Nystagmus Interpretation:
A Practical Guide for Emergency Clinicians

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How to Use This Review

This review provides a systematic approach to nystagmus identification and interpretation in the emergency department. Nystagmus is not a diagnosis in itself—it is a sign that reveals the underlying pathophysiology. Understanding the characteristics of different nystagmus types guides diagnosis and risk stratification.

The document follows a structured clinical format with numbered sections, integrated callout boxes for rapid reference, summary tables, and a references section. It is designed both as a learning resource and a quick-reference tool for practising clinicians.

 Key Point: *Foundational concepts and summary statements that anchor the core scientific content of each section.*

 Clinical Insight: Clinically relevant observations derived directly from the evidence — for direct application in assessment and diagnosis.

 Clinical Pearl: High-yield, memorable clinical points — the take-home messages most likely to influence management or examination performance.

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I. Introduction: Why Nystagmus Matters in the ED

Nystagmus—involuntary, rhythmic oscillation of the eyes—is present in approximately 20–30% of patients presenting with dizziness to emergency departments. The key clinical skill is not merely detecting nystagmus, but interpreting what type of nystagmus is present and what it signals about the underlying pathology. The 2015 American Academy of Neurology guidelines emphasise that nystagmus characteristics are among the most powerful discriminators between central and peripheral vestibular disorders [3,7].

In the ED setting, where imaging turnaround times can delay diagnosis, the bedside identification of nystagmus type can have immediate clinical consequences. Certain patterns—such as direction-changing nystagmus, pure vertical nystagmus, or nystagmus that is not suppressed by visual fixation—are red flags for central pathology (brainstem stroke, cerebellar haemorrhage) and warrant urgent neuroimaging and neurology consultation [2,8] [9].

This review provides a practical framework for ED clinicians to systematically categorise nystagmus based on direction, amplitude, frequency, and suppressibility, with emphasis on the clinical significance of each pattern and its implications for disposition and investigation [7,16].

 Key Point: *Nystagmus is a sign, not a diagnosis. Its characteristics determine urgency of investigation and referral.*

II. Nystagmus Physiology

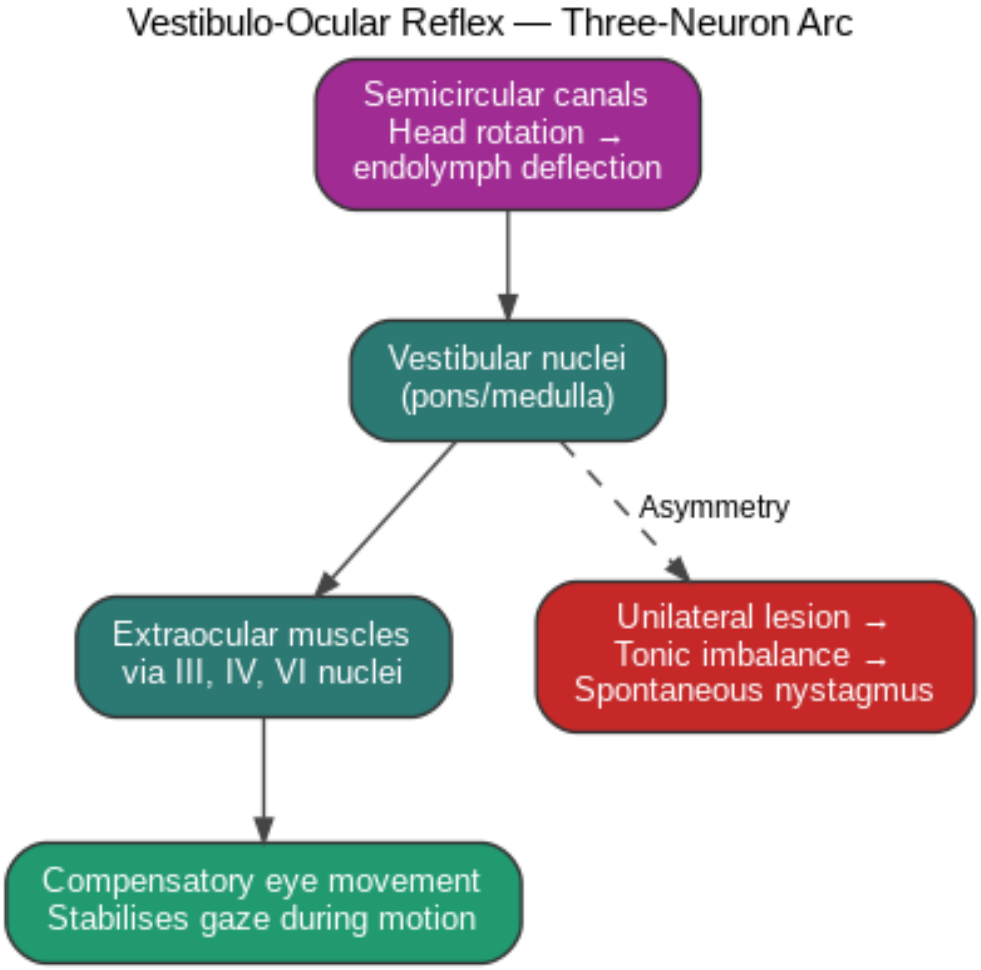

Figure 1. VOR arc and origin of spontaneous nystagmus.

