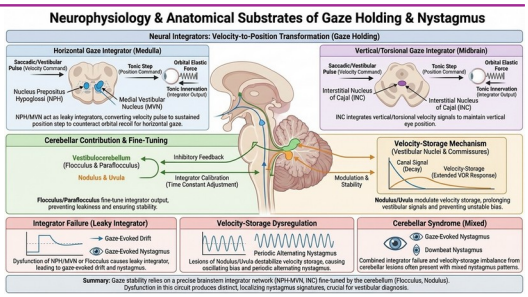
Gaze Holding Function — Clinical Cheat Sheet

OVERVIEW & NEURAL INTEGRATION



Definition: Gaze holding is the ability to maintain eccentric eye position after a saccade. It requires a neural integrator that converts velocity commands into tonic position signals to hold the eyes steady.

Neural Integrator: The cerebellar flocculus and nucleus prepositus hypoglossi (NPH) form the neural integrator. This circuit mathematically integrates (calculates) velocity-to-position transformation.

Clinical Marker: Gaze-evoked nystagmus (GEN) is the hallmark sign of neural integrator failure. Eyes drift back toward midline (centripetal drift) followed by corrective saccades back to the eccentric target.

Clinical Importance: GEN is highly specific for cerebellar flocculus dysfunction and is one of the earliest signs of cerebellar disease, often appearing before limb ataxia or dysarthria.

TYPES & PATTERNS OF GAZE-EVOKED NYSTAGMUS

Type	Mechanism	Direction	Clinical Significance
Horizontal GEN	Lateral cerebellar (flocculus) lesion	Horizontal, direction-changing	Ipsilateral cerebellar hemisphere dysfunction
Vertical GEN	Midline cerebellar (nodulus/vermis)	Vertical (up or downbeat)	Midline or bilateral cerebellar pathology
Rebound Nystagmus	Neural integrator fatigue/adaptation	Reverses direction on re-centring	Highly specific cerebellar disease marker
Periodic Alternating	Nodulus bilateral imbalance (4 min cycle)	Changes direction cyclically	Multiple system atrophy hallmark sign
Bruns Nystagmus	Combined peripheral + central	Direction changes with gaze side	CPA angle tumour (acoustic neuroma)

CLINICAL ASSESSMENT PROTOCOL

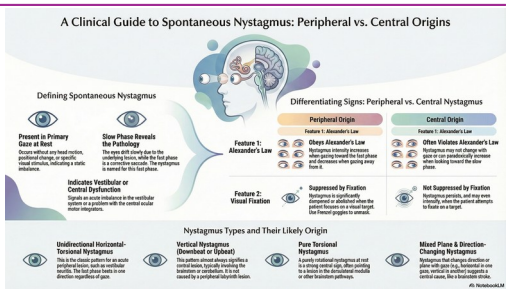
A. Gaze Testing: Have patient fixate targets at horizontal ±10°, ±20°, ±30° and vertical ±10°, ±20°. Hold each position for at least 20 seconds. Observe for nystagmus onset, amplitude, and direction.

B. Fatigue Assessment: Hold gaze at ±30° for >30 seconds. Note if nystagmus amplitude increases with sustained gaze (characteristic of cerebellar disease) or decreases (Alexander law).

C. Rebound Test: After holding extreme gaze (±30°) for 30 seconds, rapidly shift eyes to midline. Observe for brief nystagmus beating in the opposite direction — rebound nystagmus is highly specific for cerebellar pathology.

D. Recording: Use infrared video or EOG to quantify slow-phase velocity, amplitude, and frequency at each gaze position. Document symmetry between right and left gaze.

GAZE-EVOKED NYSTAGMUS: INTERPRETATION GUIDE



Finding	Localisation	Pathology Type	Key Differentiator
Horizontal GEN (one side)	Ipsilateral cerebellar hemisphere	Cerebellar stroke, tumour, atrophy	Direction-changing with gaze direction
Bilateral horizontal GEN	Bilateral cerebellar or brainstem	Medication toxicity, degeneration	Present in both lateral gaze positions
Vertical/rotary GEN	Midline cerebellum (nodulus, vermis)	MSA, spinocerebellar ataxia	Upbeat or downbeat with vertical gaze
Rebound nystagmus	Cerebellar adaptive fatigue	Cerebellar disease confirmation	Reverses direction when eyes return to midline
No GEN at ±30°	Normal neural integrator	Non-cerebellar or pre-symptomatic	Check pursuit and saccades for subtle signs

DIFFERENTIAL DIAGNOSIS: GAZE-HOLDING DEFICITS

Pattern	Cerebellar Disease	Medication Effect	Peripheral Vestibular
GEN Symmetry	Often asymmetric (worse ipsilesional)	Symmetric bilateral	Asymmetric (ipsilesional only)
Rebound Nystagmus	Present (highly specific)	Usually absent	Absent
Fixation Effect	GEN persists or worsens	GEN persists	Peripheral nystagmus suppressed by fixation
Other Signs	Limb ataxia, dysmetria, dysarthria	Sedation, ataxia, diplopia	Vertigo, hearing loss, tinnitus
Course	Progressive or step-wise	Dose-related, reversible	Acute onset, compensates over weeks

LOCALISATION & CLINICAL INTERPRETATION

Cerebellar vs Brainstem: Cerebellar GEN shows rebound nystagmus and direction-changing pattern. Brainstem GEN often has additional cranial nerve signs (facial weakness, dysarthria).

Medication-Induced GEN: Common with phenytoin, carbamazepine, benzodiazepines, alcohol. Symmetric, bilateral, dose-dependent. Always check medication list before attributing to pathology.

Alexander Law: In peripheral vestibular nystagmus, amplitude increases when gazing in the direction of the fast phase. This distinguishes peripheral from central nystagmus patterns.

Age Consideration: Mild endpoint nystagmus at extreme lateral gaze (>35°) can be physiological in up to 20% of normal subjects. Requires >10° deviation with sustained drift to be considered pathological.

Progression Monitoring: Serial gaze-holding testing can track cerebellar disease progression. Increasing GEN amplitude or development of rebound nystagmus suggests worsening neural integrator function.