MedCrave
Step into the World of Research

Journal of Otolaryngology-ENT Research

Review Article

Open Access

CrossMark

Bedside oculomotor examination in the acute vestibular syndrome: what hints can and cannot do for the otolaryngologist

Abstract

The acute vestibular syndrome is one of the few presentations in medicine where a self-limiting disorder and a life-threatening one produce an almost identical history. Vestibular neuritis and infarction in the posterior circulation both present with sudden, continuous vertigo, nausea, intolerance of head movement and spontaneous nystagmus, and neither the character of the dizziness nor the presence of vascular risk factors reliably separates them. The three-step HINTS battery — horizontal head impulse test, characterisation of nystagmus, and test of skew — was shown in 2009 to exceed early diffusion-weighted magnetic resonance imaging in sensitivity for stroke in this population, a result that has been quoted ever since as evidence that a competent bedside examination outperforms a scanner. That claim is defensible, but only under conditions that are rarely reproduced in routine practice. This narrative review sets out the physiological basis of each component, examines the accuracy data critically, and argues that the gap between the original figures and the performance achieved in general emergency and outpatient settings reflects examiner skill, patient selection and case mix rather than any intrinsic limitation of the test. Two boundaries are emphasised throughout. First, the battery is validated only in patients who genuinely meet the definition of an acute vestibular syndrome, and its operating characteristics are unknown in any other group; applying it to episodic or triggered dizziness is a taxonomic error, not merely a technical one. Second, findings consistent with a peripheral vestibulopathy reduce the probability of stroke but do not exclude it, and this remains true even when the examination is performed by a trained and experienced clinician; the examination should therefore be interpreted alongside the wider history, the general neurological assessment and the clinical course rather than used as a standalone rule-out test. Particular attention is given to the situations in which otolaryngologists encounter these patients, which are often days after the index event and after a working diagnosis has already been recorded. We conclude with the practical implications for departmental pathways and with the questions the field still needs to answer.

Keywords: acute vestibular syndrome, HINTS examination, vertigo, posterior circulation stroke, head impulse test, nystagmus, skew deviation, vestibular neuritis

Volume 18 Issue 2 - 2026

Dr Anirudda Deshpande,[1] Dr Supriya Khardenavis,[2] Dr Gayatri Godse,[3] Dr Vikram Khardenavis[4]

[1]Consultant Stroke Physician, Department of Stroke Medicine, Altnagelvin Area Hospital, Western Health and Social Care Trust, Glenshane Road, Londonderry BT47 6SB, Northern Ireland, UK
[2]Specialty Doctor, Department of Ophthalmology, Altnagelvin Area Hospital, Western Health and Social Care Trust, Londonderry, UK
[3]Senior Resident, MGM Medical College & Hospital, MGM Institute of Health Sciences, Sector 1, Kamothe, Navi Mumbai 410209, Maharashtra, India
[4]Fellow in Epilepsy, Department of Neurology, Kokilaben Dhirubhai Ambani Hospital and Medical Research Institute, Rao Saheb Achutrao Patwardhan Marg, Four Bungalows, Andheri West, Mumbai 400053, Maharashtra, India

Correspondence: Dr Anirudda Deshpande, Department of Elderly Care and Stroke, Altnagelvin Hospital, Western Health and Social Care Trust, Glenshane Road, Londonderry BT47 6SB, Northern Ireland, UK

Received: August 3, 2026 | **Published:** August 24, 2026

Abbreviations: ABCD², Age, Blood pressure, Clinical features, Duration, Diabetes score, AICA, anterior inferior cerebellar artery, AVS, acute vestibular syndrome, CT, computed tomography, DWI, diffusion-weighted imaging, HINTS, Head Impulse–Nystagmus–Test of Skew, MRI, magnetic resonance imaging; PICA, posterior inferior cerebellar artery, TiTrATE, Timing, Triggers And Targeted Examination, VOR, vestibulo-ocular reflex.

Introduction

Dizziness accounts for something in the region of three to four per cent of emergency department attendances in both North America and Europe, which in the United States alone amounts to several million visits a year.[1-3] The overwhelming majority of these patients are not having a stroke. That is precisely the difficulty. When ninety-five out of every hundred people presenting with a given symptom have a benign cause, any test used to find the remaining five must be extraordinarily good, and any clinician relying on impression alone will be right almost all of the time and catastrophically wrong occasionally.

Otolaryngologists occupy an unusual position in this problem. They are seldom the first clinician to see the patient in the acute phase, yet they are frequently the first with formal training in vestibular examination to do so. A patient who was labelled with labyrinthitis in an emergency department on a Saturday night may reach an ENT clinic ten days later, still unsteady, and the question of whether the original diagnosis was correct falls to the otolaryngologist to answer. It is a question that cannot be settled by repeating the history, because by then the history has been rehearsed several times and has acquired the shape of the diagnosis already given.