The Vestibulo-Ocular Reflex (VOR)

The VOR is a three-neuron arc connecting the vestibular nuclei to the extraocular muscles. Its function is to stabilise gaze during head movement by generating an eye movement equal in magnitude but opposite in direction to the head movement. The VOR operates at high frequency (up to 4 Hz) and completes its response in ~16 milliseconds—far faster than conscious visual processing [3,10].

Nystagmus emerges when the VOR is imbalanced or overdriven. A unilateral peripheral vestibular lesion disrupts the symmetric input to the vestibular nuclei, causing asymmetric VOR gain. The brain compensates by generating a slow drift toward the lesioned side (slow phase) followed by a corrective saccade back to centre (fast phase, or nystagmus quick phase). The direction of nystagmus is conventionally named after the quick phase [1,15].

Velocity Storage and the Neural Integrator

The cerebellar flocculus contains the "velocity storage" mechanism, which extends the time constant of vestibular input from ~10 seconds to ~30 seconds, allowing sustained compensation for head

acceleration. This mechanism is critical in low-frequency head movements and is vulnerable to brainstem pathology [3].

The neural integrator (located in the medial longitudinal fasciculus and flocculus) converts velocity commands into position commands to the extraocular muscles. Damage to the integrator (cerebellar midline lesions, specific brainstem lesions) produces gaze-evoked nystagmus—a type that emerges when the eyes try to maintain eccentric gaze [3] [13].

 Clinical Pearl: The quick phase of nystagmus is named after its direction; the slow phase points toward the lesioned vestibular apparatus. In peripheral right-sided lesions, you will see leftward (slow) drift and rightward (fast) saccades—hence "right-beating nystagmus."

III. Peripheral Nystagmus

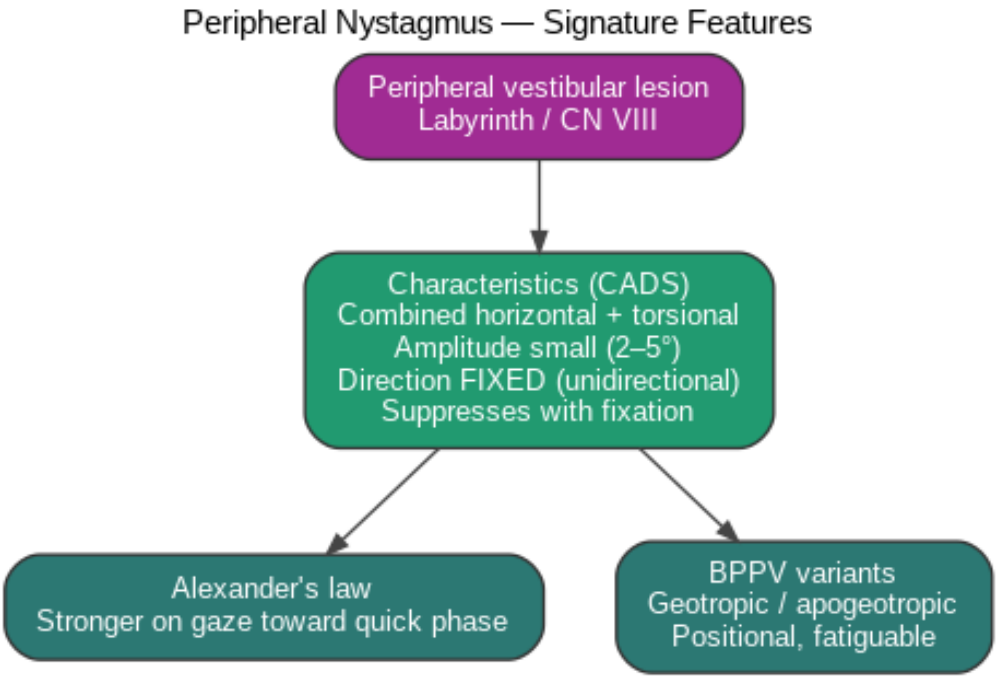

Figure 2. Peripheral nystagmus — signature features.

Characteristics of Peripheral Nystagmus

Peripheral nystagmus arises from a lesion of the labyrinth or vestibular nerve. Its features are distinctive and form the basis of the "CADS" mnemonic: rotatory component, appears during rest, decreases with visual fixation, and linear phase trajectory [1,6,15].

