vestibular syndrome triage. Because OKN engages both the slow pursuit system and the fast saccadic reset system, it can unveil subtle deficits not apparent in testing these subsystems alone [4]. For example, an asymmetric OKN (missing in one direction) can localize a lesion to the contralateral parietal cortex even when standard field testing might only show a hemianopia [3]. Likewise, early supranuclear gaze palsies can be exposed by OKN (e.g. decreased vertical saccades on OKN in progressive supranuclear palsy) before frank ophthalmoplegia manifests [4]. In infants or non-verbal patients, the presence of an OKN response indicates intact vision and central pathways, making the OKN drum a time-honoured tool for assessing visual function and detecting non-organic blindness [4]. In summary, OKN is not merely an academic reflex; it is a practical clinical tool that, when interpreted in tandem with pursuit and saccades, enriches our localization of lesions and detection of subtle ocular motor dysfunction.

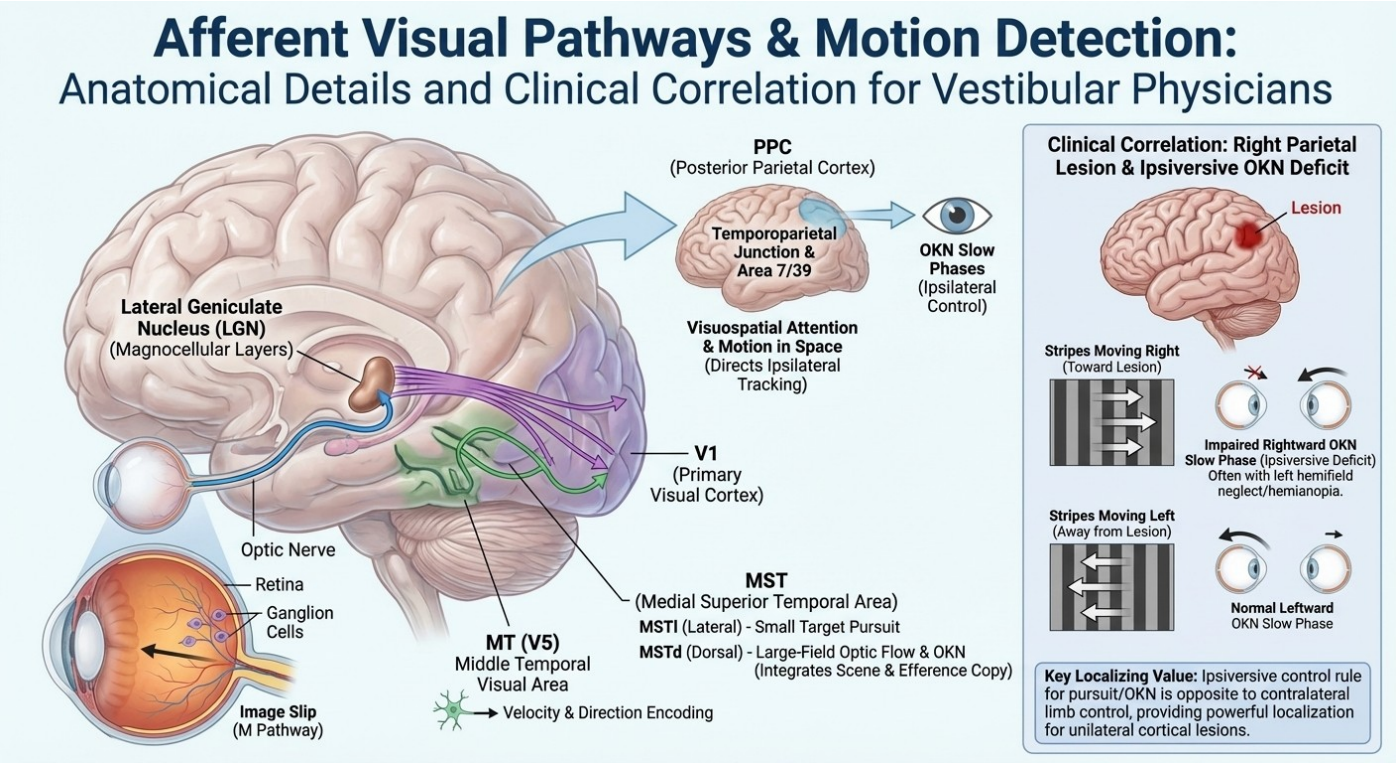
2. Neurophysiology and Anatomical Substrates of OKN

Optokinetic nystagmus arises from a complex neural network that links visual motion detection to eye movement generators. Understanding this circuitry is critical for interpreting pathological patterns. In essence, OKN is the visual counterpart to the vestibulo-ocular reflex (VOR): it stabilizes the retinal image during sustained motion of the visual environment. However, unlike the peripheral vestibular origins of the VOR, the OKN is driven by visual input and engages widespread cortical and subcortical regions.

2.1 Afferent Visual Pathways and Motion Detection

The journey begins in the retina, where specialized retinal ganglion cells sensitive to motion (particularly M pathway cells) detect image slip. These signals travel via the optic nerve to the lateral geniculate nucleus (LGN) (magnocellular layers) and then to the primary visual cortex (V1). Within V1, basic motion signals are extracted and then sent to higher-order visual motion areas. A key cortical region is the middle temporal visual area (MT or V5), which contains neurons tuned to specific motion directions and speeds. MT neurons encode the velocity and direction of retinal image drift, essentially answering “what is moving, and how fast.” From MT, information flows to the adjacent medial superior temporal area (MST), a critical hub for integrating motion signals.

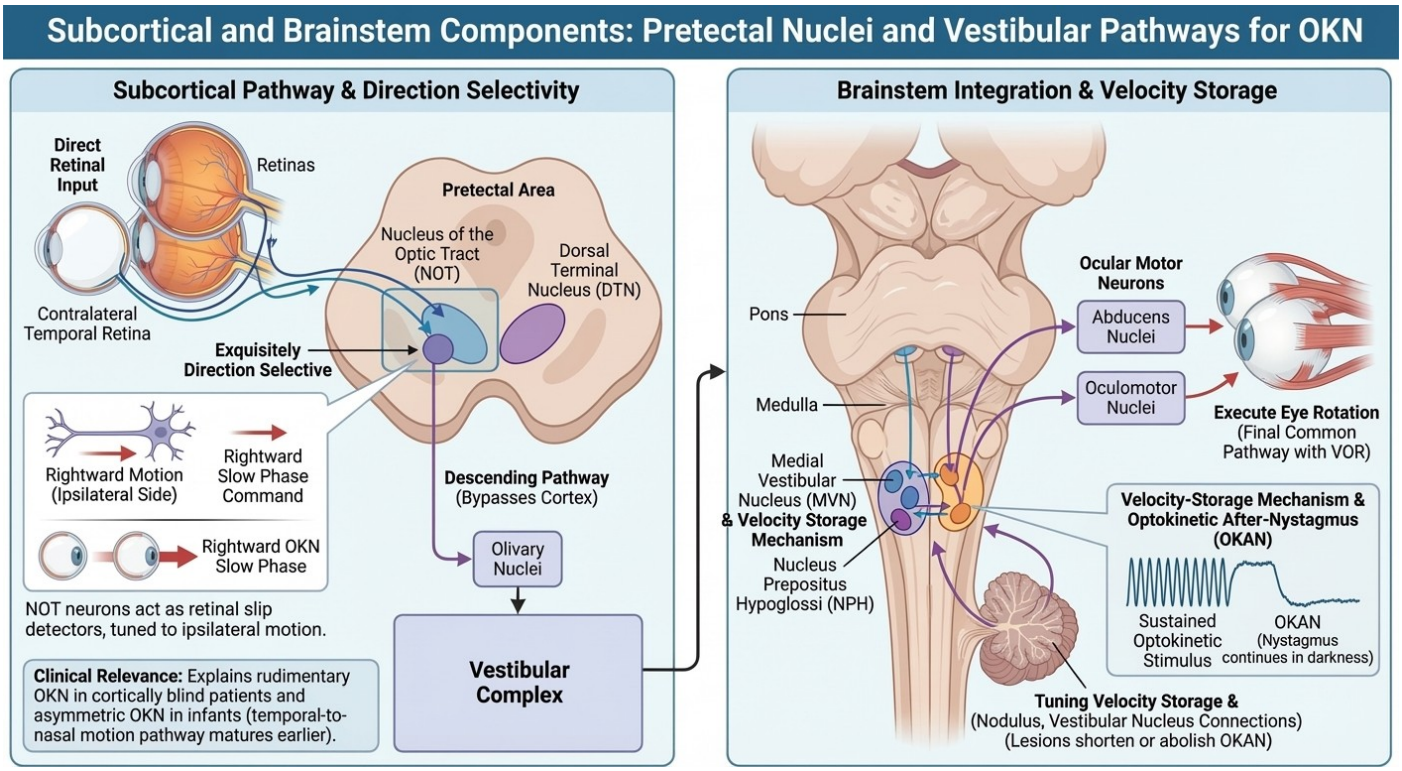
Importantly, MST is functionally subdivided: MSTl (lateral MST) specializes in tracking small, discrete targets (the classical smooth pursuit function), whereas MSTd (dorsal MST) responds preferentially to large-field optic flow, the type of motion stimulus that drives OKN. MSTd neurons integrate visual motion of the entire scene and even incorporate an efference copy of eye movements, distinguishing external world motion from self-induced motion. Lesions confined to MSTd can therefore selectively impair optokinetic responses or the perception of wide-field motion (vection), while sparing small-target pursuit. This explains why a patient with dorsal extrastriate, or parietal damage may have normal foveal pursuit of a single target but an abnormal OKN when shown full-field stripes.



Beyond MT/MST, the posterior parietal cortex (PPC) contributes to OKN by processing visuospatial attention and motion in the context of space. The parietal lobe (especially areas around the temporoparietal junction and area 7/39) helps decide where to attend and plays a role in directing tracking to the ipsilateral side of space. A useful rule of thumb is that each cerebral hemisphere largely controls optokinetic slow phases toward the ipsilateral direction, owing to a double cross in the descending pathways. Thus, a right parietal lesion will impair OKN when the eyes need to follow movement toward the right (i.e. stripes moving toward the side of the lesion), often correlating with a left hemifield neglect or hemianopia in those patients [3]. This ipsiversive control rule for pursuit/OKN is opposite to the contralateral control of limbs, and it provides powerful localizing value in unilateral cortical lesions.

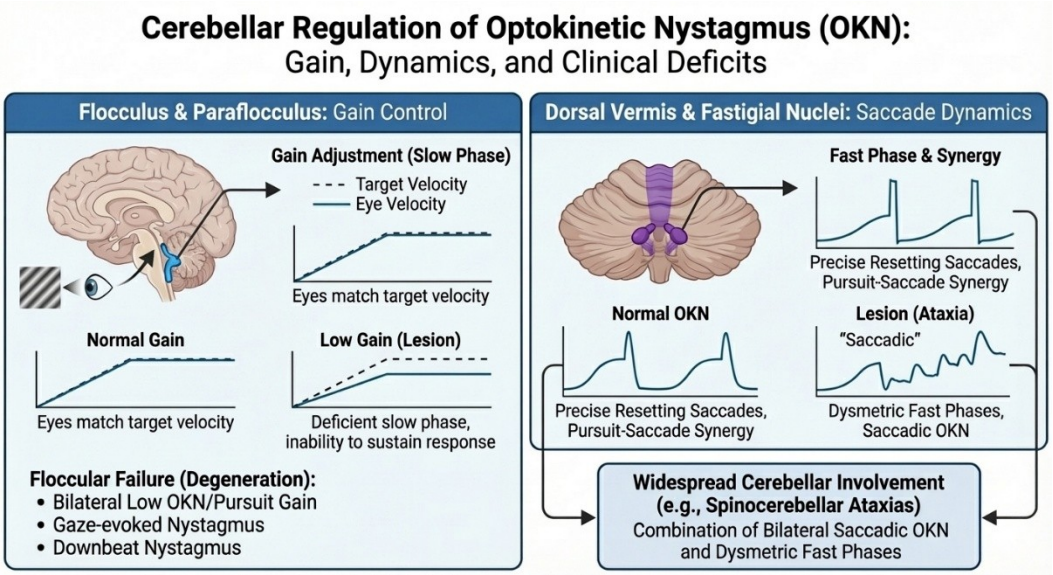
2.2 Subcortical and Brainstem Components (Pretectal Nuclei and Vestibular Pathways)

Visual motion signals for OKN do not rely solely on the cortex. There exists a direct subcortical pathway through the midbrain pretectum that can drive the optokinetic reflex even without cortical involvement (a phylogenetically ancient mechanism, evident in infants and lower vertebrates) [5, 3]. The key players are the **nucleus of the optic tract (NOT) and the dorsal terminal nucleus (DTN) of the accessory optic system,** located in the pretectal area. These nuclei receive direct retinal input (predominantly from the contralateral eye's temporal retina, which views the nasal visual field) and are exquisitely direction selective [5]. For horizontal OKN, each NOT is tuned to motion toward the ipsilateral side – for example, the right NOT fires for rightward motion across the retina (which induces a rightward slow phase) [5]. The NOT neurons act as retinal slip detectors, and their output is relayed into the olivary nuclei and down to the vestibular complex, effectively bypassing the cortex to drive eye movements [5]. This subcortical route explains why even a cortically blind patient may still demonstrate a rudimentary OKN (especially for motion in the temporal-to-nasal direction of the intact eye). It also explains the classic observation that infants have asymmetric OKN: the subcortical pathway (temporal retinal slip -> NOT) matures early, giving robust nasalward slow phases, whereas the cortical pathway needed for the opposite direction develops later [6, 6].