This review concerns the examination that has come to dominate acute neuro-otology over the past fifteen years, and the reasons why its published accuracy has proved so difficult to reproduce. Our position is not that HINTS is overrated. It is that HINTS has been asked to do a job — population-level screening by clinicians with no vestibular training — for which it was never designed and for which the evidence does not support it, while simultaneously being underused in the setting where it works best.

Scope, terminology and literature search

Literature search and article selection

This is a narrative review and was not conducted as a systematic review; no formal risk-of-bias appraisal, quantitative pooling or PRISMA-compliant screening was undertaken, and the selection of sources necessarily reflects the authors' judgement of clinical relevance.

©2026 Deshpande et al. This is an open access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and build upon your work non-commercially.

Copyright: ©2026 Deshpande et al.

PubMed/MEDLINE and the Cochrane Library were searched from inception to July 2026, supplemented by Google Scholar for citation tracking. Search terms were combined in various permutations and included "acute vestibular syndrome", "HINTS", "head impulse test", "skew deviation", "nystagmus", "vertigo", "dizziness", "posterior circulation stroke", "cerebellar infarction", "vestibular neuritis", "diagnostic accuracy" and "misdiagnosis". The reference lists of all retrieved articles, together with those of relevant clinical practice guidelines, were hand-searched for further sources.

Articles were eligible for inclusion if they were published in English and reported original diagnostic accuracy data, systematic reviews or meta-analyses, population-based epidemiological studies of misdiagnosis, or authoritative narrative accounts of the underlying oculomotor physiology. Where several studies addressed the same question, priority was given to prospective designs, to studies reporting examiner training explicitly, and to the most recent evidence synthesis. Case reports were consulted only where they illustrated a mechanism not otherwise documented. Conference abstracts without subsequent full publication were excluded, as were non-English-language reports, a limitation we acknowledge.

A note on terminology

The literature describes HINTS results variously as "positive", "negative", "dangerous", "benign", "central" and "peripheral", and the inconsistency is a genuine source of confusion at the bedside — not least because an "abnormal" head impulse test is the reassuring result, so that "positive" and "negative" carry opposite senses depending on whether the writer means the component or the battery. Throughout this review we therefore use two terms only. HINTS concerning for a central lesion denotes a battery in which at least one component shows the central pattern set out in Table 1. HINTS consistent with a peripheral vestibulopathy denotes a battery in which every component shows the peripheral pattern. We would encourage the same convention in clinical documentation, since it records what was observed rather than an inference drawn from it.

Table 1 Bedside patterns in the acute vestibular syndrome

Component	Findings consistent with a peripheral vestibulopathy	Findings concerning for a central lesion
Head impulse test	Abnormal — a visible corrective saccade on thrusting towards the affected side, indicating a failed vestibulo-ocular reflex	Normal — no corrective saccade, because the peripheral reflex arc is intact and the lesion lies beyond it
Nystagmus	Unidirectional, horizontal with a torsional component, fast phase away from the affected ear, obeying Alexander's law, suppressed by visual fixation	Direction-changing on eccentric gaze, or purely vertical, or purely torsional; often not suppressed by fixation
Test of skew	Absent — no vertical refixation on alternate cover testing	Present — vertical corrective movement, indicating disruption of graviceptive pathways, usually in the brainstem
Bedside hearing (HINTS "plus")	Hearing preserved	New unilateral hearing loss, which in this setting should raise the possibility of anterior inferior cerebellar artery territory ischaemia rather than labyrinthitis
Gait and truncal stability	Unsteady but able to walk unaided, often veering towards the affected side	Unable to sit or stand without support, out of proportion to the vertigo

One further point of scope should be stated at the outset and will be repeated, because it is the single most common source of error in the application of this battery. HINTS has been validated only in patients who meet the definition of an acute vestibular syndrome. Its sensitivity and specificity in any other group of dizzy patients are unknown, and the figures quoted in this review should not be transferred to them.

The acute vestibular syndrome and the scale of the diagnostic problem

The acute vestibular syndrome, as defined by Hotson and Baloh, describes the rapid onset over seconds to hours of vertigo, nausea or vomiting, gait unsteadiness and intolerance of head motion, accompanied by nystagmus and persisting for days to weeks.[4] The definition matters more than it appears to. It excludes the patient whose symptoms come in bursts lasting seconds when they roll over in bed, and it excludes the patient who felt terrible for twenty minutes yesterday and is now entirely well. Those patients have different differential diagnoses and require different examinations. Almost every misapplication of HINTS we encounter in practice originates in a failure to establish that the patient in front of you actually has an acute vestibular syndrome.