True peripheral nystagmus is unidirectional (beats in the same direction regardless of gaze direction). Amplitude is typically small (2–5 degrees). Frequency ranges from 2–6 Hz. The key distinguishing feature is suppression by visual fixation—when a patient focuses on a stationary object, peripheral nystagmus diminishes or disappears within 1–2 seconds [1,6,15].

Direction-Fixed and Mixed Horizontal-Torsional Nystagmus

Acute unilateral peripheral lesions (acute vestibular neuritis, acute labyrinthitis) produce a mixed horizontal-torsional nystagmus beating away from the affected side. The torsional component is often

subtle but important—the eye on the affected side appears to rotate in a way that "downslopes" the affected ear [6,15,20].

In benign paroxysmal positional vertigo (BPPV), nystagmus is often geotropic (rotates toward the earth in supine position) or apogeotropic depending on canal involvement. The latency before onset (typically 2–5 seconds), limited duration (usually <60 seconds), and fatigability (decreases with repeated positioning) are hallmark features that distinguish BPPV nystagmus from other types [4,11].

Alexander's Law

Alexander's law states that peripheral nystagmus increases in amplitude and sometimes frequency when gaze is directed in the direction of the quick phase (toward the lesion side). This makes intuitive sense: the lesioned vestibular system is being further challenged by trying to maintain eccentric gaze. Conversely, nystagmus diminishes when the patient looks away from the quick phase [3].

This law helps differentiate peripheral from central nystagmus. Central nystagmus often violates Alexander's law or shows direction-changing characteristics that peripheral lesions do not produce [3,7] [3].

 Key Point: *Peripheral nystagmus is suppressed by fixation, is direction-fixed, and follows Alexander's law (increases when eyes move toward the quick phase).*

 Clinical Insight: If a patient with acute vertigo has nystagmus that persists despite strong visual fixation (e.g., focusing on a finger 10 cm away), suspect a central lesion until proven otherwise.

IV. Central Nystagmus

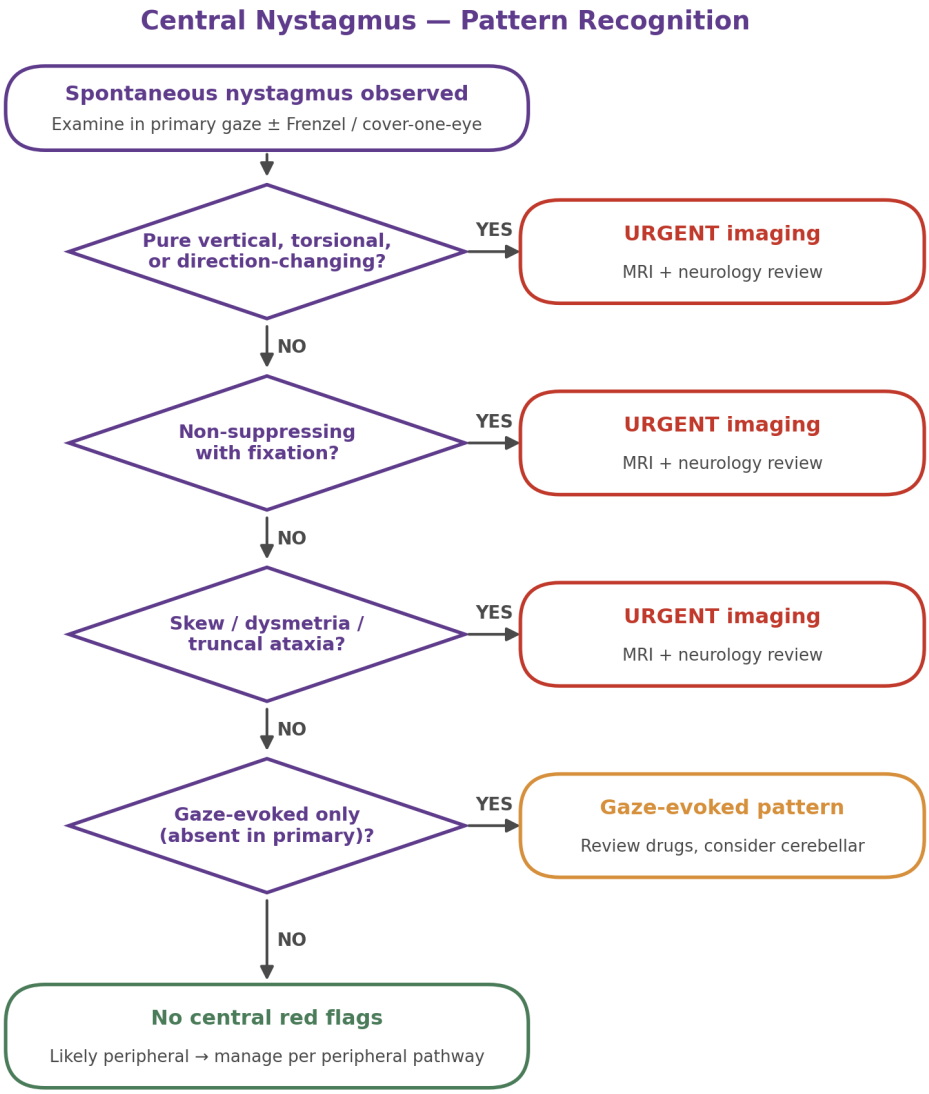

Figure 3. Central nystagmus — pattern recognition.

