The HINTS Exam:

A Stroke Triage Tool for Emergency Clinicians

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How to Use This Review

This literature review examines the Head Impulse, Nystagmus, Test of Skew (HINTS) examination battery for stroke detection in acute vestibular syndrome. The HINTS exam is a bedside diagnostic tool with sensitivity approaching 100% for central causes of acute dizziness when performed correctly.

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

Background and Clinical Significance

The sudden onset of vertigo, nausea, imbalance, and nystagmus is one of the most common presentations to the modern emergency department, accounting for between three and four percent of all ED attendances in published audits [13]. The clinical challenge lies not in recognising that the patient is dizzy, but in determining whether the underlying cause is a benign peripheral vestibulopathy or a posterior circulation stroke. Historically, clinicians have leaned on neuroimaging to exclude stroke, but computed tomography is insensitive in the first 24 hours and magnetic resonance imaging is frequently unavailable out of hours [3,5]. A structured bedside approach is therefore indispensable.

The Head Impulse, Nystagmus, Test of Skew (HINTS) battery, introduced by Kattah and colleagues in 2009, provides a rapid, reproducible framework that outperforms early diffusion-weighted MRI for central cause detection in the acute vestibular syndrome [1]. Its adoption in emergency medicine has accelerated substantially over the past decade, driven by evidence that HINTS-guided triage reduces both missed posterior circulation strokes and unnecessary imaging [6,9].

Acute vestibular syndrome (AVS) affects approximately 5–10 per 100,000 people annually, with stroke prevalence within the AVS cohort reported between 8% and 20% across large prospective series [1,11,19]. Of these strokes, the vast majority involve the vertebrobasilar territory, with posterior inferior cerebellar artery (PICA) and anterior inferior cerebellar artery (AICA) infarcts dominating the distribution. The consequences of a missed central cause are significant: cerebellar infarcts are prone to oedematous swelling, brainstem compression, and obstructive hydrocephalus within 24–72 hours of onset, and mortality in the undiagnosed group exceeds 40% [13].

Early differentiation using a reliable bedside instrument therefore carries measurable morbidity and mortality benefit [6,9]. The HINTS examination has become the cornerstone of that bedside instrument in the contemporary ED.

 Clinical Pearl: HINTS sensitivity for stroke in acute vestibular syndrome approaches 100% in expert hands. The examination takes <2 minutes and requires minimal equipment.

II. The Acute Vestibular Syndrome

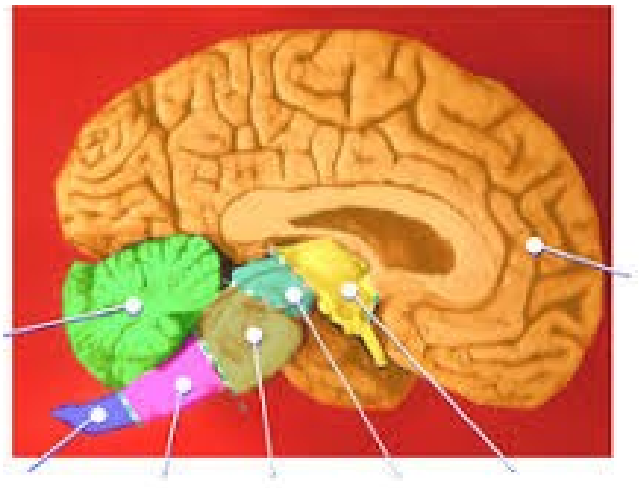

Figure A. Cerebellum and brainstem relationships — the posterior fossa structures implicated in central AVS.
Source: Wikimedia Commons

Definition and Features

Acute vestibular syndrome is defined by sustained vertigo lasting greater than 24 hours, spontaneous nystagmus, and gait instability or ataxia [1,3]. The syndrome typically develops over minutes to hours and persists continuously, distinguishing it from the brief episodic vertigo seen in benign paroxysmal positional vertigo (BPPV) or the recurrent spells of vestibular migraine and Meniere disease. Nausea and vomiting are near-universal. Most patients cannot walk unaided and will veer or fall toward the side of the lesion in peripheral disease, or exhibit midline ataxia with inability to sit unsupported in cerebellar involvement [11].

Importantly, AVS is a syndrome, not a diagnosis. Its recognition merely triggers a structured workup; the critical clinical question is whether the cause is peripheral (vestibular neuritis, labyrinthitis) or central (posterior circulation stroke, demyelination, rarely cerebellar haemorrhage) [3,6].