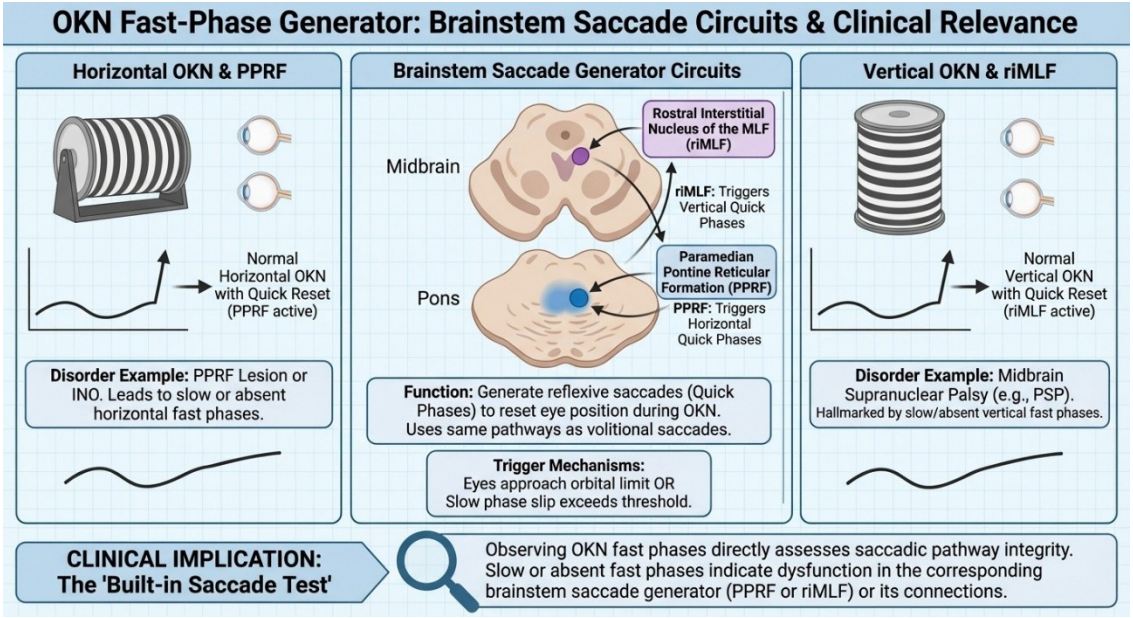
From the pretectum, signals descend to the brainstem vestibular nuclei – principally the medial vestibular nucleus (MVN) and nucleus prepositus hyoglossi (NPH) – which act as neural integrators and relay slow-phase velocity commands to the ocular motor neurons [7, 5]. Essentially, the OKN uses the same "final common pathway" as the vestibulo-ocular reflex: the abducens and oculomotor nuclei execute the eye rotation. Notably, the velocity-storage mechanism in the vestibular nuclei (which prolongs the VOR response to low-frequency rotation) is also engaged by OKN. This is evidenced by the phenomenon of optokinetic after-nystagmus (OKAN): after a sustained optokinetic stimulus is stopped, the nystagmus continues in darkness for several seconds due to stored activity in the vestibular nucleus network. Lesions of the vestibulocerebellum (nodulus and vestibular nucleus connections) can shorten or abolish OKAN, reflecting their role in tuning velocity storage.

The cerebellum indeed plays a central role in OKN regulation. The flocculus and paraflocculus (visual vestibulocerebellum) adjust the gain of the slow phase, ensuring the eyes match the target velocity [8]. Cerebellar lesions often lead to low OKN gain and inability to sustain the response [8, 8]. For example, degeneration affecting the flocculus can result in a deficient OKN (and pursuit) gain bilaterally, often with gaze-evoked and downbeat nystagmus



(floccular failure). The dorsal vermis and fastigial nuclei are more involved in the dynamics of resetting saccades and the synergy between pursuit and saccades. Thus, widespread cerebellar involvement (as in spinocerebellar ataxias) produces a combination of bilateral "saccadic" pursuit/OKN and dysmetric fast phases [8].

Finally, the fast-phase generator for OKN resides in the same brainstem circuits as volitional saccades. The paramedian pontine reticular formation (PPRF) in the pons (for horizontal saccades) and the rostral interstitial nucleus of the MLF (riMLF) in the midbrain (for vertical saccades) trigger the quick reset movements. These are typically triggered when the eyes approach the limit of the orbit or when the slow phase slip exceeds a certain threshold. Quick phases during OKN are essentially reflexive saccades that bring the eye back to central gaze or to a new target of interest [3]. Because they use the saccadic pathways, any disorder slowing saccades (e.g. a PPRF lesion or internuclear ophthalmoplegia) will also slow the OKN fast phase. Clinically, observing the fast phases during OKN is like a built-in saccade test: for instance, slow or absent fast phases on vertical OKN is a hallmark of a midbrain supranuclear palsy (as seen in PSP) [4].

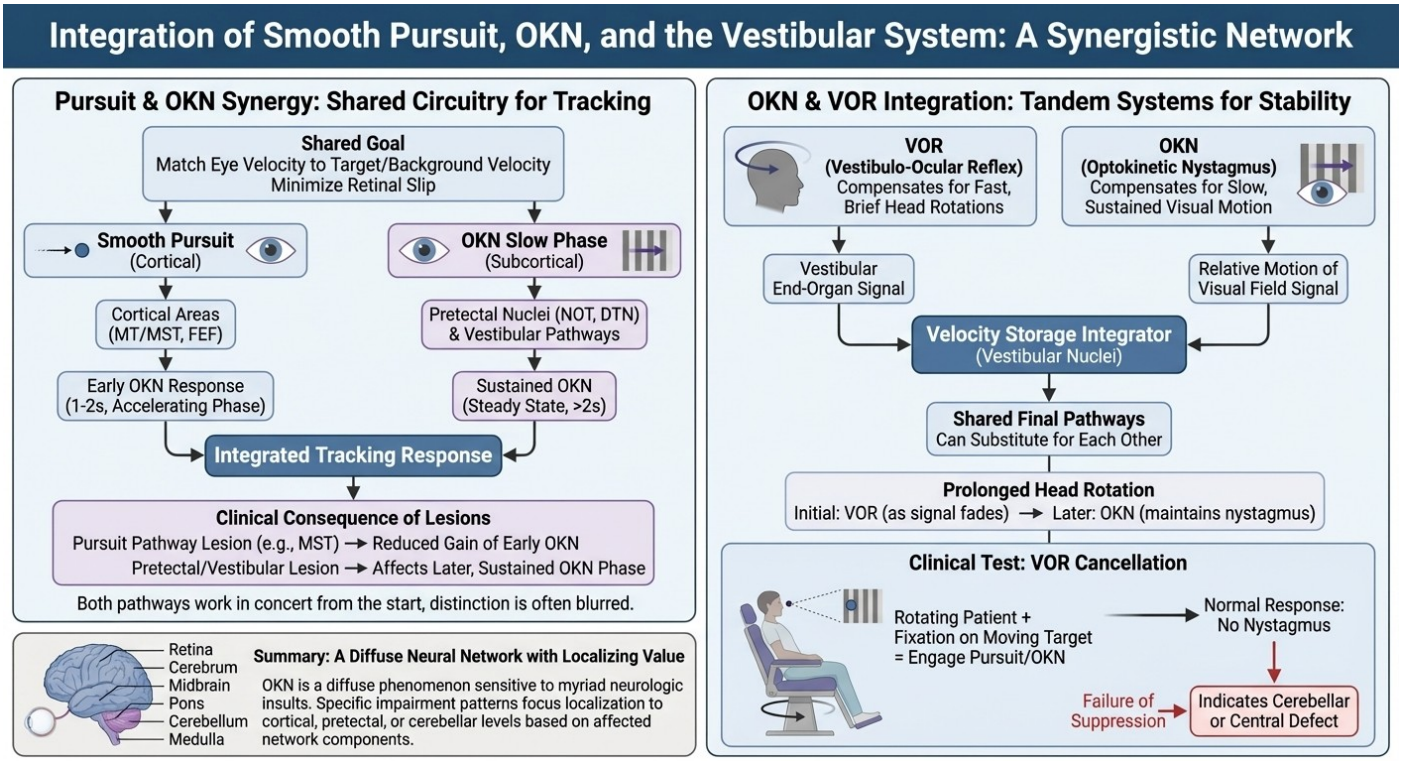


2.3 Integration with Smooth Pursuit and the Vestibular System

It is helpful to conceptualize OKN and smooth pursuit as two synergistic eye tracking systems that share circuitry. Both pursuit and OKN slow phases are driven by the goal of matching eye velocity to target (or background) velocity to minimize retinal slip [7]. Indeed, the initial 1–2 seconds of OKN (sometimes called **"optokinetic response")** rely heavily on the pursuit system – this is the accelerating, closed-loop phase where the eyes try to catch up to the moving field. During this phase, cortical areas MT/MST and frontal eye fields (FEF) contribute as if the patient were "voluntarily" pursuing a moving target [7, 7]. After a few seconds, the subcortical OKN pathway takes over to sustain the response, often termed the **"steady state" OKN** which can continue indefinitely as long as the field moves. This is why lesions in the pursuit pathways (e.g. MST) can reduce the gain of the early OKN response, while lesions in the pretectal or vestibular pathways affect the later, sustained phase. In practice, however, the distinction is blurred and both pathways work in concert from the start.

Integration with the vestibular ocular reflex (VOR) is equally important. The OKN and VOR normally work as tandem systems: the VOR compensates for fast, brief head rotations, whereas OKN compensates for slow, sustained movements of the visual surround. They share final

pathways and can substitute for each other to some extent. For example, when a head rotation is prolonged, the initial eye movement is VOR, but as the vestibular end-organ signal fades, OKN (driven by the relative motion of the visual field) kicks in to maintain nystagmus. Both feed into the velocity storage integrator of the vestibular nuclei. This interplay is evident in tests like VOR cancellation: a patient rotating in a chair who fixates a moving target (thus engaging pursuit/OKN to cancel the VOR) should have no nystagmus; failure of this suppression indicates a cerebellar or central defect.



In summary, the anatomical substrates of OKN span from the retina all the way to the cerebrum, midbrain, pons, cerebellum, and medulla. OKN is truly a diffuse neural network phenomenon, which is why it is so sensitive to myriad neurologic insults – yet specific patterns of OKN impairment can focus our localization to cortical (e.g. parietal), pretectal, or cerebellar levels based on which components of this network are affected.

3. OKN Pathway and Functional Mechanisms

Having reviewed the "wiring," we turn to the functional mechanisms of OKN – essentially how the system behaves in normal conditions. Optokinetic nystagmus is characterized by its dual-phase nature, unique modes of operation (open-loop vs closed-loop), and differences from the smooth pursuit of a single target.

3.1 Slow Phase and Fast Phase Dynamics By definition, nystagmus consists of a slow drift of the eyes in one direction and a fast corrective movement in the opposite direction. In optokinetic nystagmus, the slow phase is in the direction of the visual stimulus motion (the eyes follow the moving scene), and the fast phase is opposite to the stimulus direction (the eyes jump back to reacquire new visual detail) [9]. For example, if stripes move leftward across the patient's field,

the eyes smoothly track to the left, then rapidly flick back to the right. The slow phases are generated by the visual tracking (pursuit/OKN) system, while the fast phases are generated by the saccadic system. Clinically, we name the nystagmus for the fast phase direction – so in the above example, a leftward moving drum produces a "right-beating" nystagmus (fast phase to the right). It is often more useful, however, to pay attention to the slow phase quality: this reflects the patient's ability to smoothly follow visual motion (essentially a measure of pursuit function). At the bedside, one can observe whether the slow phase is smooth or "jerky" and whether its velocity matches the stimulus. An inability to generate a normal slow-phase eye movement in one or both directions is a hallmark of central pathology [2]. The fast phases, on the other hand, should be brisk, symmetric saccades; abnormally slow or asymmetric fast phases implicate saccadic pathway involvement (for instance, a brainstem lesion affecting the PPRF or MLF). Thus, OKN testing conveniently packages a pursuit test (slow phase) and a saccade test (fast phase) into one manoeuvre [4].

The brainstem circuitry automatically alternates these phases to produce continuous nystagmus as long as the visual stimulus moves. Each slow phase is a tracking movement under feedback control, and each fast phase recenters the eye. Notably, fast phases are not volitional – they are reflexive resets triggered by brainstem gaze centres once the slow phase cannot be sustained further. Patients cannot consciously stop the fast phases of OKN (short of suppressing the entire reflex by looking away or closing eyes). This is useful clinically: even a malingerer who resists following a target will exhibit fast phases if a full-field stimulus is moving, since the reflex will eventually kick in. That is why the presence of OKN (especially when tested monocularly) can expose functional blindness [4].