Direction-Changing Nystagmus

Direction-changing nystagmus — in the central red-flag sense — refers to nystagmus whose fast-phase direction reverses *spontaneously over time* (classic: periodic alternating nystagmus) or *with changes in head position*. It may be present in primary gaze. Although gaze-evoked nystagmus also technically changes direction with gaze (right-beating on right gaze, left-beating on left gaze), clinicians use “direction-changing” specifically to describe this more ominous pattern seen in multifocal brainstem or cerebellar lesions [7,8].

Distinguishing direction-changing from gaze-evoked: • Gaze-evoked appears ONLY on eccentric gaze, is absent in primary gaze, and beats predictably in the direction of gaze — reflects neural integrator failure (cerebellar/brainstem lesion, drugs, fatigue). • Direction-changing (red-flag sense) may be present in primary gaze, reverses spontaneously or with head position, and indicates multifocal pathology — includes periodic alternating nystagmus and central positional nystagmus.

This pattern emerges from dysfunction in brainstem nuclei (particularly the medial longitudinal fasciculus or abducens nucleus) or cerebellar flocculus damage. The most common causes in the ED are brainstem stroke, cerebellar haemorrhage, and certain drugs (phenytoin, antihistamines at high dose, benzodiazepines) [3,7].

Gaze-Evoked Nystagmus

Gaze-evoked nystagmus appears when a patient attempts to maintain gaze in an eccentric position—typically visible after 10–15 degrees of eye deviation. It is not present at rest in primary gaze. The slow phase drifts back toward centre (creep) and is corrected by saccades (quick phase), producing nystagmus that beats in the direction of gaze [3,14].

Common causes include cerebellar disease (most frequent), brainstem lesions (MS, stroke), drug intoxication (alcohol, sedatives, anticonvulsants), and fatigue. In the ED, gaze-evoked nystagmus warrants neuroimaging and consideration of brainstem or cerebellar pathology, especially if accompanied by other neurological signs [3,14] [13].

Pure Vertical Nystagmus

Pure downbeat nystagmus (beating downward) is uncommon but highly significant. It indicates dorsal brainstem or cerebellar nodulus pathology. Causes include cerebellar degeneration, Chiari malformation, brainstem stroke, and post-trauma hydrocephalus. In the acute ED setting, downbeat nystagmus is a red flag for structural pathology requiring urgent MRI [9,14] [11].

Upbeat nystagmus (beating upward) is rarer and classically associated with ventral brainstem lesions, particularly near the medial longitudinal fasciculus or interstitial nucleus of Cajal. Causes include midbrain stroke, MS, and vertically transmitted (hereditary) downbeat nystagmus transitioning to upbeat nystagmus [3,9] [11].

Pure Torsional Nystagmus

Pure torsional nystagmus (rotational oscillations around the anteroposterior axis) without horizontal or vertical components is rare in acute presentations but highly specific for central pathology when present. It suggests damage to the rostral interstitial nucleus of the medial longitudinal fasciculus (riMLF) or cerebellar flocculus. In the ED, this finding mandates urgent neuroimaging and neurology input [3].

Non-Fixation-Suppressing Nystagmus

Nystagmus that persists or only minimally decreases during strong visual fixation is a critical red flag for central pathology. The cerebellum and brainstem nuclei contribute to the fixation mechanism; disruption of these pathways prevents normal suppression [7,8].

Test this carefully: ask the patient to fixate on a target held 10 cm in front of the eyes, then observe for 3–5 seconds. If nystagmus persists undiminished, this strongly suggests central disease and warrants imaging [3,7].

⚠ **Important:** Direction-changing, purely vertical, purely torsional, non-suppressed, or gaze-evoked nystagmus are red flags for central pathology (brainstem/cerebellar disease). These warrant urgent neuroimaging.