Stroke Prevalence

Large prospective studies have found that 8–20% of patients presenting with AVS ultimately have posterior circulation stroke [1,11,19]. The remaining 80–92% have peripheral vestibular disease, most commonly vestibular neuritis. Stroke risk rises with conventional vascular risk factors (hypertension, diabetes, atrial fibrillation, prior stroke, age over 60), but the absence of these factors does not reliably exclude stroke and should not be used as a triage tool in isolation [18,20].

Younger patients with vertebral artery dissection represent a particularly important subgroup; they often lack classical vascular risk factors, and posterior dissection may present with isolated vertigo or neck pain preceding brainstem signs by hours or days [19]. A high index of suspicion remains essential across all age groups.

 Key Point: *Approximately 1 in 10 patients with acute vestibular syndrome has a stroke. The HINTS exam can identify >99% of these cases.*

III. HINTS Components

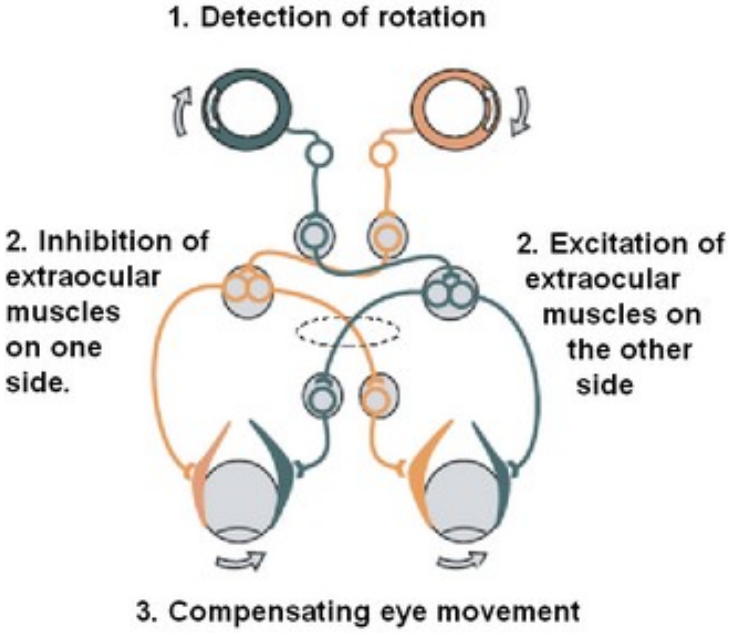

Figure B. The vestibulo-ocular reflex pathway — the neural circuit tested by the head impulse test.
Source: Wikimedia Commons