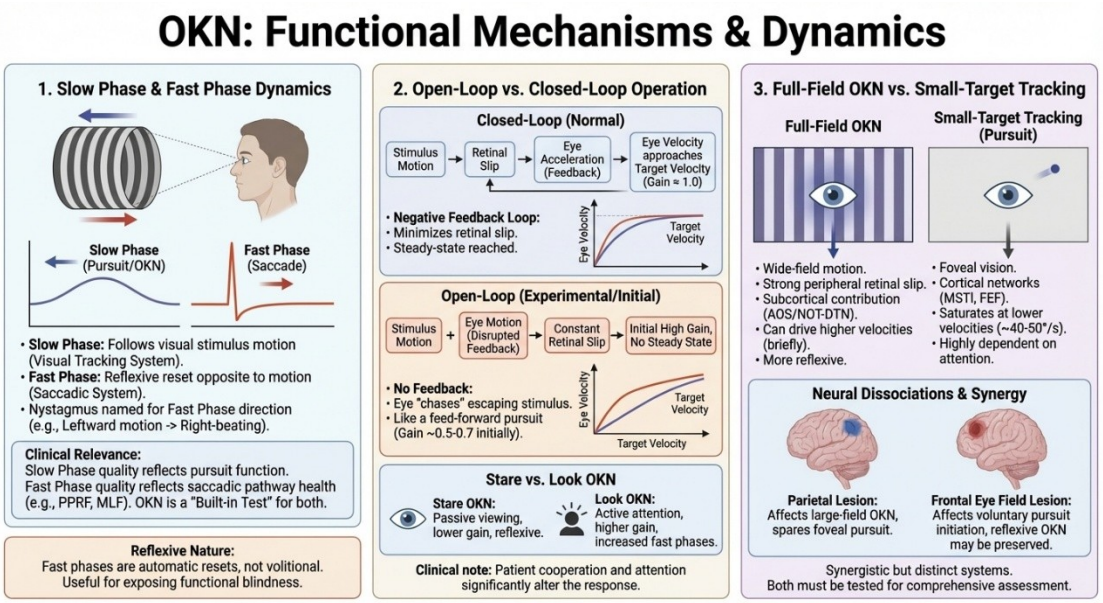
3.2 Open-Loop vs. Closed-Loop Operation The optokinetic system can operate in two modes, often referred to in research as open-loop and closed-loop OKN. In everyday terms, **closed-loop OKN** is the normal condition: the eyes' movement feeds back to reduce retinal slip, approaching a steady-state where eye velocity equals target velocity (ideally, gain = 1.0). In closed-loop operation, any difference between eye speed and stimulus speed is continually corrected by feedback – it is a negative feedback loop striving to minimize error [7]. For instance, when a striped drum begins rotating, the eyes initially lag; retinal slip is large, which drives the system to accelerate eye movement until slip is minimal and the slow-phase eye velocity nearly matches drum velocity.

In contrast, **open-loop OKN** refers to conditions where this normal feedback is disrupted or absent. Experimentally, this can be achieved by adding the measured eye velocity to the stimulus velocity, so that the pattern keeps moving at a fixed rate relative to the world regardless of the eye's motion [10]. In open-loop mode, the eye is essentially chasing a constantly escaping stimulus – true steady-state is never reached, and the response is more like a feed-forward pursuit. The initial 100–200 milliseconds of any OKN (when the visual system has not yet had time to process feedback) is effectively open-loop. During this brief interval, OKN behaves like an instinctive ocular following response with a stereotyped gain (approximately 0.5–0.7 in humans for a sudden full-field motion). After that, as visual feedback comes in, the system switches to closed-loop control and gain rises toward 0.8–1.0 if the stimulus allows [6]. The distinction matters clinically in certain scenarios: for example, in myasthenia gravis, researchers have shown that measuring open-loop OKN gain (which isolates the immediate eye movement drive) is more sensitive to neuromuscular weakness than closed-loop gain [10, 10].

Another concept is the difference between **"stare" vs "look" OKN.** This relates to how much attention and voluntary effort the patient contributes. Stare OKN is when the subject is told to

simply stare straight ahead and not actively follow the stripes; this yields a lower-gain, more reflexive nystagmus. Look OKN is when the subject is instructed to actively watch and follow individual passing stripes; this typically increases slow-phase gain and frequency of fast phases [6]. In one classic experiment, subjects had significantly lower eye velocities during “stare” instructions than during “look” instructions with the same moving pattern [6]. Clinically, this means that patient cooperation and instruction can alter the OKN response. A drowsy or inattentive patient may exhibit what looks like deficient OKN (especially if they are essentially doing stare nystagmus), whereas coaching them to “watch the stripes carefully” can augment the response. We leverage this by encouraging the patient if initial results are suboptimal: improvement with encouragement suggests that the OKN pathways are intact and the issue was attention, not anatomy.

3.3 Full-Field OKN vs. Small-Target Tracking It is important to distinguish optokinetic nystagmus from smooth pursuit of a small target, even though both involve slow eye movements. Full-field OKN is maximally stimulated when the majority of the visual field is in motion (e.g. a striped drum or a moving panorama). In this scenario, peripheral retinal slip contributes strongly to the response. The OKN reflex taps into the subcortical accessory optic system (NOT-DTN) which is most sensitive to wide-field motion across the retina [5]. In contrast, small-target tracking (classic pursuit) engages primarily foveal vision and cortical networks (MSTl, FEF, etc.) optimized for high-acuity tracking of a single object. As a result, there are differences in velocity range and in vulnerability to lesions. Pursuit of a small object typically saturates at target speeds of ~40–50°/s; beyond that, the eye cannot keep up and will fall behind, requiring catch-up saccades. Full-field OKN, however, can sometimes drive slow phases at higher velocities for short periods because the large moving background provides strong motion signals even as the eye lags. Nonetheless, even OKN has practical limits (human OKN gain falls off at very high stimulus speeds, and fast phases occur more frequently instead of increasing slow-phase speed indefinitely).



Another practical difference: in pursuit testing, the target motion is usually predictable (e.g. sinusoidal or a smoothly moving dot), and the patient’s attention is explicitly on that target. In OKN testing with a drum, the motion is repetitive, but the pattern covers the whole field, and the patient may not concentrate on any single element unless instructed (hence the “look vs stare” difference). Neurologically, patients can have dissociations between these systems. For instance, a focal lesion in the parietal lobe might abolish OKN to one side (because it knocks out large-field motion processing to that side) yet leave foveal pursuit relatively intact [3]. Conversely, a patient with a frontal eye field lesion might have trouble initiating voluntary pursuit but could

still exhibit some OKN when a full-field stimulus drives the eyes reflexively via the subcortical pathway. In summary, small-target smooth pursuit and full-field optokinetic nystagmus are two faces of the gaze stabilization coin. They share many elements (and indeed seamlessly work together in daily life, as when tracking a moving object against a background). But the presence of the fast-phase resetting in OKN and the heavy contribution of subcortical pathways in full-field motion distinguish it from pure pursuit. Clinicians should remember that a normal pursuit does not guarantee normal OKN, and vice versa – hence the value of testing both.

4. Clinical Assessment of OKN

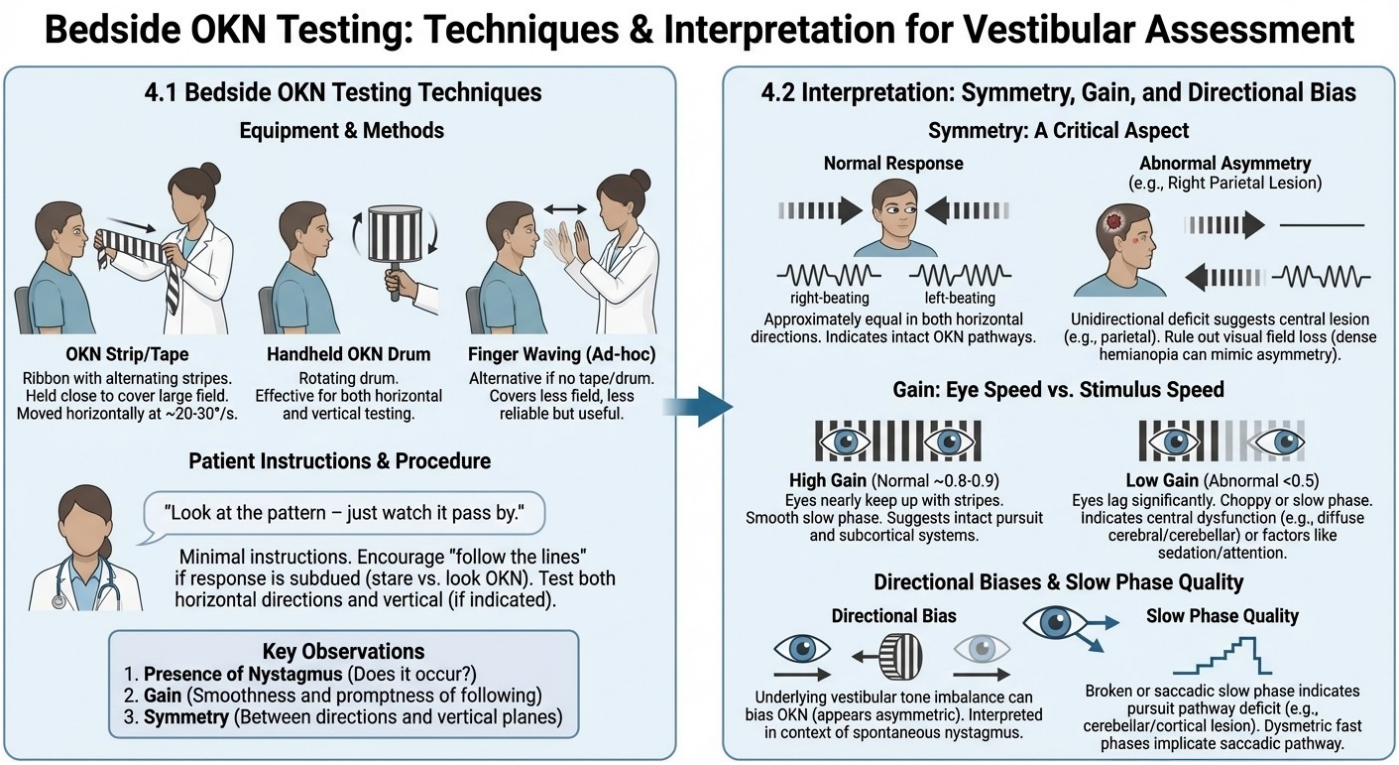
Evaluating optokinetic nystagmus in a clinical setting can be done qualitatively at the bedside and quantitatively in the laboratory. A thorough assessment includes bedside OKN testing for a gross overview and video-nystagmography (VNG) or electronystagmography (ENG) for detailed metrics. In all cases, proper technique is key to avoid misinterpretation.

4.1 Bedside OKN Testing Techniques Bedside OKN testing is straightforward and requires minimal equipment. A common tool is an OKN strip or tape – a ribbon with alternating black and white stripes. The physician holds the strip in front of the patient (who is seated) and moves it horizontally, first in one direction then the other, at a comfortable speed (typically ~20–30°/s). Alternatively, a handheld OKN drum with vertical stripes can be rotated in front of the patient's eyes. In a pinch, even the examiner's fingers waved in front of the face (with the patient instructed to "look at my moving fingers") can induce an OKN response [4]. The key is to cover a large portion of the visual field: small movements or a tiny target will not generate true OKN [2]. One should position the stimulus close enough (or use a large enough drum) such that both central and peripheral vision are stimulated by the moving stripes.

Patient instructions are minimal. Often the best instruction is simply: "Look at the pattern – just watch it pass by." This allows the reflex to occur naturally [2]. If the patient instead tries to fixate on one stripe, they will follow it, then lose it and catch the next – which is essentially what we want (smooth follow, fast refixation). It is important not to instruct the patient to look opposite the movement or to consciously jerk their eyes; such coaching can confuse them. In some cases, if the response is subdued, gently encouraging the patient to "try to follow the lines" can convert a stare nystagmus into a look nystagmus, enhancing the slow phase (as discussed earlier). One should test horizontal OKN in both directions (drum rotation to the patient's right and left), and if indicated, vertical OKN (moving stripes up and down). Vertical OKN is tested by using vertically moving stimuli (or rotating a striped drum in the vertical plane); note that normal vertical OKN can be a bit less robust than horizontal.

During bedside testing, the examiner observes for three main features: (1) the presence of a well-formed nystagmus (i.e. does OKN occur or not?), (2) the gain of the slow phase (estimated by how smoothly and promptly the eyes follow the stripes), and (3) symmetry between directions. A normal response consists of repetitive nystagmus with clearly defined slow and fast phases, and it should appear roughly symmetric when the stimulus direction is reversed [2, 2]. For example, rotating the tape to the right should produce a left-beating nystagmus of similar speed/intensity as the right-beating nystagmus produced by rotating it left. The examiner can qualitatively judge if one direction's slow phase seems "choppy" or much slower. It's also useful to note if the patient reports any sensations: normal individuals often experience a sense of motion (vection) when OKN is induced, whereas patients with certain central lesions might not perceive the movement normally.