V. Spontaneous Nystagmus Assessment

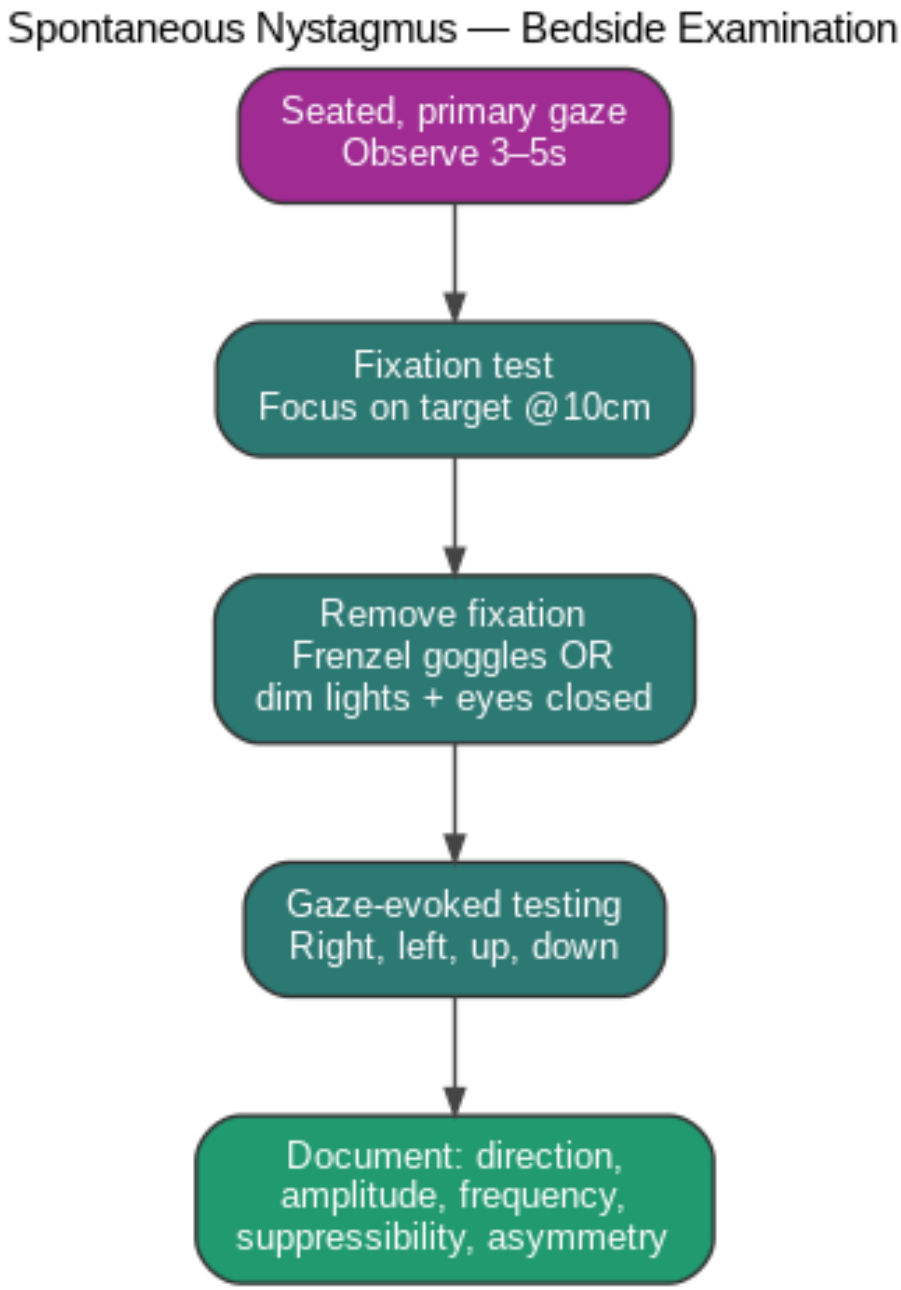

Figure 4. Spontaneous nystagmus — bedside examination sequence [17].

Bedside Examination Technique

Begin with the patient seated and looking straight ahead (primary gaze). Observe for 3–5 seconds. Note the direction, amplitude, and frequency of any nystagmus. Then ask the patient to follow your finger as it moves 30 degrees to the right, hold for 3 seconds, then 30 degrees to the left, then up, then down. At each position, observe and record the characteristics of nystagmus [3].

Next, perform the fixation test: ask the patient to focus intently on a fixed target (your finger at 10 cm distance or a penlight). Nystagmus in peripheral disease should dampen significantly; persistent

nystagmus is concerning for central pathology. Time the suppression: in normal peripheral disease, fixation suppression occurs within 1–2 seconds [3,7] [4].

Frenzel Goggles

Frenzel goggles (magnifying lenses, +20 diopters) eliminate visual fixation by magnifying the eyes and blur the visual field. They are invaluable in ED settings where ambient lighting and patient cooperation may be suboptimal. With Frenzel goggles on, peripheral nystagmus may become apparent despite the patient's strong effort to suppress it [3,16].

If you do not have access to Frenzel goggles, remove fixation by covering ONE eye (hand or occluder) while the patient looks toward a blank wall — observe the uncovered eye for nystagmus and alternate sides. Alternatively, perform ophthalmoscopy on one eye while the fellow eye is covered (any retinal movement is inverse to true eye direction). Observing both closed eyes through the eyelids is unreliable because the sclera is hidden and subtle nystagmus cannot be seen [3].