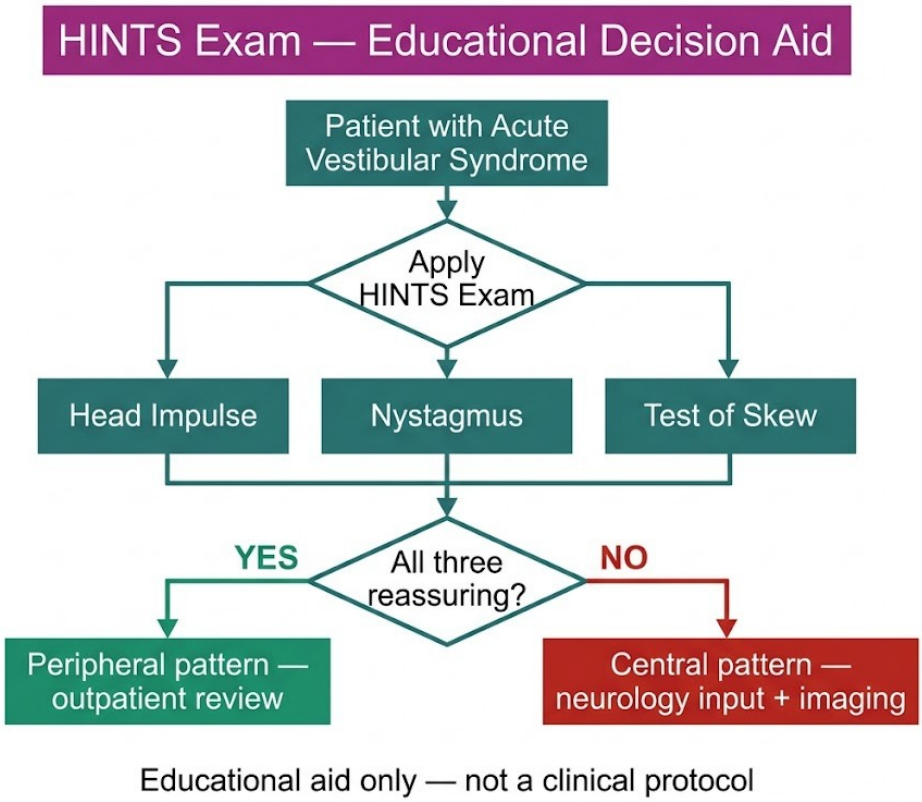

Figure 1. HINTS bedside decision pathway.

Head Impulse Test (HIT)

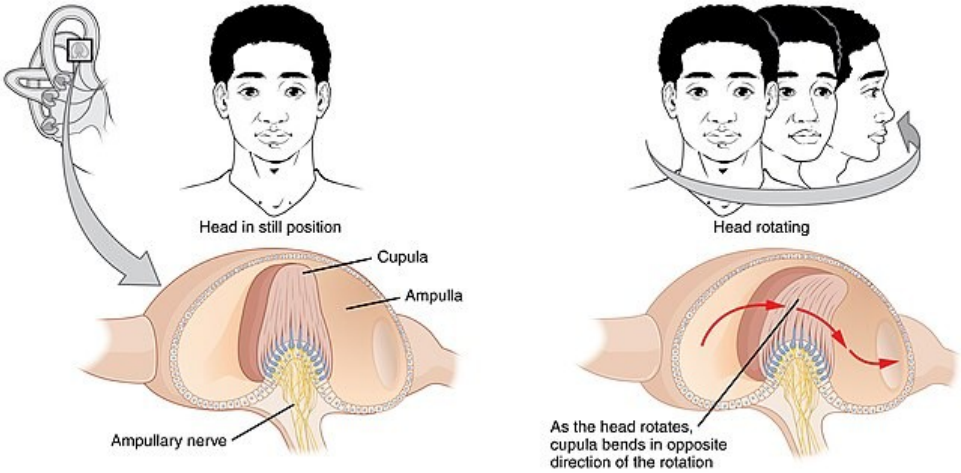

Figure C. Horizontal semicircular canal and cupula — the peripheral sensor driving the horizontal VOR.
Source: Wikimedia Commons

The head impulse test (HIT) evaluates the vestibulo-ocular reflex (VOR). The patient fixates on a central target (typically the examiner's nose) while the examiner rapidly rotates the patient's head approximately ±10–15 degrees in the horizontal plane [4,8]. A healthy VOR produces a gain close to 0.95, meaning the eyes move equal and opposite to the head and remain locked on the target throughout. If the VOR is impaired, the eyes move with the head and a corrective saccade is generated once the head stops, which the examiner visualises as a refixation saccade back to the target.

Technique matters. Head movements must be unpredictable in timing and direction, brisk (peak velocity >150°/s), and of small amplitude. Covert saccades that occur during head movement can be missed at the bedside but are detectable on video head impulse testing (vHIT), where they appear as saccades

occurring before the head has stopped. In the emergency department, standard bedside HIT alone retains excellent discriminative power when performed by trained clinicians [1,8].

A normal HIT — that is, no refixation saccade in a patient with acute ongoing vertigo and nystagmus — is paradoxically concerning: it suggests the peripheral vestibular apparatus is intact and raises strong suspicion for a central (brainstem or cerebellar) cause [1,16]. An abnormal HIT with overt corrective saccades indicates peripheral vestibular hypofunction and supports the diagnosis of vestibular neuritis. This counter-intuitive logic is the single most important principle of the HIT in the HINTS battery and is the most frequent source of clinician error during teaching sessions.