4.2 Interpretation: Symmetry, Gain, and Directional Bias Symmetry is a critical aspect. In a neurologically intact person, OKN should be approximately equal in both directions (horizontal) and, to a lesser extent, in both vertical directions. A marked asymmetry – where nystagmus is strong in one direction but weak or absent in the other – is highly suggestive of a central lesion [2]. Classically, a unidirectional OKN deficit (only generating nystagmus toward one side) localizes to the contralateral dorsal cortical pathway (e.g. a right parietal lesion causes inability to generate left-beating OKN when stripes are moved to the right) [3]. However, one must be cautious: asymmetric visual input can also mimic an OKN asymmetry. For instance, a patient with a dense left hemifield loss from an occipital stroke might seem to have poor OKN when stimuli move into that blind hemifield – yet in such a case the defect is visual sensory loss, not an actual OKN pathway failure. Notably, occipital lesions that cause homonymous hemianopia typically do not cause OKN asymmetry beyond what the field cut explains; in contrast parietal lesions often abolish OKN despite intact basic vision, a key clinical difference [3].



Gain refers to the ratio of eye speed to stimulus speed. At the bedside, we estimate gain by how far the eyes can track the stripe before needing a reset, and whether the slow phase nearly keeps up with the tape's movement. If the eyes lag significantly (requiring frequent catch-up saccades), the gain is low. Normal OKN gain is high – often around 0.8 to 0.9 for moderate stimulus speeds (meaning 80–90% of the full field velocity) [2]. If you move the stripes at a comfortable pace, the patient's eyes should almost be able to lock onto them with only slow drift. In abnormal cases, you may see the eyes barely move in the intended direction, interspersed with only small or rare fast phases (indicating the slow phase drive is weak). A very low gain (<0.5) for one or both directions at the bedside nearly always indicates central dysfunction (e.g. diffuse cerebral or cerebellar involvement). By contrast, bilateral mild reductions in gain could be from sedation, age, or poor attention.

Directional biases are also observed. Some patients with vestibular disorders have a pre-existing nystagmus or tone imbalance that can bias OKN. For example, an acute unilateral vestibular

lesion causes spontaneous nystagmus; if you test OKN in the opposite direction of that nystagmus, the two will add together, making the OKN appear stronger, whereas OKN in the other direction may be partly cancelled by the spontaneous nystagmus. This can create an apparent asymmetry that is not due to the OKN pathway per se, but rather an artifact of the underlying vestibular nystagmus. The clinician should thus note if any spontaneous nystagmus is present in primary gaze before testing OKN, and interpret results in that context [2]. Generally, if an OKN asymmetry is seen without concurrent spontaneous nystagmus, a central lesion is likely [2]. If spontaneous nystagmus is present, the asymmetry might just reflect that nystagmus (and one should attempt fixation suppression or wait for it to settle).

Finally, pay attention to the qualitative appearance of the slow phase. A “broken” or saccadic slow phase (eyes making small jumps instead of smooth pursuit) indicates a deficit in the pursuit pathway portion of OKN [1]. This could signal a lesion in the cerebellum or cortex affecting smooth pursuit generation. Similarly, if the fast phases are clearly dysmetric (overshooting or undershooting central position), that implicates saccadic pathway dysfunction (perhaps a brainstem gaze centre or cerebellar fastigial issue). These nuances can sometimes be seen at bedside: for instance, a patient with a MS lesion in the MLF might show a conspicuously slow adducting eye during the quick phase, leading to an uneven refixation movement – effectively an internuclear ophthalmoplegia revealed by OKN [4].

4.3 Pitfalls and Artifacts in Bedside OKN Testing Several potential pitfalls can lead to misinterpretation of OKN findings if not recognized:

- **Incomplete Field Stimulation:** If the stimulus does not cover the patient’s full visual field, the OKN response may be attenuated or absent. A common mistake is using a small card with stripes and moving it only in front of the eyes – the patient may just make a smooth pursuit movement instead of true OKN. Full-field motion is needed; hence a big enough drum or a wide sweep of a striped cloth is important [2].
- **Poor Visual Acuity or Visual Field:** The patient must be able to see the moving pattern. Significant uncorrected vision loss or visual field cuts can reduce OKN. For example, if a patient has severe bilateral visual loss (even if not total blindness), their OKN may appear suboptimal. Always consider a basic visual acuity and field check if OKN is unexpectedly poor. If a patient has one blind eye, monocular OKN testing of that eye will of course be absent; binocular testing might still evoke OKN primarily from the good eye’s input. As mentioned, an isolated occipital lobe lesion (causing hemianopia) will allow OKN when stimuli move toward the intact field but show a drop-out when moving toward the blind field – this is an important clue that a visual sensory deficit, not an OKN pathway lesion, is at play [4].
- **Lack of Patient Attention/Cooperation:** OKN requires the patient’s brain to engage with the moving scene. An inattentive, distracted, or somnolent patient may not mount a full OKN response (“stare” nystagmus may be minimal). This is especially relevant in elderly patients or those with altered mental status. If the OKN appears absent, always ensure the patient is watching the stimulus. Sometimes verbally encouraging them (or increasing the contrast/speed of the stimulus slightly) will bring out a response if the pathways are intact. On the flip side, excessive fixation can suppress OKN; a very anxious patient might try to fight the movement by locking their gaze, which can reduce the slow phase. In extreme cases, instructing the patient to count the stripes (thereby focusing their attention on them) can paradoxically reduce the reflex because they try to stabilize their gaze on a stripe. Thus, the examiner’s manner of instruction should strike a balance – enough engagement to get OKN, but not so much that the patient attempts to override it.

- **Pre-existing Nystagmus or Medication Effects:** As noted, spontaneous nystagmus (from a vestibular lesion) can interfere with OKN. Always account for baseline nystagmus direction and intensity. Additionally, certain medications (sedatives, anti-seizure meds, etc.) can dampen OKN. A patient on a benzodiazepine might have bilaterally reduced OKN gain simply due to drug effect on the cerebellum. If the history suggests this, interpret minor abnormalities with caution or consider re-testing off the medication if possible.
- **Hemispatial Neglect:** In patients with cortical strokes, particularly of the right parietal lobe, a phenomenon of neglect or inattention to one side may cause them to ignore moving stimuli in the contralesional hemifield. Such a patient might barely respond to stripes moving toward their neglected side (appearing as an absent OKN), yet if you draw their attention ("Look at the stripes to your left!") they may suddenly show some response. Distinguishing true OKN pathway failure from neglect can be challenging – one trick is to test each eye separately (monocular OKN) because neglect is usually less pronounced with monocular stimulation, while a structural OKN pathway lesion will still show deficit monocularly.

In summary, bedside OKN testing is a powerful, quick screen for central ocular motor function – but it must be done correctly and interpreted in context. When an abnormality is noted, confirm that it is reproducible, consistent with other exam findings, and not explained by an "operator error" or non-neurological factor.

5. Laboratory Evaluation of OKN

For a more quantitative assessment, vestibular function laboratories perform OKN testing as part of videonystagmography (VNG) or electronystagmography. This involves standardized stimuli and objective recording of eye movements, yielding metrics like gain and symmetry that can be compared to normative values.

5.1 Stimulus Delivery: OKN Drum vs. Video Projection In the lab, optokinetic stimulation can be delivered either by physical devices or digital displays:

- The classic method uses an OKN drum (or a motorized projector) that fills the patient's visual field with moving vertical stripes. Modern setups often use a video projector or large wrap-around screen displaying moving stripe patterns or dots. The patient is seated in a stationary chair within the immersive visual scene.
- A video-based OKN test (as part of computerized VNG) will project moving patterns (e.g. black-and-white bars or a moving checkerboard) on a screen surrounding the patient. Because a full-field stimulus is crucial, labs use large screens or even LED goggles that simulate a moving pattern. During testing, stimuli are typically presented at various constant velocities (for example, 20°/s, 40°/s, perhaps 60°/s) in both directions [2]. Some protocols also test vertical OKN. The patient is instructed simply to watch the moving pattern (the computer may even instruct "look at the moving stripes"). Eye movement is recorded via infrared video-oculography, and slow-phase velocity is calculated by the software.

5.2 Key Parameters: Gain, Velocity Profiles, and Symmetry The primary quantitative measure is OKN gain. In lab reports, this is usually defined as the ratio of the eye's slow-phase velocity to the stimulus velocity (often expressed as a percentage). For example, if the stripes move at 40°/s and the eyes' slow phase averages 30°/s, the gain is 0.75 (75%). A gain of 1.0 (100%) would mean the eyes perfectly keep up with the stripes with no slip. Normal ranges

depend on stimulus speed (gain tends to be a bit lower at higher speeds) and age, but generally a horizontal OKN gain above ~0.8 is considered normal in younger adults. Labs often use a cutoff such as gain < 0.6 (60%) as abnormally low [2].

Symmetry is reported as the percentage difference between right-beating and left-beating nystagmus gain. For instance, if gain is 0.9 rightward and 0.5 leftward, that is a large asymmetry (~57%). Many labs consider an asymmetry above 25% to be significant [2]. The test software typically plots gain values for each direction and marks abnormal points (often with a coloured symbol) if they fall outside thresholds [2]. Symmetry may also be summarized as a ratio or difference in some readouts.

Additionally, the peak slow-phase velocity (SPV) reached is recorded. In a well-functioning system, if you increase stimulus speed from 20 to 40°/s, the slow-phase eye velocity should also increase proportionally (though it may not reach the full 40°/s if some slip is present). An inability to increase SPV with faster stimulus may indicate a saturated response (central problem). Some reports list the SPV achieved at each stimulus velocity, which gives a sense of the response curve.

The waveform of nystagmus can also be examined: is the slow phase linear and smooth, or are there irregularities? Are the fast phases uniform in amplitude? For example, “square-wave” type intrusions during what should be a smooth slow phase would be abnormal and suggest cerebellar or attention-related issues.

Laboratory tests also often assess vertical OKN separately. Norms for vertical may be lower. A significantly reduced vertical OKN (particularly downward) with preserved horizontal can be an early sign of selective midbrain or cerebellar degeneration (since vertical pursuit/OKN can be affected in disorders like PSP disproportionately).

5.3 Interpreting Abnormal OKN Results When an OKN test yields abnormal gain or asymmetry, interpretation leans heavily on central pathology:

- **Directional asymmetry on the report (with one normal gain and one depressed gain)** is practically a fingerprint of a unilateral cortical or pretectal lesion. For example, a patient with a right parietal stroke might show normal OKN gain when stripes move leftward (left-beating nystagmus) but markedly low gain when stripes move rightward (right-beating nystagmus). In the absence of other confounders, that pattern is highly localizing to a right hemisphere lesion [3]. As the EyeWiki concisely states, lesions of the parietal or parieto-occipital cortex produce an OKN asymmetry (loss of OKN toward the side of the lesion), whereas purely occipital lesions (visual loss without parietal involvement) do not produce OKN asymmetry [3].
- **Bilateral reduction in OKN gain** (with relatively preserved symmetry) can indicate diffuse or midline pathology. This is seen in cerebellar degeneration and sedative drug effects. For instance, in degenerative ataxias, researchers find that OKN gain is reduced in all directions, reflecting the cerebellum’s role in calibrating the response [8, 8]. If the lab report shows gains like 0.5 and 0.6 (both below normal) but symmetric, one should consider systemic factors: medications, age (if very elderly, a modest bilateral reduction might be age-related), or a bilateral cerebral issue (less common, e.g. advanced neurodegenerative disease).
- **Abnormally high OKN gain** is not usually a concern except in infants (where it may indicate a different calibration). However, one scenario of apparent increased gain is if the

patient makes inappropriate back-up saccades during the slow phase (so-called “inverse nystagmus” in congenital nystagmus). In that case, the analysis algorithms might be thrown off. Generally, an OKN gain over 1.0 in adults is rare because eyes rarely outpace the stimulus.

- **OKN suppression test:** Some advanced vestibular tests check a person’s ability to suppress optokinetic nystagmus by fixation. Essentially, the patient is given a stationary target to stare at while a full-field pattern moves, and a normal individual will markedly attenuate the nystagmus. Failure to suppress (continued nystagmus despite a fixation target) is analogous to VOR cancellation failure and usually points to cerebellar (floccular) dysfunction. Not all labs perform this specific test, but it can be inferred if, for example, during rotation tests the presence of a visual target did not reduce nystagmus as expected.

When drawing conclusions, the lab findings must be correlated with the clinical picture. For example, an OKN asymmetry on VNG in a patient with known stroke on MRI is confirmatory; the same asymmetry in an undiagnosed patient with dizziness would prompt imaging of the contralateral brain. If OKN is normal in the lab but pursuit was clearly saccadic at bedside, one might question attentional factors or an intermittent issue. In essence, lab results add confidence and detail to bedside observations.

It’s also worth noting that **vertical OKN** testing can help differentiate disorders like PSP (where vertical OKN, especially downward, is severely impaired early) from other causes of parkinsonism [4]. A specialized lab battery might include measuring upward vs downward OKN gain.