Within a genuine acute vestibular syndrome, roughly a quarter of cases in hospital series prove to be central, most commonly ischaemic infarction in the territory of the posterior inferior or anterior inferior cerebellar arteries.[5,6] Posterior circulation events account for about a fifth of all ischaemic strokes but are misdiagnosed roughly twice as often as anterior circulation events.[7] Population-level analyses suggest that approximately nine per cent of cerebrovascular events are missed at the index emergency presentation, and that this figure rises steeply — to somewhere between a quarter and sixty per cent depending on the series — when the presenting symptoms are mild, transient or non-specific.[8-10] For posterior circulation strokes presenting specifically with dizziness, frontline misdiagnosis appears to occur in roughly a third of cases.

The consequences are not evenly distributed. A missed lacunar infarct in the internal capsule is a lost opportunity for secondary prevention. A missed cerebellar infarct is a patient who may return forty-eight hours later with obstructive hydrocephalus and brainstem compression, or who may not return at all. A missed vertebrobasilar occlusion is worse still. It is this asymmetry of harm, rather than the raw frequency of error, that has driven the interest in bedside diagnosis.

Citation: Deshpande A, Khardenavis S, Godse G, et al. Bedside oculomotor examination in the acute vestibular syndrome: what hints can and cannot do for the otolaryngologist. *J Otolaryngol ENT.* 2026;18(2):66–72. DOI: 10.15406/joentr.2026.18.00590

Copyright: ©2026 Deshpande et al.

There is a second cost that receives less attention. The response to diagnostic anxiety in most institutions is imaging, usually computed tomography, which is close to worthless for this purpose. It answers a question nobody was asking — is there blood? — and returns a normal result that is then misread as reassurance. The examination that would actually help is the one that is not performed.

Why the old paradigm failed

For four decades the standard approach to dizziness rested on the taxonomy proposed by Drachman and Hart in 1972, which asked the patient to sort their sensation into vertigo, presyncope, disequilibrium or a residual category of lightheadedness.[11] The logic was that the quality of the symptom would localise the lesion. Vertigo meant vestibular; presyncope meant cardiovascular; and so on.

The difficulty is that patients cannot do this reliably. In a study conducted in an acute care setting, over half of patients gave a different answer about the quality of their dizziness when asked again a few minutes later, whereas their descriptions of timing and triggers remained stable.[12] Asking a nauseated, frightened person to introspect about whether the room is spinning or whether they merely feel faint produces an answer, but not a reproducible one. Worse, the answer then determines which examination is performed, so an unreliable input propagates into a confident but unfounded conclusion.

Edlow put the objection bluntly: the wrong paradigm was being taught.[13] What replaced it is a framework built on timing and triggers, sometimes abbreviated as TiTrATE, which sorts patients into an acute vestibular syndrome, a triggered episodic vestibular syndrome and a spontaneous episodic vestibular syndrome before any examination is chosen.[14,15] HINTS belongs to the first of these categories and to no other. Performing a head impulse test on a patient with benign paroxysmal positional vertigo is not merely unhelpful; it generates a finding that will be interpreted, and the interpretation will be wrong.

The three components, and what each is actually testing

The horizontal head impulse test

Described by Halmagyi and Curthoys in 1988, the head impulse test assesses the high-frequency vestibulo-ocular reflex.[16] The examiner turns the patient's head rapidly through a small arc, perhaps ten to twenty degrees, with high acceleration and unpredictable timing, while the patient fixates on the examiner's nose. If the reflex arc is intact the eyes remain locked on target. If the labyrinth or vestibular nerve on that side is damaged, the eyes are dragged with the head and a visible corrective saccade brings them back.

The counterintuitive part, and the part most often forgotten, is the direction of inference. Within an acute vestibular syndrome, an *abnormal* head impulse test is reassuring, because it localises the lesion to the peripheral apparatus. A *normal* head impulse test in a patient with florid ongoing vertigo and nystagmus is concerning, because it says the peripheral reflex is working and the problem lies somewhere central to it.[17] Clinicians accustomed to thinking that abnormal findings are bad and normal findings are good will get this exactly backwards, and in our experience they frequently do.

The test has an important failure mode. Infarction in the territory of the anterior inferior cerebellar artery can involve the labyrinth itself, because the labyrinthine artery is usually a branch of it. In that situation the head impulse test is abnormal, the pattern appears peripheral, and the patient is having a brainstem stroke. This is not a rare curiosity; it is the specific scenario for which the third component of the battery exists.

Characterisation of nystagmus

A peripheral vestibular lesion produces a unidirectional, predominantly horizontal nystagmus with a torsional component, beating away from the affected side, increasing in amplitude on gaze in the direction of the fast phase in accordance with Alexander's law, and suppressed by visual fixation. Nystagmus that changes direction with the direction of gaze is central. So is nystagmus that is purely vertical, whether downbeat or upbeat, and so is nystagmus that is purely torsional. A peripheral lesion does not produce a pure vertical nystagmus, and the appearance of one should end the discussion.