Documentation and Notation

Document nystagmus systematically: direction (e.g., "right-beating"), amplitude (small/medium/large or in degrees), frequency (slow/moderate/rapid or Hz), suppressibility (suppressed by fixation or not suppressed), and any phase characteristics (linear, decreasing amplitude over time, etc.). Example: "Horizontal right-beating nystagmus, small amplitude (~2–3 degrees), 4 Hz, suppressed by fixation in primary gaze; increases with gaze to the right (Alexander's law)." [3,16] [3]

 Clinical Insight: Testing should be systematic and reproducible. Always test primary gaze first, then the four cardinal directions, then document suppressibility with fixation. Consistency in your examination technique improves accuracy.

VI. Gaze-Evoked and Positional Nystagmus

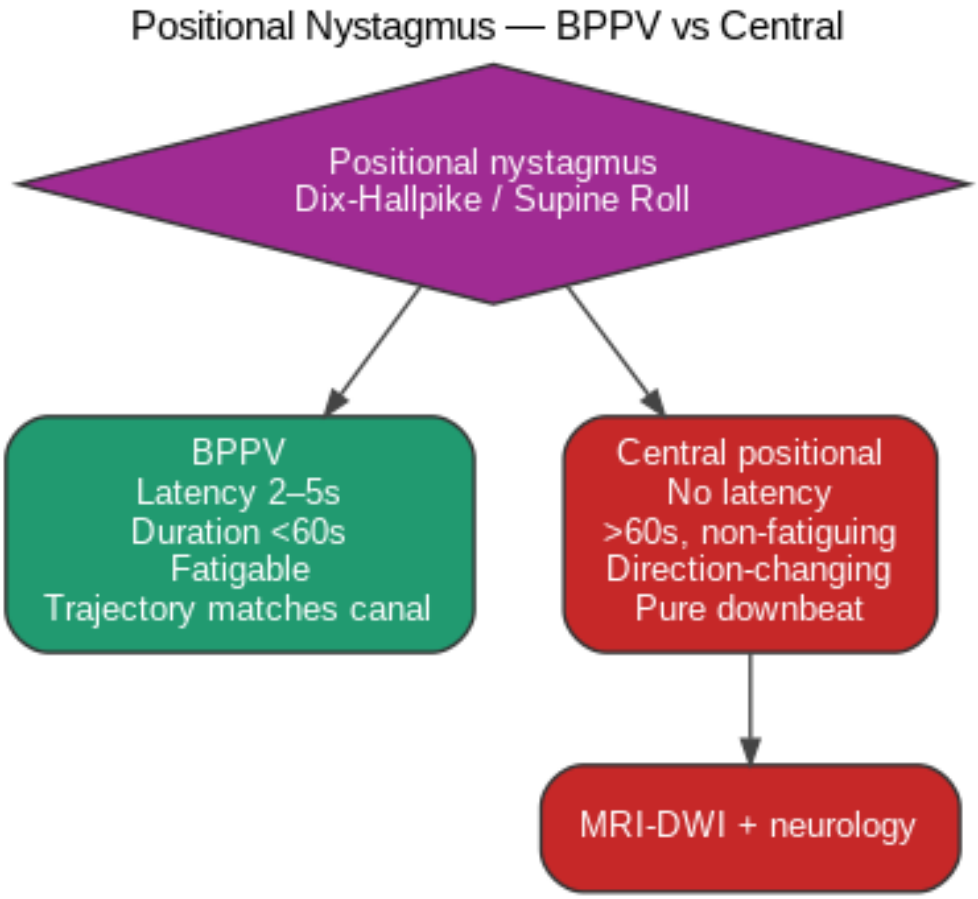

Figure 5. Positional nystagmus — BPPV vs central.

Gaze-Evoked Nystagmus Redux

Gaze-evoked nystagmus emerges after eccentric eye position (typically beyond 20–30 degrees from centre). It is absent in primary gaze and grows progressively in amplitude and frequency as deviation increases. The slow phase shows exponential decay (creep), and saccadic quick phases correct the drift [3,14].

In the ED, gaze-evoked nystagmus warrants exclusion of structural disease (MRI) and consideration of medication effects (sedatives, anticonvulsants, alcohol, antihistamines). If accompanied by ataxia, dysarthria, or other focal signs, emergent neuroimaging is indicated [7,13] [13].

Positional Nystagmus: BPPV vs Central

BPPV produces geotropic or apogeotropic nystagmus with characteristic latency (2–5 seconds), limited duration (<60 seconds), and fatigability. The Dix-Hallpike manoeuvre (head hung 20 degrees below horizontal with 45-degree rotation) should produce nystagmus in posterior canal BPPV. Horizontal canal BPPV is tested with the supine roll test (rapid 90-degree turns side to side) [4,11].

Central positional nystagmus (from brainstem or cerebellar lesions) typically has no latency, persists longer than 60 seconds, does not fatigue, and may be direction-changing. A patient with posterior fossa

stroke may have nystagmus that appears positional but does not fatigue and is accompanied by other brainstem signs (dysarthria, gait ataxia, cranial nerve palsies) [3,7] [9].