Below is an example of recorded OKN responses from a VNG report, illustrating a normal symmetric response versus an abnormal asymmetric response:

Example of a normal horizontal OKN test result (20°/s and 40°/s stimulus velocities to left and right). Slow-phase eye velocity is nearly equal in both directions, yielding gains in the 80–95% range bilaterally. Such symmetry and high gain are indicative of an intact optokinetic system [2, 2].

Example of an abnormal OKN result. Here, rightward stimuli (producing left-beating nystagmus) evoke much lower gain responses than leftward stimuli. At 40°/s, the gain to the right is around 50%, with multiple flat segments in the slow phase (indicating the eyes are lagging), whereas to the left the gain was closer to normal. This marked asymmetry (>25%) and low gain in one direction is suggestive of a central lesion (e.g. a lesion in the hemisphere responsible for tracking toward the weak side) [2]. In a clinical context, such a pattern would warrant imaging of the contralateral brain or further neuro-ophthalmologic evaluation [2].

In practice, many vestibular labs incorporate automated interpretation, but a skilled clinician will always review the raw tracing to ensure artifacts (blinks, calibration errors) are not misclassified as pathology. The combination of bedside and lab evaluation of OKN provides a robust assessment, sensitive to both gross and subtle abnormalities of the ocular motor system.

6. Pathological Patterns and Clinical Relevance

Distinctive patterns of OKN abnormality can yield insights into the site of lesion or nature of a disorder. Here we consider some common pathological OKN findings and their implications.

6.1 Unidirectional or Asymmetric OKN – Cortical Lesion Indicator As alluded to above, a unidirectional loss of OKN (absent or greatly diminished response when the visual field moves in one direction) is a classic sign of a **contralateral cortical lesion.** The most frequently involved region is the **parietal lobe** (particularly the dorsal extrastriate cortex and parietal eye fields). For instance, a large right parietal infarct may result in almost no OKN when stripes move to the right (the eyes cannot perform the slow phase to the right), while OKN to the left remains intact [3]. This occurs because the right parietal region is crucial for generating rightward slow phases (ipsiversive control). In many cases, such patients also have a left homonymous hemianopia and perhaps hemispatial neglect; OKN asymmetry provides additional evidence that the lesion is more than just visual (involving the motion/attention network) [3]. Notably, if the hemianopia were purely occipital (no parietal involvement), OKN would be preserved despite the field cut, aside from the patient not seeing the stripes in the blind field – a phenomenon sometimes tested by neurologists: an occipital lobe stroke patient can still generate OKN in the blind field via the intact subcortical pathway [4].

Unilateral lesions of the frontal eye field (FEF) can also cause asymmetric pursuit/OKN, though parietal lesions are more notorious for it. FEF lesions tend to recover more, whereas parietal lesions often leave a persistent deficit in ipsilateral tracking.

In practice, seeing an asymmetrical OKN in a patient – especially if they have other signs like asymmetric pursuit or gaze preference – should prompt consideration of a structural lesion in the contralateral hemisphere. This is very useful in evaluating patients with complaints like dizziness or imbalance where an MRI might be normal: if you find a unilateral OKN deficit, it may indicate a subtle cortical dysfunction (e.g. from a prior stroke or demyelination) that warrants further workup.

6.2 Central vs. Peripheral Patterns of OKN Impairment Peripheral vestibular disorders (like vestibular neuritis or labyrinthine dysfunction) typically do not directly impair the optokinetic pathways, since those are central. A patient with an acute unilateral vestibulopathy will often have normal OKN when tested, aside from the interference by their spontaneous nystagmus. In fact, preservation of OKN (and smooth pursuit) in the face of acute vertigo strongly suggests a peripheral cause. For example, in acute vestibular syndrome (spontaneous nystagmus, vertigo, imbalance), if one observes that the patient's OKN slow phases are smooth and symmetric (once you account for the spontaneous nystagmus), it supports a diagnosis of vestibular neuritis rather than stroke.

Central disorders, on the other hand, frequently show OKN abnormalities. These may be bilateral or unilateral depending on the pathology. A cerebellar stroke or degeneration often causes bilaterally impaired OKN (low gain both ways, with saccadic intrusions) [8]. A brainstem stroke (e.g. lateral medulla or pons) might cause a bias in one direction or affect the fast phases if the saccadic burst generator is involved. Wallenberg syndrome (lateral medullary infarct) for instance can cause a bias called "lateropulsion" where pursuits and OKN toward the side of the lesion are disrupted. In internuclear ophthalmoplegia (MLF lesion, often from MS), OKN can reveal a slowed adducting eye during horizontal OKN (the adducting eye doesn't keep up during the slow phase when it needs to adduct, and the fast phase in that direction may show a lag as well) [4].

One specific central sign is **failure of fixation suppression of nystagmus,** sometimes tested by seeing if a patient can suppress OKN or vestibular nystagmus with fixation. Normally, if a patient is viewing a small target, they can suppress reflex nystagmus (whether vestibular or

optokinetic). If the cerebellar flocculus is lesioned, this suppression fails – so even with a fixation point, full-field motion will continue to drive nystagmus. Clinically, this is analogous to an abnormal VOR cancellation test, and it is a red flag for central pathology.

Vertical vs Horizontal OKN differences can also be telling: a disproportionate impairment of vertical OKN (especially downward) is characteristic of midbrain disorders such as progressive supranuclear palsy. In PSP, patients eventually lose the ability to generate vertical saccades and pursuit; early on, one can demonstrate that when a vertically striped pattern is moved up or down, there is an absence of the expected vertical nystagmus (e.g. no upward fast phases on downward-moving stimulus) [4]. Peripheral vestibular disease never selectively knocks out vertical OKN while preserving horizontal.

To summarize: peripheral lesions = normal OKN (unless contaminated by spontaneous nystagmus), central lesions = often abnormal OKN. Thus OKN testing is one more piece of the puzzle in differentiating dizzy patients [1, 1]. It has even been noted that abnormal OKN (along with pursuit and saccades) can appear in central vertigo cases where the classic HINTS signs might be equivocal, thereby aiding early diagnosis of stroke [1].

6.3 Optokinetic (VOR) Suppression Failure We briefly mentioned this, but it deserves emphasis: the inability to suppress the optokinetic reflex when appropriate is itself a pathological sign. A normal person can choose to ignore full-field motion if needed (for example, when fixating on a stationary object in a moving environment). The neural basis of this is intact cerebellar and cortical control to override the reflex. If a patient cannot do this – say, even when they try to stare at a central target, their eyes are dragged to follow a moving background – it implies a loss of that override mechanism. This is typically cerebellar (floccular) in origin. Clinically, one might see this during rotational chair testing: the patient is rotated in light with a head-fixed target (so they should cancel out nystagmus), but an impaired patient will still have nystagmus (so-called failure of fixation suppression).

In everyday OKN terms, if you ask such a patient to “stare at my nose” while you move a striped background in front of them, their eyes might still jerk with the stripes. That is abnormal – a healthy brain can prioritize the small fixation target over the moving field, essentially switching off OKN. This finding is a central sign, often pointing to the vestibulocerebellum. It correlates with other cerebellar signs like gaze-evoked nystagmus and inability to cancel VOR.

In summary, pathological patterns of OKN – whether it’s a one-way loss, global reduction, or lack of suppression – all point toward central nervous system dysfunction and help localize the lesion to cortical, pretectal, brainstem, or cerebellar structures depending on the pattern.

7. OKN in Specific Neurological Disorders

Optokinetic nystagmus testing yields characteristic findings in several neurological conditions. Below we outline the OKN manifestations in a variety of disorders, highlighting how OKN aids diagnosis and understanding of each.

7.1 Parietal Lobe Lesions Lesions of the parietal lobe, especially the posterior parietal cortex, classically cause a loss of OKN toward the side of the lesion. As described earlier, a right parietal lesion (e.g. stroke or tumour) results in absent or grossly diminished slow phases when attempting OKN to the right (stimulus moving rightward) [3]. The fast phases toward the left may still appear (due to intact left hemisphere driving them), but the rightward tracking slow phase is

essentially gone. Patients with right parietal damage often have left hemineglect and a left homonymous hemianopia; OKN testing is a sensitive way to demonstrate the parietal component because an occipital (visual) lesion alone would not abolish OKN [3]. In fact, an old clinical pearl is that in a **patient with hemianopia, testing OKN can help determine if the lesion is parietal or occipital: if OKN is asymmetric (absent when stripes move toward blind field) it suggests parietal lobe involvement; if OKN is normal (despite the field cut) it suggests a more posterior (occipital) lesion** [3].

Physiologically, this happens because the dorsal cortical pathway (MT/MSTd → parietal) that drives wide-field motion to that side is disrupted. The contralesional NOT and brainstem may still receive some input from the intact hemisphere, but it's not sufficient to generate a normal reflex. OKN testing is thus part of the neuro-ophthalmologic exam for parietal lobe function. It complements tests for neglect and saccades: for example, a patient might also have difficulty with voluntary saccades toward the contralesional side (Balint syndrome features, etc.), but OKN picks up the reflexive tracking deficit.

In summary, unidirectional OKN loss = think parietal lobe. Recovery of OKN can occur if there is some redundancy, but often this deficit persists, making it a reliable sign of an old parietal stroke on that side as well.

7.2 Infantile (Congenital) Nystagmus and OKN Inversion Patients with infantile nystagmus syndrome (INS) – formerly called congenital motor nystagmus – show a very peculiar OKN phenomenon: **optokinetic inversion.** In a normal subject, as noted, a drum rotated to the right produces a rightward slow phase and left-beating nystagmus. In many patients with congenital nystagmus, the opposite occurs: the fast phase beats in the same direction as drum rotation. In other words, when the drum is rotated rightward, instead of rightward slow phase and left beat, the patient has a leftward slow drift and a right-beating nystagmus (fast phase to the right, which is the direction of drum movement). This is termed OKN inversion or reversal, and it is pathognomonic for congenital nystagmus. Essentially, the congenital nystagmus interferes with the normal optokinetic reflex and flips it.

Eye movement physiologists describe this as the smooth pursuit system being "inverted" in congenital nystagmus patients. Rather than locking onto and following the moving stripes, the eyes of INS patients often drift opposite to the motion and then jerk in the direction of motion (perhaps related to their inherent nystagmus null position strategy). This can be demonstrated in infants to support a diagnosis of congenital nystagmus versus acquired nystagmus. It's one of the "two signature signs" of INS, the other being a characteristic waveform (foveation periods, etc.) [3]. The presence of OKN inversion helps rule out other causes of nystagmus (for example, early-onset sensory nystagmus due to blindness usually does not cause OKN inversion; it's specific to idiopathic infantile nystagmus) [3].

From a practical standpoint, one doesn't often need to test OKN in congenital nystagmus patients to know they have it – their primary gaze oscillation is obvious. But doing so can be educational and can help ensure we're not missing an additional sensory deficit. Additionally, asymmetric OKN in infancy (different from inversion) is seen in infantile strabismus (infantile esotropia), where babies lack a normal nasal-to-temporal OKN response monocularly. That is more of a developmental sign related to monocular visual development and not the same as INS inversion, but it underscores that OKN testing is used in paediatric neurology and ophthalmology for diagnosing congenital disorders.

In summary, congenital nystagmus = consider OKN inversion: if you see the nystagmus beats in the direction of drum rotation, you are dealing with an infantile nystagmus syndrome. This finding is essentially never seen in acquired lesions and thus is a neat diagnostic nugget.

7.3 Parkinsonian Syndromes (PD, Atypical Parkinsonism) In Parkinson's disease (PD), eye movement abnormalities are generally mild compared to disorders like PSP. However, subtle OKN and pursuit changes do occur. Patients with PD often have what's described as **"cogwheel" or saccadic pursuit** – small interruptions in smooth tracking due to bradykinesia of eye movements. On optokinetic testing, this may manifest as slightly reduced gain and more frequent small corrective saccades in the slow phase. Horizontal OKN can be almost normal in early PD, but vertical tracking (especially upward) might be a little impaired due to baseline range limitations. That said, OKN is not a prominent deficit in typical PD. It is usually symmetric and only modestly low in gain. A PD patient might have an OKN gain of 0.7–0.8 (a bit low) with some minor "stutters," which could be attributed to age as much as disease. Thus, OKN testing is not primary in diagnosing PD, but it can exclude more serious pathology: the presence of relatively normal OKN (especially vertical) in a parkinsonian patient leans toward idiopathic PD rather than an atypical syndrome.