Two practical points deserve emphasis. First, fixation suppresses peripheral nystagmus, which means it may be invisible unless fixation is removed — with Frenzel lenses, an ophthalmoscope viewing one fundus while the other eye is covered, or simply a blank sheet of paper held close to the face. A clinician who examines only under bright fixation may record "no nystagmus" in a patient who has plenty. Second, gaze should be tested at eccentric positions well short of the extremes, since a few beats of physiological end-point nystagmus at extreme lateral gaze are normal and are regularly overcalled as direction-changing.

Test of skew

Skew deviation is a vertical misalignment of the visual axes arising from disruption of the graviceptive pathways that carry otolithic information from the vestibular nuclei to the ocular motor nuclei. It is detected by alternate cover testing: the examiner covers and uncovers each eye in turn and watches for a vertical refixation movement. It forms part of the ocular tilt reaction, alongside head tilt and ocular counter-roll.

Skew is the least sensitive of the three components. In the original series it was present in only seventeen per cent of central cases, and its distribution was informative: four per cent of peripheral cases, four per cent of pure cerebellar strokes, and thirty per cent of those with brainstem involvement.[5] What makes it worth performing despite that low yield is its specificity and, more importantly, the company it keeps. In the original cohort, skew correctly identified lateral pontine stroke in two of the three cases in which an abnormal head impulse test had falsely suggested a peripheral lesion. It exists to catch precisely the failure described above.

The "plus" — bedside hearing assessment

Adding a bedside assessment of hearing, whether by finger rub or by tuning fork, converts HINTS into HINTS "plus". The rationale rests on vascular anatomy. The labyrinthine artery is an end artery without meaningful collateral supply, arising in most people from the anterior inferior cerebellar artery, so ischaemia in that territory can deafen the ear and infarct the brainstem in the same event.[18] New unilateral hearing loss accompanying an acute vestibular syndrome is therefore not evidence of labyrinthitis. It is a reason to think harder about the posterior circulation. This is a point on which otolaryngological and stroke practice have historically diverged, and it forms the subject of a companion review.

The trade-off is measurable. The Cochrane analysis found that adding hearing assessment preserved sensitivity but reduced specificity, which is what one would expect from broadening the range of findings that count as concerning for a central lesion.[19] In a setting where the cost of a false positive is an MRI and the cost of a false negative is a brainstem stroke, that trade is usually worth making.

Citation: Deshpande A, Khardenavis S, Godse G, et al. Bedside oculomotor examination in the acute vestibular syndrome: what hints can and cannot do for the otolaryngologist. *J Otolaryngol ENT.* 2026;18(2):66–72. DOI: 10.15406/joentr.2026.18.00590

Copyright: ©2026 Deshpande et al.

Note that the interpretation of the head impulse test is inverted relative to intuition. A battery in which any single component falls in the right-hand column is described throughout this review as HINTS concerning for a central lesion. The pattern is valid only in a patient who genuinely meets the definition of an acute vestibular syndrome.

The evidence, read carefully

The foundational study enrolled 101 patients with acute vestibular syndrome and at least one vascular risk factor at a single academic centre.[5] Twenty-five had peripheral causes and seventy-six central ones, of which sixty-nine were ischaemic infarcts, four were haemorrhages and three were something else. The presence of any one of a normal head impulse test, direction-changing gaze-evoked nystagmus, or skew deviation was one hundred per cent sensitive and ninety-six per cent specific for stroke. Early diffusion-weighted imaging, by contrast, was falsely negative in twelve per cent, all within forty-eight hours of onset.

These are remarkable numbers and they are frequently quoted without their context. Three features of the design constrain how far they travel. The population was deliberately enriched for risk, so the prevalence of stroke was seventy-five per cent — an order of magnitude higher than in an unselected dizzy population. Predictive values calculated in such a cohort do not transfer. The examinations were performed by neuro-otologists with a career's familiarity with these signs. And the comparison was against early MRI, not against MRI at seventy-two hours, which is a considerably harder target. A reported sensitivity of one hundred per cent in a sample of this size is also compatible, on any reasonable confidence interval, with a true sensitivity appreciably below that figure, and it should not be read as evidence that the examination never misses.

Subsequent work has both confirmed and complicated the picture. A systematic review found that bedside oculomotor signs consistently outperformed general neurological examination and imaging in the acute phase.[6] HINTS was shown to outperform the ABCD² score decisively,[20] which is worth stating plainly because ABCD² continues to be applied to dizzy patients in some units despite never having been designed or validated for that purpose. The Cochrane review published in 2023 pooled sixteen studies and 2,024 patients, of whom 569 (28.1%) had a central cause, and reported sensitivity of approximately ninety-four per cent and specificity of approximately eighty-seven per cent for the clinical examination.[19] In ten of those sixteen studies, however, the examiner was a neurologist or neuro-otologist. Only two studies used emergency physicians.

