

Optokinetic Nystagmus (OKN) — Clinical Cheat Sheet

OVERVIEW & DEFINITIONS

Definition: Reflexive nystagmus elicited by full-field moving visual stimulus, consisting of slow-phase tracking in the direction of stimulus motion and catch-up fast-phase saccades in the opposite direction.

Purpose: Assess brainstem and cortical integration of visual motion processing. Sensitive to CNS lesions affecting gaze control, visual-vestibular interaction, and velocity storage mechanisms.

Clinical Value: Asymmetric OKN strongly suggests hemispheric lesion. Normal OKN with abnormal VOR = peripheral vestibular disease. OKN deficit + abnormal pursuit = central pathology.

Neural Basis: Dual pathway — subcortical (brainstem vestibular nuclei sustain tracking) and cortical (V5/MT, MST provide voluntary enhancement). Interactions with the velocity storage mechanism in the vestibular nucleus.

NEURAL PATHWAYS & CIRCUITRY

| Pathway | Key Structures | Function | Lesion Effect |
|------------------|----------------------------------|---|--|
| Subcortical OKN | Brainstem vestibular nuclei, NOT | Sustain slow-phase tracking | Reduced gain, narrow velocity range |
| Cortical OKN | V5/MT, MST, DLPFC | Enhance gain, voluntary control | Directional asymmetry, contralateral bias |
| Saccadic System | SC, PPRF, CN VI | Generate catch-up fast-phase | Slow or absent fast-phase saccades |
| Pursuit Network | Cortex → pontis → cerebellum | Smooth slow-phase component | Deficit mirrors pursuit if cerebellar |
| Velocity Storage | Vestibular nuclei, commissure | Prolong OKN after stimulus stops (OKAN) | Asymmetric OKAN indicates vestibular imbalance |

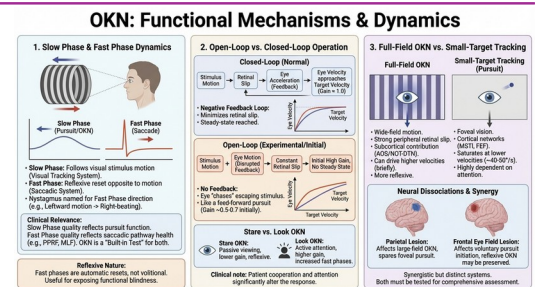
CLINICAL ASSESSMENT PROTOCOL

A. Equipment: Full-field rotating drum (40-60 rpm) or full-screen video display with optokinetic stripes/dots. Must fill >80% of visual field for valid test.

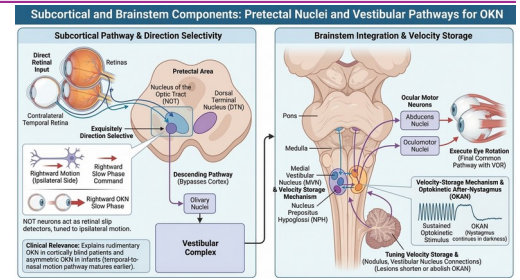
B. Setup: Patient seated 1 metre from stimulus, eyes level with centre, head fixed (chin rest), fully alert and attentive. Test in well-lit room.

C. Stimulus Protocol: Horizontal CW and CCW at 20°/sec, 40°/sec, 60°/sec. Vertical up and down at 20°/sec and 40°/sec. Each direction for 30-60 seconds.

D. Recording: Measure slow-phase gain (eye velocity / stimulus velocity), fast-phase frequency, directional asymmetry index, and OKN suppression ability.



OKN INTERPRETATION GUIDE



| Parameter | Normal | Abnormal Finding | Clinical Significance |
|-----------------------|-------------------------|-------------------------------|---|
| OKN Gain (slow-phase) | >0.80 at 20-40°/sec | <0.40 | Severe brainstem or cortical dysfunction |
| Directional Asymmetry | <20% L-R difference | >30% difference | Ipsilateral hemispheric lesion likely |
| Velocity Range | Normal at 10-60°/sec | Reduced across all velocities | Subcortical: flat; cortical: velocity-dependent |
| OKAN Duration | 15-30 sec post-stimulus | <5 sec or absent | Vestibular velocity storage dysfunction |
| Fast-Phase Frequency | 2-4 per second | <1 per second | Brainstem saccade generator impairment |

ASYMMETRIC OKN PATTERNS & LOCALISATION

| Pattern | Description | Lesion Location | Differential Diagnosis |
|-----------------------|-------------------------------------|---------------------------------|--------------------------------------|
| Unilateral cortical | Reduced OKN toward lesion side | Contralateral parietal (MT/MST) | Stroke, tumor, MS plaque |
| Bilateral cortical | Globally reduced OKN all directions | Bilateral cortex or subcortical | Progressive degenerative disease |
| Brainstem | Reduced gain + poor fast-phase | Pons (PPRF), medulla | Brainstem stroke, demyelination |
| Cerebellar | Mirrors pursuit deficit pattern | Flocculus, vermis, fastigial | Cerebellar ataxia, degeneration |
| Peripheral vestibular | Asymmetric OKAN only (OKN normal) | Unilateral vestibular loss | Vestibular neuritis, Meniere disease |

OKN VS VOR VS PURSUIT: DIFFERENTIAL PATTERNS

OKN Normal + VOR Abnormal: Peripheral vestibular disease (vestibular neuritis, BPPV, Meniere). Central pathways intact. Expected pattern for acute unilateral loss.

OKN Abnormal + Pursuit Abnormal: Central pathology — cortical (MT/MST) or cerebellar. Shared neural pathways affected. Both rely on smooth eye movement generation.

OKN Asymmetric + Pursuit Normal: Subcortical lesion — brainstem vestibular nuclei or velocity storage. Pursuit uses different cortical input than subcortical OKN.

All Three Abnormal: Brainstem lesion affecting common final pathway. Check saccades — if also impaired, consider diffuse brainstem disease or medication toxicity.

Testing Pearls: OKN is less affected by attention/fatigue than pursuit. Useful in patients unable to cooperate with pursuit testing (children, dementia, inattention).