Parkinson-plus syndromes present more striking OKN findings:

- In **Progressive Supranuclear Palsy (PSP),** as noted before, there is a **vertical OKN failure.** Early in PSP, vertical saccades slow and vertical OKN fast phases become weak or absent [4]. Horizontal OKN might be relatively spared early on. A classic early sign is that downward OKN (looking at a tape moved downward) fails to generate upward fast phases; instead, the patient might exhibit the convergence-retraction nystagmus of dorsal midbrain syndrome when attempting upgaze, as PSP overlaps with those midbrain findings [4]. In mid to late PSP, no OKN can be generated vertically, and horizontal becomes impaired too. So, an older patient with parkinsonism who shows an inability to follow vertical stripes and absent vertical nystagmus on OKN testing likely has PSP rather than PD [4]. PSP patients also often cannot suppress OKN or VOR (due to midbrain and cerebellar involvement).
- **Multiple System Atrophy (MSA),** particularly the cerebellar subtype (MSA-C), can cause a cerebellar ocular motor syndrome: gaze-evoked nystagmus, impaired pursuit, and thus impaired OKN. OKN gain can be bilaterally reduced, and gaze-holding is poor. MSA patients may also have some positional downbeat nystagmus. None of these are specific to MSA, but in context (parkinsonism with autonomic failure, etc.), seeing a cerebellar type OKN abnormality (low gain, saccadic intrusions) is supportive of the diagnosis. By contrast, PD typically wouldn't have gaze-evoked nystagmus or severe OKN deficit.
- **Corticobasal Degeneration (CBD)** (one of the cortical atrophy syndromes with parkinsonism) can produce asymmetrical eye movement issues, especially if one parietal lobe is affected more (since CBD often causes asymmetric cortical dysfunction). These patients might have an asymmetric OKN similar to a stroke in parietal cortex (since effectively there is cortical degeneration). Additionally, eye apraxia (difficulty initiating gaze shifts) can be present. So in a patient with asymmetric parkinsonism and dementia (suspected CBD) who has a unilateral OKN deficit, it reflects cortical involvement on that side, fitting CBD's pathology.

In short, OKN helps differentiate parkinsonian disorders:

- PD: largely normal or mildly impaired OKN (no dramatic asymmetry; vertical OKN present until very late).
- PSP: profoundly impaired vertical OKN early [4], horizontal may also slow; a big red flag for PSP.
- MSA/Cerebellar ataxic syndromes: bilaterally impaired OKN with gaze-evoked nystagmus (cerebellar signs) [8].
- CBD (cortical atrophy): possible asymmetric OKN due to cortical damage. Dementia with Lewy bodies or other dementias may have some pursuit/OKN deficits, but those are usually overshadowed by PSP and MSA changes when present.

7.4 Progressive Supranuclear Palsy (PSP) Given its importance, PSP merits its own emphasis. PSP is an atypical parkinsonian disorder where the **hallmark is a supranuclear vertical gaze palsy.** The OKN test is practically an office examination for early PSP detection. By asking the patient to follow a vertically moving striped pattern, one can unmask absent vertical saccades: instead of the normal up-beating or down-beating nystagmus, the patient's eyes may show only a drift or a blink with no true vertical fast phase [4]. In a study, vertical OKN was used to diagnose PSP before ophthalmoplegia: patients had essentially no downward saccades on OKN, even though they could still move their eyes somewhat in exams, highlighting the sensitivity of OKN [4].

PSP often shows a specific asymmetry: downward gaze is affected first and more than upward. So **downward OKN** (stimulus moving downward, requiring upward fast phases) **fails earliest** [4]. Upward OKN (requiring downward fast phases) also becomes impaired but sometimes slightly later. Horizontally, PSP patients eventually develop trouble as well – horizontal saccades slow, and horizontal OKN gain drops – but that's later. In contrast, multiple other conditions (including PD) do not cause such a selective vertical OKN loss. Hence, finding vertical OKN impairment in an older person with postural instability strongly suggests PSP.

Another sign in PSP is the **"round-the-houses"** phenomenon: because vertical movements are limited, patients may try to use a sequence of horizontal movements to look up or down (i.e. curving trajectory). While not directly tested by OKN, it's part of the overall eye movement exam.

In summary, PSP = vertical OKN loss (especially downward) early on [4]. This is a practically useful bedside nugget to differentiate PSP from benign Parkinson's or other parkinsonian syndromes.

7.5 Alzheimer's Disease and Cortical Atrophy Alzheimer's disease (AD) primarily affects cognitive and memory circuits, but it can involve visual processing networks (especially in posterior cortical atrophy variants). Patients with AD can have eye movement abnormalities, though these are subtle compared to movement disorders. Studies have shown that AD patients may exhibit saccadic intrusions, impaired smooth pursuit, and "irregular" OKN on detailed testing [10, 10]. "Irregular OKN" means the nystagmus during full-field motion is not smooth and periodic as it should be instead it might be broken up by inappropriate saccades or have variable slow-phase velocity. This reflects frontal and parietal cortical involvement in AD – attentional deficits can lead to lapses in tracking, and visuospatial deficits can alter how motion is perceived.

One case report on an AD patient noted difficulty initiating reading and abnormal OKN as part of the picture [10]. AD patients often complain of reading problems out of proportion to visual acuity, partly due to impaired eye movement control (they skip lines or lose their place, which in the lab is seen as poor stepwise reading eye movements [10]). On OKN testing, an AD patient might

have a symmetric response but with low gain (due to reduced cortical support) and small spontaneous saccades (square-wave jerks) intruding [10].

Another scenario is posterior cortical atrophy (PCA), an atypical AD variant affecting the visual cortex and parietal regions. Those patients often have Balint's syndrome (simultanagnosia, optic ataxia) and can have extremely disrupted OKN and pursuit – essentially their brain cannot integrate the moving field properly. They might not experience vection and their OKN could be absent or bizarre despite no eye muscle paralysis.

While OKN is not a diagnostic test for AD, it can reflect the degree of cortical visual processing impairment. If an older patient shows significantly reduced OKN gain without a clear focal lesion, and especially if accompanied by cognitive complaints, it might hint at diffuse cortical involvement. However, one has to be cautious not to overcall age-related changes as AD, since normal aging also reduces OKN performance (discussed in the next section).

In summary, AD and cortical degenerations: minor OKN and pursuit deficits, often bilateral and symmetric, related to cortical processing slowing. No specific "signature" like PSP's vertical palsy, but rather a general degradation of performance (low gain, small intrusions) consistent with diffuse cortical atrophy [10].

7.6 Multiple Sclerosis (MS) Multiple Sclerosis can affect practically any part of the ocular motor network depending on lesion locations. Two findings are particularly associated with MS:

- **Internuclear Ophthalmoplegia (INO):** MS demyelination frequently involves the medial longitudinal fasciculus (MLF), causing INO. In INO, the adducting eye is slow or unable to move past midline on attempted horizontal gaze. A subtle or partial INO might not show obvious limitation on a brief exam, but OKN can unmask it. If you have the patient follow an OKN stimulus that requires the affected eye to adduct during either the slow or fast phase, you may note a lag. Specifically, on horizontal OKN, the adducting eye lags behind on the fast phase, resulting in a disconjugate reset (one eye completes the fast phase faster than the other) [4]. One can sometimes see a tell-tale **"lagging abducting nystagmus"** – the abducting eye beats nystagmus (because of the disparity) while the adducting eye is slow. In the AAN abstract series, a case was highlighted where horizontal OKN demonstrated a slow adducting saccade in INO that was otherwise not obvious [4]. So OKN is a useful provocative test for INO, which in an appropriate patient is likely due to MS (though brainstem stroke can also cause INO).
- **Upbeat or Downbeat Nystagmus:** MS plaques in the brainstem or cerebellum can cause acquired nystagmus types, including upbeat, downbeat, or torsional nystagmus. These would appear as spontaneous nystagmus but can be exacerbated by optokinetic stimuli or certain gaze positions. For example, a plaque in the medial vestibular nuclei or periaqueductal region might cause impaired downward pursuit and downbeat nystagmus. On OKN, you might see that downward slow phases cannot be maintained (similar to floccular lesions). While not specific to MS, these findings in a young person could be a clue.

Additionally, MS patients with cerebellar involvement will have the cerebellar OKN signature (bilateral low gain, saccadic pursuit) [8]. MS can essentially mimic any central pattern depending on lesion load. A patient with MS could have one-sided OKN asymmetry (if they have a lesion in one cerebral hemisphere), or could have a bilateral problem (if lesions affect the brainstem or both sides).

Thus, in MS:

- If INO: look for dissociated eye movement on OKN (slow adducting eye).
- If cerebellar lesions: look for bilateral OKN impairment, gaze-evoked nystagmus.
- If brainstem lesions: possibly directional nystagmus or vertical abnormalities. OKN is not routinely tested in MS clinics, but an astute examiner might use it to bring out subtle INO or confirm a suspected lesion localization. For example, an MS patient complaining of "blurred vision when tracking things" might have an INO that is mild; doing an OKN test could demonstrate it and direct the MRI to look at MLF.

7.7 Hereditary Ataxias and Cerebellar Degeneration In cerebellar degenerations (such as Spinocerebellar Ataxias, Friedreich ataxia, or multisystem atrophy with cerebellar features), eye movement abnormalities are common and include gaze-evoked nystagmus, dysmetric saccades, and impaired smooth pursuit [8]. OKN, which relies on a functional vestibulocerebellum to maintain calibration, is invariably impaired in these conditions.

A **hallmark is bilateral reduction of OKN gain** – patients cannot generate normal slow phase velocities, often producing saccadic OKN where the eyes series of small jumps instead of a smooth follow. Studies have quantitatively shown that patients with degenerative ataxias (like olivopontocerebellar atrophy, etc.) have significantly lower OKN gains compared to controls [8, 8]. For example, one study noted that across SCA and Friedreich ataxia patients, average OKN gain was ~0.5–0.7 versus ~0.9 in healthy individuals [8]. Moreover, in such patients the impairment is present at both 30°/s and 90°/s stimuli (meaning it's not just a saturation at high speed but a fundamental lowering) [8, 8]. This reflects cerebellar inability to sustain the slow phase drive.

Another tell-tale feature is the presence of downbeat nystagmus in many cerebellar disorders (especially those involving the flocculus, like SCA6, or toxin-induced ataxias from lithium or anticonvulsants). Downbeat nystagmus implies a tonic imbalance with eyes drifting up and correcting down, and it is exacerbated when the patient tries to look laterally or with full-field stimulation sometimes. If you do vertical OKN in a patient with downbeat nystagmus, you'll find they cannot follow downward-moving stripes (since that requires upward slow phase, and their eyes tend to drift up already). Instead, they will have a strong tendency for the eyes to drift upward (the pathological slow phase), with corrective downward saccades that can mingle or interfere with the expected OKN. In fact, what you might see is that on upward OKN stimulus (stripes moving up), they get an exaggerated downbeat (pathological plus reflex), and on downward stimulus, they might paradoxically still show downbeating because the pathology overwhelms the reflex. This can be complicated, but essentially any pre-existing nystagmus from cerebellar disease will distort the OKN test.

Hereditary ataxias often can be differentiated by particular oculomotor profiles:

- SCA3 (Machado-Joseph): gaze-evoked nystagmus, impaired pursuit, possible periodic alternating nystagmus – all will cause irregular OKN.
- SCA6: prominent downbeat nystagmus – vertical OKN severely affected.
- Friedreich Ataxia: often has fixation nystagmus and OKN gain reduction symmetrical (plus peripheral neuropathy can reduce visual stability).
- Ataxia Telangiectasia: can have abnormal OKN and also issues with vestibular reflexes.

One often cited principle: smooth pursuit, OKN gain, and VOR cancellation are all sensitive indicators of cerebellar dysfunction, but not very specific to which part [8]. They usually all go hand-in-hand: a patient with pan-cerebellar degeneration will have saccadic pursuit, low OKN gain, and failure of VOR suppression. These tests won't tell you which ataxia, but they confirm that the cerebellum (particularly flocculus and vermis) is not doing its job.

In summary, cerebellar ataxias = bilateral OKN impairment [8]. This can be one of the earliest signs in mild cases, appearing before the patient has dramatic limb ataxia. For example, a person with an CACNA1A gene mutation (episodic ataxia or early SCA6) might present with only subtle gaze-evoked nystagmus and need eye movement testing to uncover how much the cerebellum is affected.

The above survey illustrates that optokinetic nystagmus, as a composite ocular motor behaviour, is affected in diverse neurologic conditions. The patterns – whether unidirectional loss, vertical vs horizontal differences, or overall slowing – must be interpreted in the clinical context, but they frequently provide decisive clues. A summary is provided in the following table:

Table – Characteristic OKN Findings in Selected Disorders:

Disorder	OKN Finding	Interpretation/Notes
Parietal lobe lesion (unilateral)	Absent OKN toward lesion side	Contralateral hemispheric deficit [3]
Occipital lobe lesion (unilateral)	OKN preserved (aside from blind hemifield)	Subcortical pathway intact [3]
Infantile (Congenital) Nystagmus	OKN inversion (fast phase same direction as drum)	Pathognomonic for infantile nystagmus syndrome (INS) [3]
Parkinson's Disease (PD)	Mildly reduced gain, normal symmetry	Reflects age or slight basal ganglia effect; vertical OKN largely normal until late.
Progressive Supranuclear Palsy (PSP)	Severely impaired vertical OKN (early) [4]; horizontal OKN later impaired	Indicates midbrain vertical gaze palsy; helps distinguish PSP from PD.
Multiple System Atrophy (MSA-C)	Bilaterally low OKN gain; gaze-evoked nystagmus	Cerebellar involvement (floccular dysfunction).
Multiple Sclerosis (MS)	Depends on lesion: INO causes disconjugate OKN (lagging adducting eye) [4]; cerebellar lesion causes low gain	OKN can unmask subtle INO or mimic cerebellar findings.
Cerebellar Degeneration/Ataxia	Bilateral low gain, saccadic intrusions [8]; ±downbeat nystagmus on vertical testing	Highly sensitive to cerebellar (vestibulocerebellum) dysfunction.
Alzheimer's / Cortical Atrophy	Mild symmetric reduction; "irregular" tracking	Due to impaired visual attention and processing [10].