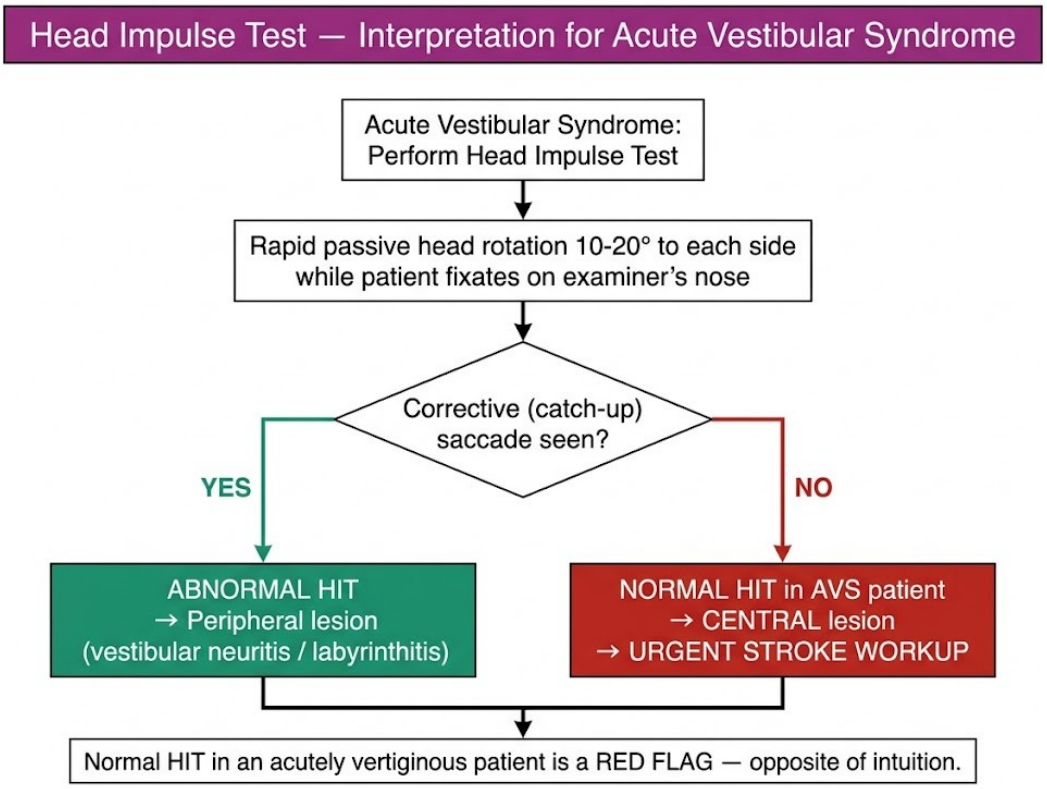

Figure 2. Head Impulse Test — interpretation flow.

Two caveats apply. First, AICA territory infarction may mimic peripheral disease on HIT because the labyrinthine artery, a branch of AICA, can be involved and produce genuine peripheral vestibular dysfunction alongside central signs [5]. Second, bilateral vestibulopathy (for example from aminoglycoside toxicity) may produce bilaterally abnormal HIT that mimics central disease [10]. These pitfalls underscore the need to interpret HIT in the context of the full HINTS battery rather than in isolation.

 Clinical Pearl: The head impulse test is the single most powerful component of HINTS for identifying central causes.

Nystagmus Assessment

In peripheral vestibular disease, nystagmus is direction-fixed — it beats in the same direction regardless of gaze direction, and conforms to Alexander's law (amplitude and velocity increase when the patient looks toward the fast phase) [3,16]. In central disease, nystagmus may be direction-changing on gaze, purely vertical, torsional, or gaze-evoked. Pure vertical nystagmus (upbeat or downbeat) is highly suggestive of a brainstem or cerebellar localisation and is essentially never peripheral [16].