 Key Point: *BPPV nystagmus fatigues (decreases with repeated testing); central positional nystagmus does not fatigue and often lacks latency.*

 Clinical Insight: If a patient with "BPPV" symptoms has direction-changing or non-fatiguing nystagmus, strongly consider imaging to rule out brainstem pathology.

VII. Pathological Patterns and Red Flags

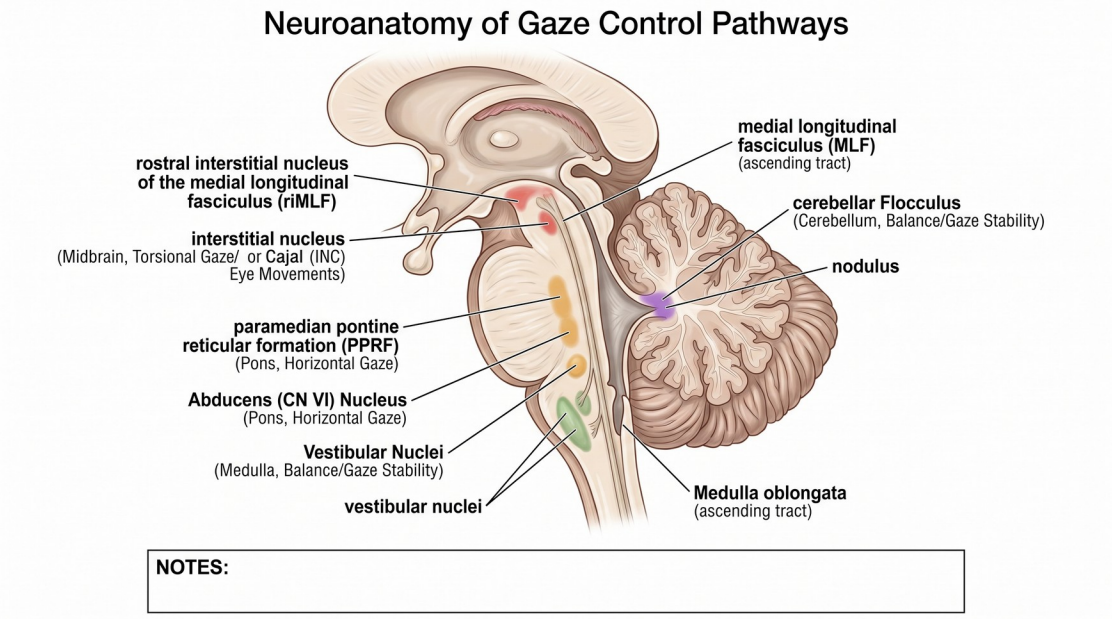

Figure F. Neuroanatomy of gaze control pathways — sagittal view. See Table F below for a detailed legend of each nucleus and its role.

Table F. Key nuclei and tracts in central gaze control

Structure	Region	Role in eye movement
riMLF (rostral interstitial n. of MLF)	Midbrain	Burst generator for vertical & torsional saccades. Lesion → loss of vertical/torsional rapid eye movements.
INC (interstitial nucleus of Cajal)	Midbrain	Neural integrator for vertical & torsional gaze holding. Lesion → torsional/vertical nystagmus, ocular tilt reaction.
PPRF (paramedian pontine reticular formation)	Pons	Horizontal saccade generator. Projects to ipsilateral abducens nucleus. Lesion → ipsilateral horizontal gaze palsy.
Abducens nucleus (CN VI)	Pons	Final common pathway for horizontal gaze — contains motor neurons to lateral rectus and interneurons to contralateral medial rectus via MLF.
Vestibular nuclei (superior, medial, lateral, inferior)	Medulla / pontomedullary	Receive primary afferents from semicircular canals & otoliths. Drive the VOR; integrate head motion signals. Asymmetric lesion → spontaneous nystagmus.
MLF (medial longitudinal fasciculus)	Brainstem tract	Connects abducens interneurons to contralateral medial rectus subnucleus of CN III. Lesion → internuclear ophthalmoplegia (INO).

Flocculus	Cerebellum (vestibulocerebellum)	Adaptive gain control of VOR and smooth pursuit. Lesion → gaze-evoked, downbeat, rebound nystagmus; impaired VOR cancellation.
Nodulus (and uvula)	Cerebellum (vestibulocerebellum)	Processes low-frequency vestibular signals; time constant control. Lesion → periodic alternating nystagmus, central positional nystagmus.