That distribution matters, because when the analysis is restricted to emergency physicians the conclusion changes. A meta-analysis by Ohle and colleagues found that HINTS performed in isolation by emergency physicians was not sufficiently accurate to exclude stroke.[21] A retrospective review from the same group reported similar findings in routine practice,[22] and a separate audit found that only eighteen per cent of patients presenting with acute vertigo had any documented HINTS examination at all.

There is a counterargument, and it is a reasonable one. Later systematic work focusing specifically on clinicians who had received structured education and training reported accuracy approaching that of specialists, suggesting that the earlier negative findings reflected the absence of training rather than any intrinsic limitation of the test.[23] We find this persuasive as far as it goes. It does, however, concede the central point: the published accuracy of HINTS is a property of the examiner as much as of the examination, and quoting the 2009 figures to justify discharging a patient examined by someone who has never been formally taught the technique is a misuse of the evidence(Table 2).

Table 2 Selected accuracy data.The apparent inconsistency between these studies largely dissolves once examiner training and case mix are taken into account

Study	Setting and examiner	Principal finding
Kattah et al.[5] (n = 101)	Single academic centre; neuro-otologists; 75% prevalence of central cause	The presence of any one component concerning for a central lesion was 100% sensitive and 96% specific for stroke; early diffusion-weighted MRI was falsely negative in 12%
Newman-Toker et al.[20] (n = 190)	Emergency department; trained examiners	HINTS substantially outperformed the ABCD² score for identifying stroke in acute continuous vertigo
Ohle et al.[21] (meta-analysis)	Pooled data restricted to emergency physicians	When performed by emergency physicians in isolation, HINTS was not sufficiently accurate to exclude stroke
Gottlieb et al.[19] (Cochrane; 16 studies, 2024 patients)	Mixed; a neurologist or neuro-otologist performed the examination in 10 of 16 studies	Pooled sensitivity approximately 94% and specificity approximately 87%; adding hearing assessment preserved sensitivity but reduced specificity; video assistance did not clearly improve either

Why performance degrades outside specialist hands

Several mechanisms operate at once, and they are worth separating because they have different remedies.

The head impulse test is technically demanding. The head movement must be of small amplitude, high acceleration and unpredictable timing; performed too slowly or too widely it fails to challenge the reflex and produces a spuriously normal result. Covert saccades, which occur during rather than after the head movement, are invisible to the naked eye and require video-oculography to detect. Cervical spondylosis, recent neck trauma and suspicion of arterial dissection all constrain how vigorously the manoeuvre can be performed, and in some patients it should not be performed at all.

The battery is also frequently interpreted as a gestalt rather than as a rule. HINTS is not a scoring system in which two reassuring components outvote one worrying component. A single component showing the central pattern renders the whole examination concerning for a central lesion, irrespective of the other two. Clinicians who are uncertain about one component and reassured by the other two will average them, and averaging is the wrong operation.

Then there is the selection error already described, which we would place first in order of importance. A great deal of what is recorded in case notes as a HINTS examination was performed on a patient who

Citation: Deshpande A, Khardenavis S, Godse G, et al. Bedside oculomotor examination in the acute vestibular syndrome: what hints can and cannot do for the otolaryngologist. *J Otolaryngol ENT.* 2026;18(2):66–72. DOI: 10.15406/joentr.2026.18.00590

Copyright: ©2026 Deshpande et al.

did not have an acute vestibular syndrome. In that population the test has no established operating characteristics whatsoever.

Finally, and least discussed, there is no feedback loop. A clinician who performs a head impulse test badly and reaches the wrong conclusion is rarely told. Skills that are never corrected do not improve with repetition; they simply become more confident.

The practical synthesis we would offer is asymmetric. For a clinician without formal training, HINTS concerning for a central lesion is a reliable prompt to investigate for stroke, and its positive likelihood ratio is high enough to act on; a result consistent with a peripheral vestibulopathy in the same hands carries little weight and should not be used to discharge anybody. For a trained and experienced examiner, a result consistent with a peripheral vestibulopathy meaningfully lowers the probability of a central cause and can reasonably inform disposition — but it does not exclude stroke, and it should not be treated as though it did. It remains one element of an assessment that must also take account of the vascular risk profile, the general neurological examination, gait and truncal stability, and the subsequent clinical course. Departments that adopt the examination should say explicitly, in writing, which of their staff have been trained to perform it and what weight a reassuring result is permitted to carry.

What imaging can and cannot contribute

Computed tomography of the head misses more than eighty per cent of acute ischaemic strokes in the first hours, and its performance in the posterior fossa is worse still because of beam-hardening artefact from the surrounding bone. Its legitimate role is the exclusion of haemorrhage and of a mass lesion, and it should be understood as answering that narrow question only. A normal CT in a dizzy patient carries no information about whether they have an infarct.