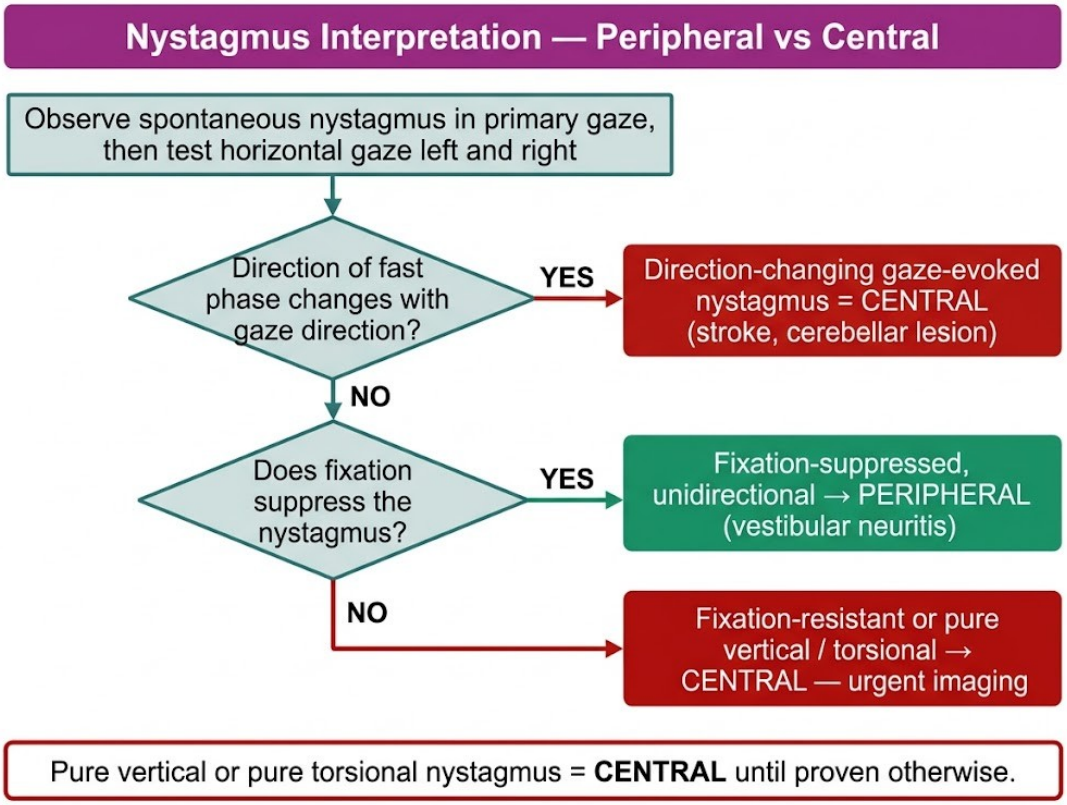

Figure 3. Nystagmus interpretation algorithm.

Visual fixation normally suppresses peripheral vestibular nystagmus; removal of fixation (Frenzel lenses, or simply covering one eye while observing through the other) typically enhances peripheral nystagmus but has little effect on central nystagmus. This "failure of fixation suppression" is an adjunctive central sign and can be assessed without specialised equipment [12].

Test of Skew

The test of skew assesses vertical eye alignment. Using a cover-uncover technique, the examiner alternately covers each eye and observes for vertical corrective movement on uncovering. Any vertical corrective saccade constitutes a positive test and indicates skew deviation, a sign of brainstem or cerebellar dysfunction highly specific for central pathology [14]. Skew is classically absent in peripheral vestibular disease [1].