Characteristic OKN Findings in Selected Neurological Disorders: A Clinical Guide for Vestibular Physicians

Disorder	OKN Finding	Interpretation/Notes
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Infantile (Congenital) Nystagmus	**OKN inversion** (fast phase same direction as drum) — Inversion!	Pathognomonic for infantile nystagmus syndrome (INS) [3]
Parkinson's Disease (PD)	Mildly reduced gain, **normal symmetry** — Normal symmetry	Reflects age or slight **basal ganglia effect**; vertical OKN largely normal until late.
Progressive Supranuclear Palsy (PSP)	**Severely impaired vertical OKN** (early) [4]; horizontal OKN later impaired — Severely impaired vertical	Indicates midbrain vertical gaze palsy; helps distinguish PSP from PD.
Multiple System Atrophy (MSA-C)	Bilaterally low OKN gain; gaze-evoked nystagmus	Cerebellar involvement (floccular dysfunction).
Multiple Sclerosis (MS)	Depends on lesion: INO causes disconjugate OKN (lagging adducting eye) [4]; cerebellar lesion causes low gain — Disconjugate/Low Gain	OKN can unmask subtle INO or mimic cerebellar findings.
Cerebellar Degeneration/Ataxia	Bilateral low gain, saccadic intrusions [8]; ±downbeat nystagmus on vertical testing	Highly sensitive to cerebellar (vestibulocerebellum) dysfunction.
Alzheimer's / Cortical Atrophy	Mild symmetric reduction; "irregular" tracking	Due to impaired visual attention and processing [10].

8. Pharmacological and Toxicological Effects on OKN

The influence of drugs and toxins on eye movements is significant, and optokinetic nystagmus is particularly sensitive to **CNS depressants.** Many medications can reduce OKN gain or alter the nystagmus waveform, sometimes mimicking central pathology. Vestibular physicians must recognize drug effects to avoid misinterpretation.

- **Sedatives and Tranquilizers (Benzodiazepines, Barbiturates):** These enhance GABAergic inhibition in the cerebellum and brainstem. Clinically, benzodiazepines like diazepam or lorazepam cause a dose-dependent reduction in smooth pursuit and OKN gain, often producing a clearly saccadic pursuit/OKN. The eyes show frequent small corrective saccades (similar to a cerebellar patient) and overall slow-phase velocity is reduced. In essence, a patient on heavy sedatives can look similar to one with a pancerebellar syndrome on oculomotor testing. This effect is acute and reversible – once the drug is metabolized, pursuit/OKN normalize. Barbiturates would have a similar effect (though less commonly encountered now). This is why, for example, a patient in status epilepticus treated with IV benzodiazepines may transiently have very poor OKN/pursuit.
- **Alcohol (Ethanol):** Acute alcohol intoxication is notorious for causing gaze nystagmus and impaired ocular tracking. Alcohol impairs cerebellar flocculus function, leading to the classic **"positional alcohol nystagmus"** and an inability to cancel VOR. On OKN testing, an intoxicated individual typically has low OKN gain and gaze-evoked nystagmus, often referred to colloquially as "alcohol gaze nystagmus". This is so reliable that the field sobriety tests used by law enforcement include a form of OKN/pursuit assessment (the Horizontal Gaze Nystagmus test) – a person with a high blood alcohol level will show saccadic pursuit and endpoint nystagmus consistently. The mechanism is transient cerebellar dysfunction. As the alcohol wears off, these signs resolve. Chronic alcohol abuse, however, can cause permanent cerebellar damage (mostly to the anterior vermis) and lead to lasting pursuit/OKN deficits.

- **Anticonvulsants (Phenytoin, Carbamazepine, etc.):** Many antiseizure drugs have cerebellar-depressant effects, especially phenytoin. Toxic levels of phenytoin classically produce nystagmus, ataxia, and dysarthria – so on eye exam, one sees gaze-evoked nystagmus and poor pursuit/OKN. Even at therapeutic levels, some patients on anticonvulsants have mildly reduced OKN gain and minor gaze nystagmus. Phenytoin can cause a permanent cerebellar atrophy with long-term use, hence the persistent deficits sometimes seen. Newer anticonvulsants (like gabapentin, etc.) can also cause dizziness and nystagmus as side effects, presumably via similar pathways.
- **Lithium:** Lithium is well known to cause a specific syndrome of cerebellar toxicity, particularly involving the **flocculus.** A signature sign is **downbeat nystagmus in lithium toxicity**. Lithium can accumulate in the cerebellum; chronic use or high levels produce an acquired downbeat nystagmus and ataxia, which fortunately can be reversible if caught early. On OKN testing, a patient on lithium (especially if toxic) might show a vertical pursuit/OKN asymmetry – inability to follow downward targets smoothly – due to floccular impairment (the flocculus facilitates downward eye movement, so its failure leads to upward drifts and downbeat nystagmus). This looks very much like a cerebellar degenerative picture and indeed should be treated by stopping lithium.
- **Antipsychotics (Neuroleptics):** Typical antipsychotics (haloperidol, chlorpromazine, etc.) can cause mild oculomotor slowing. They are dopamine antagonists; while their major effects are on saccadic latency and perhaps some dystonic eye movements (like oculogyric crisis), they also can cause generally reduced pursuit gain and slightly slowed OKN. It's usually not dramatic. However, one confounder with long-term neuroleptic use is tardive dyskinesia, which can include erratic eye movements or difficulty maintaining fixation (oculomotor tardive syndromes). A patient with tardive issues might appear to have "noisy" OKN with lots of intrusions that are actually due to unrelated spontaneous saccades. Differentiating drug effect from true neurodegeneration can be tricky; one clue is that drug effects are typically bilateral and symmetric. If you see a symmetric mild pursuit/OKN deficit in someone on multiple CNS depressants, it's probably medication-related. If you stop the offending agent and retest a few weeks later, you should see improvement.
- **Opioids:** Opiates can cause pinpoint pupils and drowsiness; they are less often discussed in terms of eye movement, but significant opioid intoxication will reduce alertness enough to degrade OKN (similar to any sedative). Interestingly, morphine or high-dose opioids can also produce transient nystagmus or even a downbeat nystagmus by affecting brainstem interneurons. This is not commonly tested, but anecdotally, patients on opioids may have trouble with visual tracking.
- **Toxins:** Various toxins (industrial or environmental) can damage eye movement control. For instance, certain solvent inhalants can cause cerebellar syndrome acutely – an individual acutely intoxicated on **toluene**, for example, might show gaze nystagmus and low OKN gain. Some heavy metals like mercury can cause permanent cerebellar damage, likewise affecting OKN. These are rarer scenarios.

Pharmacological and Toxicological Effects on OKN: A Clinical Guide

Agent Class/Toxin	Specific Agent(s)	OKN Finding/Effect	Interpretation/Mechanism
Sedatives & Tranquilizers	Benzodiazepines (e.g., diazepam), Barbiturates	Dose-dependent reduction in gain, saccadic pursuit/OKN (frequent corrective saccades) — Saccadic, Low Gain	Enhances GABAergic inhibition (cerebellum/brainstem). Mimics cerebellar syndrome. Reversible.
Alcohol (Ethanol)	Acute & Chronic Alcohol Use	**Acute**: Low gain, gaze-evoked nystagmus ("alcohol gaze nystagmus"). **Chronic**: Persistent deficits.	Impairs cerebellar flocculus (acute/transient); anterior vermis damage (chronic). Basis of field sobriety test.
Anticonvulsants	Phenytoin, Carbamazepine, Gabapentin	Gaze-evoked nystagmus, poor pursuit/OKN gain. Phenytoin toxicity: marked nystagmus/ataxia. — Toxicity: Marked	Cerebellar-depressant effects. Phenytoin can cause permanent cerebellar atrophy.
Lithium	Lithium (toxicity or chronic high levels)	Downbeat nystagmus, vertical pursuit/OKN asymmetry (downward tracking impaired)	Floccular toxicity (accumulates in cerebellum). Flocculus failure -> downward drift & downbeat nystagmus.
Antipsychotics (Neuroleptics)	Typical (haloperidol), Atypical	Mildly reduced pursuit gain, slightly slowed OKN. Tardive dyskinesia: "noisy" OKN.	Dopamine antagonists (saccadic latency effects). Long-term use -> tardive oculomotor issues.
Opioids	Morphine, High-dose Opioids	Reduced OKN gain due to drowsiness/alertness. High-dose: transient nystagmus, possible downbeat.	Affect brainstem interneurons; general CNS depression reduces visual tracking.
Toxins	Solvents (toluene), Heavy Metals (mercury)	**Acute**: Gaze nystagmus, low OKN gain. **Chronic/Heavy Metals**: Permanent damage.	Cerebellar syndrome (acute); permanent cerebellar damage (chronic).

General Principle: CNS depressants tend to suppress OKN slow phase, promoting a "cerebellar" profile (bilateral saccadic pursuit, gaze nystagmus). Review medications before diagnosing structural pathology.

In all of the above, the unifying theme is depression of central ocular motor integrators, especially the cerebellum. Therefore, **drug-induced OKN** changes often resemble those seen in cerebellar diseases: **bilateral saccadic pursuit, gaze-evoked nystagmus, reduced OKN gain.** The table in section 7 of the Smooth Pursuit review summarizes many of these effects.

For the clinician, awareness of these effects is crucial. Before ascribing an abnormal OKN to a stroke or degenerative disorder, one should review the patient's medications. It has been wisely stated that when a patient presents with bilateral saccadic pursuit and gaze nystagmus, rule out medications (like anticonvulsants or sedatives) before ordering an MRI for a presumed cerebellar lesion. Often, simply adjusting the drug regimen can normalize the eye movements on follow-up.

In sum, pharmacologic agents – particularly CNS depressants – tend to suppress the OKN slow phase and promote a "cerebellar" eye movement profile. Recognizing this prevents misdiagnosis. Conversely, from a research perspective, some have used OKN gain as a quantitative measure of drug effect (for example, measuring open-loop OKN gain to evaluate benzodiazepine sedation level [10]).

Finally, one can also consider the opposite: drugs that improve eye movements. There is some evidence (mostly experimental) that stimulants or dopaminergic medications might slightly enhance pursuit/OKN in certain disorders (like in PD, giving levodopa can improve smooth pursuit gain marginally, as dopamine pathways are involved in eye movement control). But in clinical practice the main concerns are those drugs that worsen ocular motor performance.

9. Age-Related Changes in OKN

Aging has a significant impact on the oculomotor system, and optokinetic nystagmus is no exception. It is essential to distinguish changes that are "normal for age" from those that are truly pathological, especially when evaluating older patients. Misinterpreting an age-related decline as a sign of central pathology can lead to unnecessary alarm or testing.

Several well-documented age effects include:

- **Reduction in OKN (and pursuit) Gain:** As people age, the gain of smooth pursuit and OKN gradually declines. This decline is linear and can be quite marked in advanced age. For example, a 80-year-old might naturally have an OKN slow-phase gain of ~0.6–0.7, whereas a 20-year-old would be ~0.9. Thus, what might be borderline abnormal in a young adult could be perfectly normal in an octogenarian. This is attributed to a diffuse loss of neurons across the visual and oculomotor pathways: fewer cortical motion-sensitive neurons (MT/MST), some atrophy in the cerebellum, and general slowing of CNS processing.
- **Increased Saccadic Interruptions:** Older individuals often exhibit what's called **"square-wave jerks"** or small saccadic intrusions during attempted steady fixation or pursuit. These can give the appearance of slightly jerky slow phases on OKN as well. In a healthy elderly patient, these intrusions are usually of small amplitude and not consistently in one direction (distinguishing them from pathological nystagmus). When recording eye movements, one might see more variability in slow-phase velocity in an older patient, whereas a young person's tracing would be smoother.
- **Vertical vs Horizontal:** Vertical eye movements tend to degrade more with age than horizontal. So, an 80-year-old might have noticeably poor downward pursuit/OKN gain (maybe only 0.4 or 0.5), but they can still track horizontally at 0.6–0.7. This is important because one could otherwise suspect PSP if not aware of age. The difference is that in normal aging, it's a gradual, symmetric decline without a complete abolition of vertical OKN (unlike PSP where vertical is essentially gone relatively early). Also, horizontal movements in PSP eventually fail too, whereas in aging, horizontal usually remains functional albeit slowed.
- **Latency and Initiation:** Elderly patients may have a slight delay in initiating tracking. When you move the OKN tape, a young person's eyes latch on almost immediately; an older person might have a 100-ms longer delay and possibly need a cue or a larger stimulus to kickstart the reflex. This can sometimes be seen as needing a few seconds of drum rotation before nystagmus builds up in the elderly – which is why one should sustain the stimulus a bit longer when testing older patients, to allow the response to manifest.
- **Central Processing and Attention:** Aging also affects the attention component. An older patient may become momentarily distracted or less able to maintain focus on the task, which can result in an apparent "waxing and waning" of their OKN slow phase. Encouragement or re-focusing them can improve it. This ties into the cognitive side of aging; mild cognitive impairment might show up as inconsistent pursuit/OKN due to lapses in attention.