Diffusion-weighted MRI is far better, but it is not the arbiter it is often assumed to be. False negative rates of ten to twenty per cent in the first forty-eight hours are consistently reported, and the misses are concentrated exactly where they matter: in the posterior fossa, and in lesions under about ten millimetres.[5] This is the point at which the argument becomes uncomfortable, because a clinician may examine a patient competently, find oculomotor signs concerning for a central lesion, order the definitive scan, and receive a normal report.

A normal early scan in that situation reduces the probability of infarction but does not settle the question, and the clinical assessment should not simply be discarded in its favour. In patients with persistent clinical concern for a central lesion despite a negative early MRI, urgent neurological or stroke-team assessment should be considered, together with repeat MRI and/or vascular imaging according to the clinical context. The threshold for that escalation should fall as the pre-test probability rises — with advancing age, an accumulation of vascular risk factors, neck pain or a history suggesting dissection, preceding transient brainstem symptoms, truncal ataxia disproportionate to the vertigo, or any additional neurological sign. Where symptoms resolve rapidly and the wider assessment is unremarkable, observation with clear safety-netting and arranged follow-up may be reasonable. What should not happen is that a single normal early scan is allowed to overturn a competent examination without any further thought being given to the matter.

Instrumented approaches

Video head impulse testing quantifies vestibulo-ocular reflex gain and detects covert saccades that cannot be seen at the bedside, removing much of the subjectivity from the first component of the battery. Video-oculography systems that capture nystagmus and head impulse data together have been proposed as an "eye ECG" — a recordable, transmissible, reviewable trace that could be acquired by a nurse and interpreted remotely, in the same way that a twelve-lead tracing is today. The analogy is attractive and the underlying idea is sound.

The evidence is not yet where the enthusiasm is. The Cochrane analysis found that video assistance did not clearly improve sensitivity or specificity over the clinical examination, though it noted that the number of studies was small.[19] Cost, calibration, cleaning between patients and the need for someone to interpret the output remain real obstacles in a district general hospital, and the argument that instrumentation will solve a training problem deserves scepticism: an uninterpreted trace is not better than an unperformed test. Machine learning applied to oculomotor recordings is promising and, at the time of writing, unvalidated in any population resembling routine practice.

Implications for otolaryngology practice

Before any of what follows, the categorisation step must be completed. The recommendations in this section apply to patients who meet the definition of an acute vestibular syndrome — continuous vertigo lasting days to weeks with nystagmus, head-motion intolerance and gait unsteadiness. A patient with brief positional attacks, or with episodes that have already resolved, requires a different examination and a different differential, and the accuracy figures discussed above do not apply to them.

Within that group, otolaryngologists meet these patients in three distinct situations, and the reasoning differs in each.

The first is acute referral, where the question is whether the patient needs imaging and admission. Here the examination should be performed and documented component by component, with the presence or absence of hearing loss recorded explicitly, because a later reviewer cannot reconstruct what was found from a note that says "HINTS peripheral". The record should also state who performed the examination and what training they have had.

The second, and the one where otolaryngology has the greatest leverage, is the delayed clinic presentation. A patient labelled with labyrinthitis a week or two earlier who remains symptomatic, or who has direction-changing nystagmus, or who has new hearing loss, or who cannot stand unsupported, warrants MRI rather than another course of vestibular sedatives. The passage of time is not reassuring in this context; a small cerebellar infarct does not announce itself, and the patient who has been unwell for a fortnight has had a fortnight without secondary prevention.

The third is the patient referred for vestibular rehabilitation whose deficit does not fit the label. A pattern of vestibular loss that does not correspond to a single nerve territory, or that fails to compensate on the expected timescale, is worth reconsidering from first principles.

Two ancillary points are worth making. Gait assessment is the most underused element of the whole evaluation. Inability to sit or stand without support, disproportionate to the severity of the vertigo, is central until proven otherwise, and it takes ten seconds to elicit. Truncal ataxia has been proposed as a cardinal sign alongside HINTS for exactly this reason.[24] And prolonged prescription of vestibular suppressants impairs central compensation and obscures the signs on which any later assessment depends; three days is generally sufficient, and beyond that the drug becomes part of the problem.

Citation: Deshpande A, Khardenavis S, Godse G, et al. Bedside oculomotor examination in the acute vestibular syndrome: what hints can and cannot do for the otolaryngologist. *J Otolaryngol ENT.* 2026;18(2):66–72. DOI: 10.15406/joentr.2026.18.00590

Copyright: ©2026 Deshpande et al.

What the field still needs

Four questions stand out. First, prospective accuracy studies in unselected populations, with the training received by examiners prespecified and reported rather than assumed — the single largest source of heterogeneity in the existing literature. Second, a dose-response relationship for education: how much teaching, of what kind, produces what accuracy, and how quickly does it decay without practice. Third, an honest cost-effectiveness analysis of video-oculography in non-academic hospitals, where most of these patients are actually seen. Fourth, whether formal audiometric assessment should be incorporated into the acute pathway rather than left to bedside estimation, given the anatomical argument linking cochlear and brainstem ischaemia.