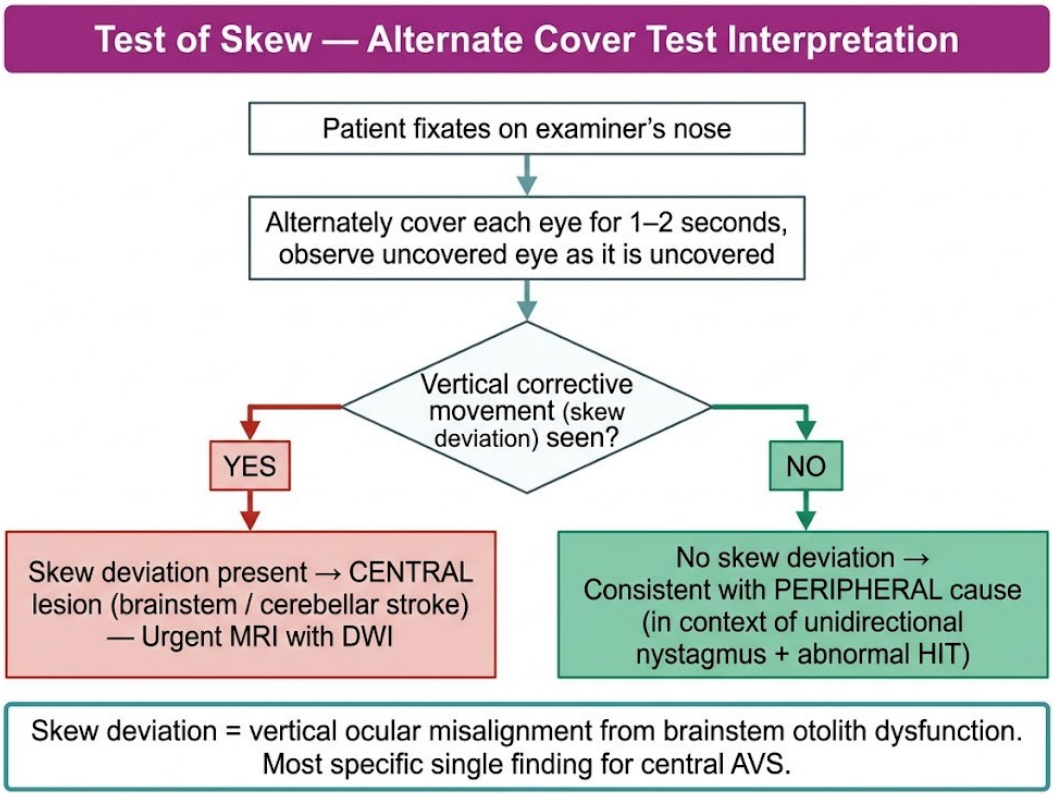

Figure 4. Test of Skew — assessment pathway.

Skew deviation arises from disruption of the graviceptive pathways connecting the otolith organs, vestibular nuclei, and ocular motor nuclei. A small-amplitude skew may be subtle; the examiner should perform several cover–uncover cycles on each side and compare the findings with the patient's usual baseline where possible.

Table 1. HINTS Examination Findings

Finding	Peripheral	Central
Head Impulse Test	Abnormal (saccades)	Normal (no saccades)
Nystagmus Type	Direction-fixed	Direction-changing or vertical
Test of Skew	Normal	Abnormal (skew present)

⚠ **Important:** The combination of normal HIT, direction-changing nystagmus, or skew deviation indicates central pathology and mandates urgent MRI and neurology consultation.

IV. HINTS Validation and Sensitivity

The Kattah 2009 Landmark Study

Kattah and colleagues prospectively performed HINTS on 101 patients presenting with acute vestibular syndrome. Sixty-eight had peripheral vestibular neuritis, 25 had posterior circulation stroke, and eight had alternative central diagnoses (multiple sclerosis, cerebellar haemorrhage) [1]. A "central" HINTS pattern — defined as any one of: normal HIT, direction-changing nystagmus, or skew deviation — identified all central causes with 100% sensitivity and 96% specificity. Critically, HINTS outperformed early diffusion-

weighted MRI, which missed approximately 12% of posterior fossa strokes imaged within the first 24–48 hours.

Subsequent validation studies in emergency department cohorts have broadly replicated these findings, with pooled sensitivity of 96–97% and specificity of 85–95% when performed by clinicians with appropriate training [2,9]. The addition of hearing assessment (HINTS-Plus) further raises sensitivity by detecting AICA-territory infarcts that present with vertigo plus acute hearing loss [5].

Comparison with Imaging

CT brain has sensitivity of only 10–30% for posterior fossa ischaemic stroke in the first 24 hours and remains a poor triage tool in this setting, although it retains a role in excluding haemorrhage [3,6]. MRI with diffusion-weighted imaging is the gold standard with 90–95% sensitivity overall, but sensitivity falls to approximately 80% for small brainstem infarcts imaged within the first six hours [1,6]. HINTS, by contrast, achieves 100% sensitivity in the acute presentation and can be performed at the bedside in under three minutes.