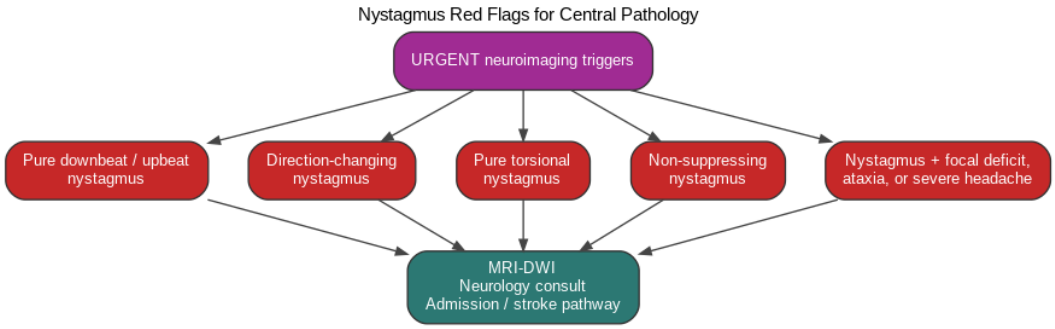

Figure 6. Nystagmus red flags for central pathology.

Head-Shaking Nystagmus

Head-shaking nystagmus (HSN) is elicited by asking the patient to shake their head side to side at ~2 Hz for 10 seconds, then stop abruptly while you observe the eyes. In peripheral vestibular disease (healed unilateral lesion, chronic bilateral vestibular hypofunction), HSN beats away from the lesioned side. In central disease, HSN can be direction-changing or persist beyond a few beats [1,3] [9].

HSN is particularly useful in chronic dizziness but less so in acute ED presentations. However, it can reveal subtle asymmetries in patients recovering from acute vestibular events or those with incomplete compensation [1].

Table 1: Peripheral vs Central Nystagmus Features

Feature	Peripheral Nystagmus	Central Nystagmus
Direction	Unidirectional, fixed	Direction-changing (with gaze) or purely vertical/torsional
Suppressibility	Suppressed by fixation (1–2 sec)	Not suppressed by fixation or partial suppression
Amplitude	Small (2–5°)	Variable, often larger
Associated symptoms	Severe vertigo, nausea	May be less severe vertigo; neurological signs (dysarthria, ataxia, CN palsies)
Rotational component	Often torsional (mixed)	Pure horizontal, vertical, or torsional
Latency (if positional)	2–5 seconds (BPPV)	No latency or immediate onset
Fatigability (if positional)	Yes (BPPV fatigues)	No, persists

Red flags for central nystagmus include direction-changing pattern, lack of fixation suppression, purely vertical or torsional direction, and associated focal neurological signs [7,8] [4].

Red Flags for Central Pathology

Any of the following warrant urgent neuroimaging and neurology consultation:

- **Pure downbeat or upbeat nystagmus** — suggests brainstem or cerebellar lesion [12].
- **Direction-changing nystagmus** — indicates multifocal brainstem involvement.
- **Purely torsional nystagmus** — highly specific for riMLF or flocculus lesion.
- **Non-suppressing nystagmus** — persistent despite strong fixation effort; implies CNS disease.
- **Acute downbeating with ataxia** — possible cerebellar haemorrhage or infarction with oedema.
- **Nystagmus + acute focal neurological signs** (dysarthria, hemiparesis, gait ataxia, cranial nerve palsies) — suspect brainstem stroke.
- **Nystagmus + severe headache + vomiting** — possible posterior fossa pathology.

Examining the "Dangerous" Peripheral Presentation

A critical pitfall is the patient with acute vertigo, horizontal nystagmus that appears to be peripheral (direction-fixed, suppressed by fixation), but actually has a brainstem lesion. Classic examples include lateral medullary stroke (Wallenberg syndrome), which can present with peripheral-appearing nystagmus but is accompanied by ipsilateral facial pain/sensory loss, ipsilateral Homer's syndrome, and contralateral body sensory loss [2,8,17].

Always examine the whole patient. Perform a focused neurological assessment: gait (ataxia?), cranial nerves (facial droop, tongue deviation?), motor (hemiparesis?), sensory, and coordination tests (finger-to-nose, rapid alternating movements). If any focal findings are present alongside nystagmus, imaging is mandatory [2,8].

 Clinical Pearl: Do not rely on nystagmus characteristics alone to exclude central disease. Always perform a complete neurological examination. Wallenberg syndrome can "look peripheral" on eye examination but has multiple central signs.

VIII. Conclusions

Nystagmus is a powerful bedside sign that guides ED diagnosis and risk stratification in dizziness. The distinction between peripheral and central patterns is clinically crucial and usually identifiable with systematic examination [7,8,16] [8].

Peripheral nystagmus is unidirectional, suppressed by fixation, often torsional, and typically accompanied by severe vertigo. Central nystagmus is direction-changing or purely vertical/torsional, not suppressed by fixation, and often accompanied by other neurological signs [1,6,15] [9].

When red flags for central pathology are identified—direction-changing nystagmus, purely vertical or torsional direction, non-suppression, or associated focal neurological findings—urgent imaging (CT head ± CTA spine, followed by MRI) and neurology consultation are indicated [2,8,13] [9].

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