A fifth question follows from the position taken in this review: what happens to patients in whom a trained examiner finds a pattern consistent with a peripheral vestibulopathy and who are discharged on that basis. The rate of subsequent cerebrovascular events in that specific group has not been established prospectively, and until it is, the true rule-out value of the examination in ordinary practice remains an estimate.

Standardised reporting would help all of these. A minimum dataset specifying the syndrome category, each component result, hearing status, gait, examiner grade and training, and final adjudicated diagnosis would allow the accumulated experience of ordinary departments to become evidence rather than anecdote.

Conclusion

HINTS is a good examination that has been asked to carry an unreasonable load. Applied to a patient who genuinely has an acute vestibular syndrome, by an examiner trained to perform it, it outperforms early diffusion-weighted imaging and can meaningfully direct management. Applied indiscriminately, and in particular to patients whose dizziness is episodic or triggered rather than continuous, it generates false reassurance in exactly the patients who can least afford it. Establishing that the syndrome is present is not a preliminary to the examination; it is the first and most important part of it.

Two cautions follow. A result consistent with a peripheral vestibulopathy lowers the probability of stroke but does not exclude it, whoever performed the examination, and it should be weighed alongside the vascular risk profile, the general neurological assessment, gait, and the clinical course rather than treated as a discharge criterion in its own right. And a negative early MRI does not settle the question either; where clinical concern persists, neurological or stroke-team assessment and repeat or vascular imaging should be considered according to the clinical context.

For otolaryngologists, the most valuable contribution may lie less in the acute department than in the clinic a week later, where a diagnosis of labyrinthitis is presented as settled and the examination that would unsettle it takes under five minutes. Asking whether the label is right is not second-guessing a colleague. It is the only remaining opportunity to change the outcome.

Declarations

Funding

Authors: no funding was received by any of the authors.

Ethical approval

Not applicable. This is a narrative review of published literature and involved no patients, patient-identifiable data or animal subjects.

Author contributions

AD: conceptualisation, literature search, writing — original draft, writing — review and editing. SK, VK, GG

Acknowledgements

None

Conflicts of interest

The authors declare that there are no conflicts of interest.

References

1. Newman-Toker DE, Hsieh YH, Camargo CA Jr, et al. Spectrum of dizziness visits to US emergency departments: cross-sectional analysis from a nationally representative sample. *Mayo Clin Proc.* 2008;83(7):765–775.
2. Bisdorff A, Bosser G, Gueguen R, et al. The epidemiology of vertigo, dizziness, and unsteadiness and its links to co-morbidities. *Front Neurol.* 2013;4:29.
3. Kerber KA, Meurer WJ, West BT, et al. Dizziness presentations in U.S. emergency departments, 1995–2004. *Acad Emerg Med.* 2008;15(8):744–750.
4. Hotson JR, Baloh RW. Acute vestibular syndrome. *N Engl J Med.* 1998;339(10):680–685.
5. Kattah JC, Talkad AV, Wang DZ, et al. HINTS to diagnose stroke in the acute vestibular syndrome: three-step bedside oculomotor examination more sensitive than early MRI diffusion-weighted imaging. *Stroke.* 2009;40(11):3504–3510.
6. Tarnutzer AA, Berkowitz AL, Robinson KA, et al. Does my dizzy patient have a stroke? A systematic review of bedside diagnosis in acute vestibular syndrome. *CMAJ.* 2011;183(9):E571–E592.
7. Gurley KL, Edlow JA. Avoiding misdiagnosis in patients with posterior circulation ischemia: a narrative review. *Acad Emerg Med.* 2019;26(11):1273–1284.
8. Tarnutzer AA, Lee SH, Robinson KA, et al. ED misdiagnosis of cerebrovascular events in the era of modern neuroimaging: a meta-analysis. *Neurology.* 2017;88(15):1468–1477.
9. Newman-Toker DE, Moy E, Valente E, et al. Missed diagnosis of stroke in the emergency department: a cross-sectional analysis of a large population-based sample. *Diagnosis.* 2014;1(2):155–166.
10. Arch AE, Weisman DC, Coca S, et al. Missed ischemic stroke diagnosis in the emergency department by emergency medicine and neurology services. *Stroke.* 2016;47(3):668–673.
11. Drachman DA, Hart CW. An approach to the dizzy patient. *Neurology.* 1972;22(4):323–334.
12. Newman-Toker DE, Cannon LM, Stofferahn ME, et al. Imprecision in patient reports of dizziness symptom quality: a cross-sectional study conducted in an acute care setting. *Mayo Clin Proc.* 2007;82(11):1329–1340.
13. Edlow JA. Diagnosing dizziness: we are teaching the wrong paradigm! *Acad Emerg Med.* 2013;20(10):1064–1066.
14. Newman-Toker DE, Edlow JA. TiTrATE: a novel, evidence-based approach to diagnosing acute dizziness and vertigo. *Neurol Clin.* 2015;33(3):577–599.