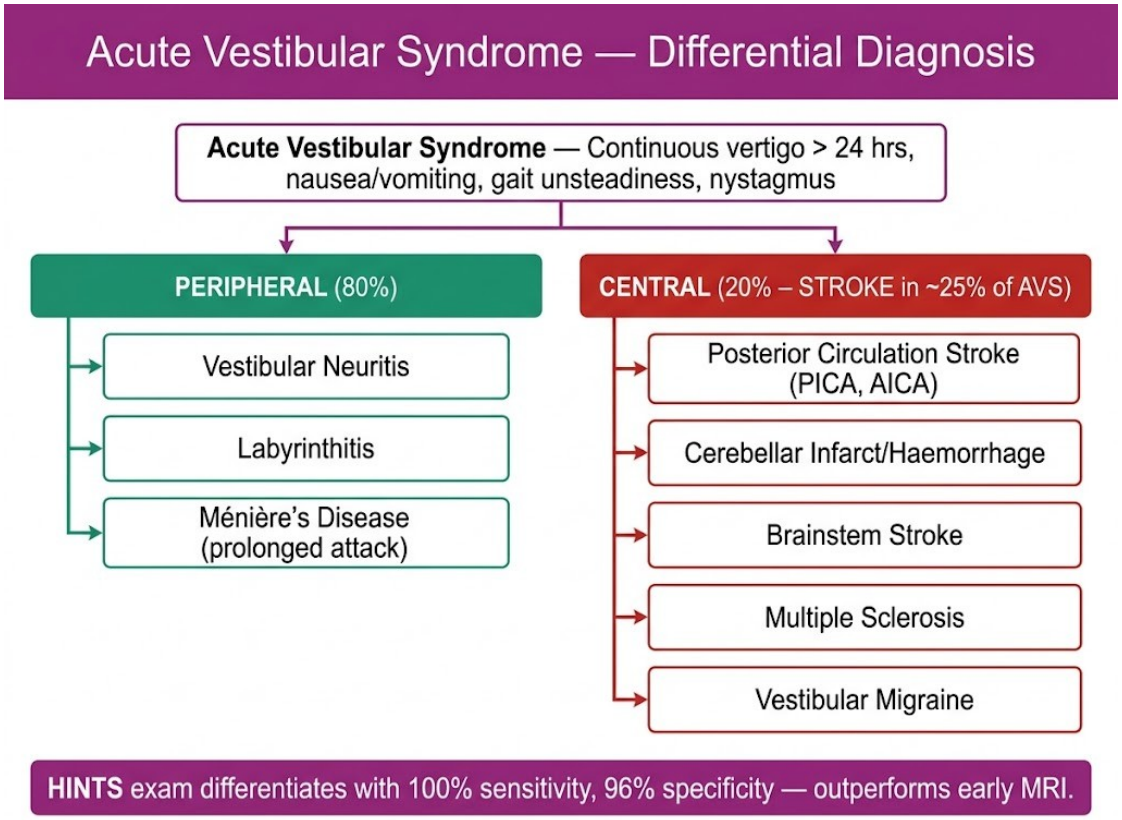

Figure 5. Acute vestibular syndrome — central vs peripheral differential.

The practical implication is that a normal early CT and even a normal early MRI do not exclude posterior circulation stroke, and in a patient with a central HINTS pattern, clinical suspicion should prevail. Repeat MRI at 48–72 hours is recommended in this scenario [6,9].

 Clinical Pearl: Kattah 2009: HINTS sensitivity 100%, specificity 96%. This landmark study established HINTS as a high-value bedside tool.

V. HINTS-Plus and Additions

Hearing Loss Assessment

HINTS-Plus extends the standard three-step battery by adding bedside hearing assessment to detect anterior inferior cerebellar artery (AICA) territory infarction [5]. The labyrinthine artery is usually a branch of AICA, and AICA stroke can therefore produce combined vertigo and acute unilateral sensorineural hearing loss in addition to central signs. Bedside finger rub or whispered voice testing is sensitive enough to identify significant acute hearing loss for this purpose; formal audiometry is not required in the ED.

The combination of acute vertigo and new unilateral hearing loss should be treated as AICA territory infarction until proven otherwise, even if the rest of the neurological examination is normal. Cases presenting in this way have occasionally been misclassified as labyrinthitis with significant downstream morbidity [5,19].

 Key Point: *In acute vestibular syndrome with unexplained hearing loss, AICA territory stroke must be excluded.*

VI. Pitfalls and Limitations

Timing Windows

HINTS is most reliable in the first 72 hours of acute vestibular syndrome. After this window, central compensation in peripheral vestibulopathy may normalise the head impulse test, and resolution of acute central oedema may mask central nystagmus features, reducing overall diagnostic yield [1,10]. The examination was explicitly validated for "acute" presentations, and its operating characteristics have not been fully established in sub-acute or chronic vertigo.

A corollary is that patients presenting outside the first 72 hours require a lower threshold for neuroimaging, and the absence of central signs on HINTS should not be taken as confidently exclusionary in this delayed-presentation group.

Bilateral Vestibular Disease

Bilateral vestibular disease is a known pitfall where the head impulse test may be abnormal on both sides, potentially mimicking central pathology on one side and causing diagnostic confusion [10]. Causes include aminoglycoside ototoxicity, autoimmune inner ear disease, and bilateral sequential neuritis. A careful drug history, symmetry of findings, and the relative preservation of nystagmus and test of skew features usually allow the clinician to reach the correct conclusion.