Age-Related Changes in OKN: A Clinical Guide for Vestibular Physicians

Age Effect	Description/Mechanism	Clinical Presentation/Finding	Interpretation/Differential Diagnosis
Reduction in OKN (and Pursuit) Gain	Linear decline with age. Due to diffuse neuron loss in visual/ oculomotor pathways (cortical, cerebellar) and slowed CNS processing.	Lower slow-phase gain (e.g., ~0.6-0.7 at age 80 vs ~0.9 at age 20). Waveforms appear saccadic with frequent catch-up saccades. (Young / Old)	Normal for age; use age-adjusted norms. Contrast with pathological low gain.
Increased Saccadic Interruptions	Appearance of "square-wave jerks" or small saccadic intrusions during attempted steady fixation or pursuit.	Slightly "jerky" slow phases. Intrusions are small amplitude, not consistently directional.	Distinguish from pathological nystagmus. Considered a "senescent change" if symmetric.
Vertical vs. Horizontal Decline	Vertical eye movements degrade more than horizontal with age.	Noticeably poorer downward pursuit/OKN gain (e.g., 0.4-0.5) compared to horizontal (e.g., 0.6-0.7).	Crucial differential with PSP. Aging is a gradual, symmetric decline, not complete abolition. PSP
Latency and Initiation Delay	Slight delay in initiating tracking. May need a stronger stimulus or cue to kickstart reflex. (Stimulus onset / Delay)	Delayed onset of nystagmus. Response may take a few seconds to build up.	Sustain stimulus longer; use encouragement. Not a sign of profound pathology on its own.
Central Processing and Attention	Affected by attentional lapses or mild cognitive decline. Can result in apparent "waxing and waning".	Inconsistent tracking that improves with re-focusing or encouragement.	Highlights cognitive component. Improvement with encouragement suggests intact circuits.

Key Principles: Symmetry is paramount; significant asymmetry is pathological, not age-related. Distinguish benign age-related decline (symmetric, gradual, improves with encouragement) from pathology like PSP or cerebellar disorders. Use age-adjusted expectations.

From a diagnostic perspective, the key is calibration of expectations. Many labs have age-adjusted norms (for instance, a gain of 0.7 might be flagged as abnormal for 20-year-old but normal for 75-year-old). Clinically, when we see an older patient with "saccadic pursuit/OKN," we interpret it in light of age and other neuro signs. Symmetry remains crucial: even in old age, a significant asymmetry is not normal. So if a 78-year-old has an OKN gain of 0.5 leftward and 0.5 rightward, that could be just age. But if it's 0.8 one way and 0.3 the other, that asymmetry is likely pathological. Similarly, if pursuit/OKN are mildly saccadic but everything is symmetric and there are no other neuro signs, it is probably "senescent change".

It has been suggested that age-related vestibular and ocular motor decline could partly explain why older individuals experience dizziness more commonly – their combination of slower VOR, weaker OKN, and poorer balance leads to increased symptomatology under conditions that a younger person might compensate for. Clinicians should be cautious not to overcall mild saccadic movements in an otherwise intact 85-year-old as a stroke or degenerative disease. Conversely, if an older patient's oculomotor function is perfect (like a 20-year-old), one can be quite confident there's no central issue at that time.

In summary, normal aging leads to a moderate, symmetric decline in OKN performance. This includes lower gain and more frequent catch-up saccades. It is a benign change that must be distinguished from early PSP or cerebellar disorders by careful attention to asymmetries and associated signs. A practical tip is that encouraging an older patient ("keep looking, follow it as best you can!") often improves their tracking a bit – if it does, that suggests their circuits are intact but just needed boosting (attention effect), whereas if it's truly a lesion, no amount of encouragement will normalize an asymmetric or profoundly low response.

10. Advanced Clinical Insights: Attention, Non-Verbal Testing, and Cognitive Factors

Beyond standard examination, optokinetic nystagmus provides intriguing insights into the interplay of attention, prediction, and cognitive load in eye movements. These advanced considerations can enhance clinical use of OKN, especially in challenging patient populations.

10.1 Interaction Between Attention and OKN OKN might be a reflex, but it is one that can be modulated by attention and mental state. We have touched on "stare" vs "look" OKN, which is fundamentally an attentional modulation [6]. When a patient actively engages attention on the moving pattern, the slow-phase gain increases; when they disengage (just stare blankly), gain decreases. This provides a means to gauge a patient's level of cooperation or alertness during the test.

In anxious or inattentive patients, one often sees an inconsistent OKN: some slow phases are normal, others fall behind, and extra saccades pop in. The distinguishing feature from a structural lesion is that this variability can often be reined in with focusing the patient's attention. For example, if a patient's OKN looks saccadic, the examiner might say, "Take a deep breath and really try to follow the stripes with your eyes." If the next run of OKN is smoother, it suggests the prior abnormality was due to inattention or anxiety (not neurological). In contrast, if it's a true neurological deficit, coaching won't remove the saccadic nature. This is analogous to what is done in pursuit testing: improved pursuit with encouragement = likely non-organic cause.

Another aspect is distractibility. Some patients, especially those with anxiety or hyperactivity, cannot sustain their gaze on the pattern, resulting in irregular OKN. They might start following a stripe then suddenly look away or blink excessively. This might mimic a central problem but is actually an attentional issue. The examiner can try to minimize distractions, ensure the patient is comfortable (not in pain or vertiginous during the test), and possibly even do multiple trials to average it out.

There is also research interest in using OKN suppression as a measure of attention [11]. The idea is, if you ask a patient to perform a secondary task (cognitive task) while being exposed to moving visual field, their ability to suppress or modulate OKN might change. For instance, in early dementia or after brain injury, patients may not suppress OKN well when asked to concentrate on something else, indicating attentional network deficits [11]. This is not yet a standard clinical tool, but it shows the principle: attention and OKN are intertwined in the brain. Practically, for clinicians, it serves as a reminder: always interpret OKN findings in light of the patient's alertness and focus at that moment.

10.2 Use of OKN in Non-Verbal or Pediatric Patients One of the most valuable uses of optokinetic nystagmus is in patients who cannot follow commands or verbalize, such as infants, young children, or individuals with severe neurological impairment (including coma). Since OKN is a reflex that does not require a verbal response, it can be used to assess vision and some neurological function in these populations:

- **Infants:** In paediatric ophthalmology and neurology, a presence of OKN response indicates that the infant's visual system and brainstem pathways are functioning at least to a basic degree. For example, to test an infant's visual acuity or detect malingering in toddlers, a doctor might use a Catford OKN drum (striped drum). If the infant's eyes exhibit nystagmus when the drum is spun, it implies the child can see the stripes and that the subcortical OKN pathway is intact [12, 13]. Lack of an OKN might suggest poor vision (due to ocular problem or cortical blindness). Additionally, as mentioned, asymmetric OKN in infancy (naso-temporal asymmetry) can be a clue to conditions like infantile esotropia.

Usually, infants under ~3–6 months have a natural asymmetry (they prefer temporal-to-nasal motion monocularly), but persistence of a strong asymmetry beyond that age can indicate a disorder of binocular vision development.

- Young Children: In toddlers or young children who won't cooperate with a formal visual field test or acuity test, OKN tape can be a game-like way to gauge function. Many children will naturally follow a moving stripe pattern. The examiner can note if the nystagmus is present and relatively symmetric, which would imply both hemifields are seeing. If a child has, say, a homonymous hemianopia from perinatal stroke, you might notice that OKN is absent when moving the pattern toward the affected field (similar to adults, although in children sometimes these tests are harder to interpret due to variable cooperation). OKN testing is also a way to detect functional (psychogenic) blindness in older children – if a child says they can't see but their eyes still have OKN with one eye covered, it means that eye is sending visual information to the brain [4].
- **Comatose or Non-Communicative Adults:** The OKN reflex can be used in intensive care or neuro settings to assess brainstem function in patients who cannot follow commands (though vestibulo-ocular reflex via head impulse or calorics is more common in ICU, because those don't require vision). However, in cases of suspected hysterical coma or feigned unresponsiveness, an old trick is to use an OKN drum: a truly unconscious patient won't generate OKN, whereas a conscious-but-feigning patient often cannot suppress it. There's also the famous "belladonna stupor" vs true coma story – where in psychogenic coma the presence of OKN was historically used as evidence the patient's visual pathways were intact (modern tests like cold calorics and MRI are of course more definitive now). Another scenario: in severe stroke where a patient can't speak or move limbs well, seeing OKN could reassure that the brainstem visual pathways are working (e.g. in locked-in syndrome, OKN can be present because cranial nerves III/VI and the pretectum are intact even though the patient can't move otherwise).
- **Differentiating Structural vs Non-Structural Vision Loss:** As mentioned via the AAN abstract example, if someone claims to be blind in one eye, you can cover the good eye and wave stripes in front of the "blind" eye – if you get any OKN, that eye is functioning [4]. This is a classic test for malingering or functional vision loss. It's not foolproof (a very astute malingerer might deliberately suppress their OKN by not looking at the stripes, but usually if you do it quickly they are caught off guard). Typically, you'd do it by rolling a striped drum in front of each eye separately and watching for any nystagmus. Even a few beats of nystagmus in the allegedly blind eye is proof of at least light perception and some form vision [4].

Thus, OKN is a powerful tool in those who cannot communicate normally. It gives a "yes/no" type answer about whether the visual pathways to midbrain are intact. It is also generally easier to elicit than a pursuit in uncooperative patients because the large moving field naturally draws the eyes (whereas a small target might not even catch their attention).

10.3 Predictive Mechanisms and Cognitive Loading The smooth pursuit system has a known predictive capability – it can anticipate regular target motion – but what about OKN? In traditional terms, OKN is considered a reflex without volition, but there is some evidence of "cognitive" influence even on OKN.

For instance, if a moving pattern has a subtle higher-order regularity, subjects might show a slight enhancement in OKN if they expect certain movements. However, pure optokinetic reflex is largely reactive. What is more relevant is when the brain is performing other tasks (cognitive loading) and how that affects OKN. Studies have indicated that when subjects are given a

secondary task (count backward by sevens, for example) while viewing optokinetic stimuli, the OKN slow phase gain can decrease, presumably because attention is diverted. This effect is more pronounced in older adults, which ties into using OKN suppression as an index of cognitive function [11].

In clinical settings, one might not formally test “predictive OKN,” but these concepts remind us how integrated the ocular motor system is with cognition. For example, consider a patient with schizophrenia – a disorder known to have pursuit deficits linked to working memory dysfunction. While OKN per se is not typically tested in schizophrenia, one can imagine that their ability to engage with a full-field stimulus might be altered. In fact, research in schizophrenia has noted abnormal optokinetic after-nystagmus decay and other subtle OKN differences, thought to reflect cortical processing anomalies. This is more of a research area, but it underscores that OKN is not a purely “dumb” reflex; it is influenced by the cerebral cortex and thus by conditions affecting cognition.

Another cognitive aspect: hysterical nystagmus vs organic. Occasionally patients present with bizarre voluntary nystagmus or eye flutter that they might be doing intentionally or as a functional disorder. OKN has a pretty stereotyped appearance, so a nonsensical nystagmus that does not resemble any physiological pattern might be recognized as functional. But also, one can use optokinetic stimulation to see if a bizarre nystagmus entrains or changes. An organic nystagmus (like a vestibular nystagmus) will typically be suppressed in light and might not follow an OKN stimulus precisely, whereas a functional one could stop or start depending on what the patient believes is expected.

In summary, OKN spans the divide between reflex and volitional eye movements. It is largely reflexive, yet it can be modulated by higher cortical states. Clinically:

- Use cognitive tricks (like dual tasks or encouragement) to differentiate organic vs attentional causes of impaired OKN.
- Employ OKN in those who can’t otherwise communicate, as it requires no conscious effort on their part (and note that if it’s absent, it might be due to severe impairment or simply inattention if the patient isn’t processing the visual input).
- Recognize that an engaged patient yields the best OKN; if a patient is panicking or highly anxious (or conversely extremely somnolent), their OKN will not reflect their true neurological potential.

To conclude this comprehensive review: Optokinetic nystagmus is a window into a broad swath of the nervous system – from retina to cortex to brainstem to cerebellum. Its assessment is invaluable in localizing lesions (central vs peripheral, cortical vs brainstem), in detecting subtle disorders (from infantile nystagmus to PSP), and in evaluating patients who cannot otherwise participate. By understanding the neurophysiology of OKN, mastering the clinical examination techniques, and recognizing pathological patterns, the vestibular physician adds a powerful tool to their diagnostic arsenal. The key is always to integrate OKN findings with the rest of the ocular motor exam and the clinical context, thereby achieving the most accurate interpretation and ultimately, the best care for the patient [1].

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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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