15. Edlow JA, Gurley KL, Newman-Toker DE. A new diagnostic approach to the adult patient with acute dizziness. *J Emerg Med.* 2018;54(4):469–483.
16. Halmagyi GM, Curthoys IS. A clinical sign of canal paresis. *Arch Neurol*. 1988;45(7):737–739.
17. Newman-Toker DE, Kattah JC, Alvernia JE, et al. Normal head impulse test differentiates acute cerebellar strokes from vestibular neuritis. *Neurology.* 2008;70(24 Pt 2):2378–2385.
18. Lee H, Sohn SI, Jung DK, et al. Sudden deafness and anterior inferior cerebellar artery infarction. *Stroke.* 2002;33(12):2807–2812.
19. Gottlieb M, Peksa GD, Carlson JN. Head impulse, nystagmus, and test of skew examination for diagnosing central causes of acute vestibular syndrome. *Cochrane Database Syst Rev.* 2023;11(11):CD015089.
20. Newman-Toker DE, Kerber KA, Hsieh YH, et al. HINTS outperforms ABCD2 to screen for stroke in acute continuous vertigo and dizziness. *Acad Emerg Med.* 2013;20(10):986–996.
21. Ohle R, Montpellier RA, Marchadier V, et al. Can emergency physicians accurately rule out a central cause of vertigo using the HINTS examination? A systematic review and meta-analysis. *Acad Emerg Med.* 2020;27(9):887–896.
22. Dmitriew C, Regis A, Bodunde O, et al. Diagnostic accuracy of the HINTS exam in an emergency department: a retrospective chart review. *Acad Emerg Med.* 2021;28(4):387–393.
23. Aldridge S, Krishnan K. Using HINTS in acute vestibular syndrome: a practical guide for the acute care physician. *J R Coll Physicians Edinb.* 2025;55(2):139–145.
24. Carmona S, Martinez C, Zalazar G, et al. The diagnostic accuracy of truncal ataxia and HINTS as cardinal signs for acute vestibular syndrome. *Front Neurol*. 2016;7:125.
25. Kerber KA, Brown DL, Lisabeth LD, et al. Stroke among patients with dizziness, vertigo, and imbalance in the emergency department: a population-based study. *Stroke*. 2006;37(10):2484–2487.
26. Lee H, Sohn SI, Cho YW, et al. Cerebellar infarction presenting isolated vertigo: frequency and vascular topographical patterns. *Neurology.* 2006;67(7):1178–1183.
27. Cnyrim CD, Newman-Toker D, Karch C, et al. Bedside differentiation of vestibular neuritis from central "vestibular pseudoneuritis". *J Neurol Neurosurg Psychiatry*. 2008;79(4):458–460.
28. Brodsky MC, Donahue SP, Vaphiades M, et al. Skew deviation revisited. *Surv Ophthalmol*. 2006;51(2):105–128.
29. Saber Tehrani AS, Kattah JC, Kerber KA, et al. Diagnosing stroke in acute dizziness and vertigo: pitfalls and pearls. *Stroke.* 2018;49(3):788–795.
30. Newman-Toker DE. Missed stroke in acute vertigo and dizziness: it is time for action, not debate. *Ann Neurol.* 2016;79(1):27–31.
31. Paul NLM, Simoni M, Rothwell PM. Transient isolated brainstem symptoms preceding posterior circulation stroke: a population-based study. *Lancet Neurol.* 2013;12(1):65–71.
32. Kim JS, Lee H. Inner ear dysfunction due to vertebrobasilar ischemic stroke. *Semin Neurol.* 2009;29(5):534–540.
33. Kerber KA, Meurer WJ, Brown DL, et al. Stroke risk stratification in acute dizziness presentations: a prospective imaging-based study. *Neurology.* 2015;85(21):1869–1878.
34. Krishnan K, Bassilious K, Eriksen E, et al. Posterior circulation stroke diagnosis using HINTS in patients presenting with acute vestibular syndrome: a systematic review. *Eur Stroke J*. 2019;4(3):233–239.
35. Lee H. Neuro-otological aspects of cerebellar stroke syndrome. *J Clin Neurol.* 2009;5(2):65–73.
36. Nakatsuka M, Molloy EE. The HINTS examination and STANDING algorithm in acute vestibular syndrome: a systematic review and meta-analysis involving frontline point-of-care emergency physicians. *PLoS One.* 2022;17(5):e0266252.

Citation: Deshpande A, Khardenavis S, Godse G, et al. Bedside oculomotor examination in the acute vestibular syndrome: what hints can and cannot do for the otolaryngologist. *J Otolaryngol ENT.* 2026;18(2):66–72. DOI: 10.15406/joentr.2026.18.00590