Other pitfalls include alcohol intoxication (may cause gaze-evoked nystagmus mimicking central disease), anticonvulsant toxicity, and the rare presentations of vestibular migraine in the ED. In all ambiguous cases, the default should be to triage the patient as central until proven otherwise [18].

⚠ **Important:** HINTS findings should always be integrated with the full clinical picture. When uncertain, obtain MRI DWI.

VII. ED Implementation

HINTS should be performed early in the evaluation of acute vestibular syndrome, before or immediately after ordering neuroimaging [9]. If the examination is entirely reassuring — abnormal HIT, direction-fixed nystagmus, no skew — peripheral vestibular disease is overwhelmingly likely, and the patient can usually

be managed symptomatically with outpatient follow-up. If any central sign is present, or if the examination is indeterminate because of patient cooperation or clinician unfamiliarity, urgent MRI with diffusion-weighted sequences and neurology input should be pursued.

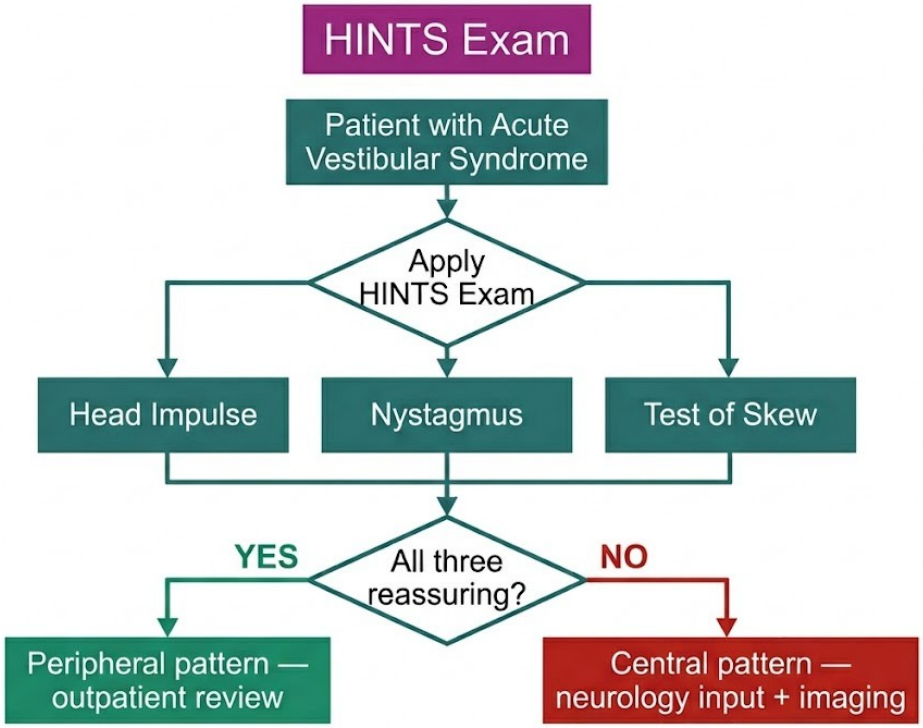

Figure 6. ED disposition following HINTS assessment.

Institutional implementation is best supported by staff education, a standardised documentation template in the ED record, and a clear escalation pathway for central-pattern HINTS findings. Audit data from centres that have adopted a HINTS-based pathway show both reduced time to diagnosis of posterior circulation stroke and reduced CT utilisation in peripheral presentations [6,13].

 Clinical Pearl: HINTS is not a replacement for MRI; it is a rapid triage tool that guides imaging urgency and specialist consultation timing.

VIII. Conclusions

The HINTS examination is a high-yield, evidence-based bedside tool that dramatically improves ED physicians' ability to differentiate central from peripheral causes of acute vestibular syndrome [1,2]. When performed correctly and within the first 72 hours, it outperforms early MRI for central cause detection while being immediately available, inexpensive, and repeatable.

Pitfalls are real but manageable: clinician training, structured documentation, and adherence to the full battery (rather than cherry-picking components) underpin reliable results [6,9]. HINTS is best viewed as the bedside stroke rule-out test for the acute vertiginous patient, complementary to — not replaced by — imaging and neurology consultation where indicated.

 Clinical Insight: For emergency physicians, HINTS is one of the highest-value bedside skills: rapid, sensitive, specific, and immediately actionable in stroke triage.

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