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Usefulness of Nystagmus Patterns in Distinguishing Peripheral From Central Acute Vestibular Syndromes at the Bedside: A Critical Review

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Vertigo and dizziness are amongst the most frequent presenting symptoms in the emergency room, accounting for up to 4% of all emergency consultations. The broadness of their differential diagnosis and the often transient nature of these symptoms pose a significant challenge to the treating physician. Combining various subtle oculomotor signs at the bedside has been very successful in distinguishing peripheral from central causes in acutely dizzy patients meeting diagnostic criteria for the acute vestibular syndrome (i.e., acute and prolonged vertigo or dizziness accompanied by nausea or vomiting, gait imbalance, motion intolerance, and [not mandatory] nystagmus). While the diagnostic accuracy of the HINTS (Head-Impulse—Nystagmus—Test-of-Skew) algorithm has been studied extensively, less is known about the value of various nystagmus patterns seen at the bedside in patients with an acute vestibular syndrome. Here we review both spontaneous and triggered presenting nystagmus patterns and discuss their impacts and limitations, including primary-gaze horizontal, vertical, and torsional nystagmus, nystagmus during eccentric gaze, and nystagmus triggered by stimuli such as head-shaking, hyperventilation, positional testing, vibration, and the Valsalva maneuver. We conclude that the usefulness of nystagmus patterns in discriminating peripheral and central causes strongly depends on the pattern seen and the type of testing performed, being highly predictive of a central cause for torsional and vertical spontaneous nystagmus, downbeat, or apogeotropic horizontal and treatment-refractory positional nystagmus. The predictive value for central causes was moderate only for vertical nystagmus after horizontal head-shaking ("perverted" head-shaking nystagmus) since it can also occur in peripheral cases, while the predictive value was low for vibration-induced nystagmus and Valsalva-induced nystagmus.

Keywords acute vestibular syndrome; dizziness; vertigo; diagnostic accuracy; nystagmus, pathologic.

INTRODUCTION

Vertigo and dizziness are amongst the most common presenting symptoms in the emergency room (ER) as well as in general practice, accounting for 2%–4% of all emergency consultations.[1-4] The differential diagnosis of acute and episodic vertigo is extremely broad and spans numerous specialties, including neurological, psychiatric, internal medicine, and orthopedic conditions. As a result, clinicians from a wide range of specialties are confronted with this symptom. Diseases of the vestibular system (e.g., peripheral vestibular vertigo) represent only about one-third of all causes of vertigo or dizziness. A clear classification of dizziness complaints is made difficult by no single cause accounting for more than 5% of all dizziness diagnoses.[2] Typically 10%–15% of dizzy patients have a serious underlying medical condition, with the rate increasing with age.[2] Initial assessments of dizzy patients must therefore focus on identifying those patients who urgently require

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further diagnostic and therapeutic measures, such as stroke assessments in a stroke unit or cardiac assessments in the case of cardiac arrhythmia.[5]

Obvious focal neurological symptoms may be missing in up to two-thirds of patients presenting with acute prolonged vertigo or dizziness accompanied by nausea or vomiting, gait imbalance, motion intolerance, and (not mandatory) nystagmus (i.e., meeting diagnostic criteria of an acute vestibular syndrome [AVS]).[6] Furthermore, early magnetic resonance imaging (MRI) (i.e., performed within the first 24−48 h) including diffusion-weighted imaging may produce false-negative results in up to 20% of cases.[7] Luckily, excellent diagnostic accuracy can be achieved at the bedside by assessing various oculomotor signs including eccentric gaze-holding (assessing for gaze-evoked nystagmus [GEN]), the alternating cover test (assessing for skew deviation), and the head-impulse test (HIT; assessing the integrity of the angular vestibulo-ocular reflex [aVOR]) (Supplementary Video 1 in the online-only Data Supplement).[7] This simplified combination of tests has been referred to as the HINTS (Head-Impulse—Nystagmus—Test-of-Skew) algorithm[8] and may be further extended by adding an assessment for new-onset unilateral hearing loss (referred to as HINTS-plus[9]). While assessing spontaneous nystagmus (SN) is not part of HINTS, including this may further improve the diagnostic accuracy when combined with HINTS or similar algorithms such as the STANDING algorithm.[10] Furthermore, reliable HINTS findings require moderate training, and so its diagnostic accuracy will be substantially lower when applied by an inexperienced emergency physician or neurology resident.[7] Alternatively, assessing the gait using a grading system[11] or nystagmus patterns during primary gaze or when triggered by specific conditions may be a more-feasible and thus valuable approach.

Horizontal or horizontal-torsional SN beating away from the affected ear is a hallmark sign in patients with acute unilateral vestibulopathy (AUVP)[12] and may also be observed in patients with vertebrobasilar stroke.[13] Furthermore, it has been proposed that the SN pattern may be useful in distinguishing between peripheral and central causes, with torsional and vertical SN pointing to the latter cause[6] and various triggered nystagmus patterns having been linked to either peripheral or central causes.

In this critical review we discuss the value of the bedside assessment of various nystagmus patterns in the diagnostic workup of acutely dizzy patients. We summarize both the current evidence and also emphasize the limitations and pitfalls. A principal distinction is made between primary-gaze nystagmus (i.e., SN), eccentric-gaze nystagmus, and triggered nystagmus patterns.

OVERALL FLOW OF BEDSIDE EXAMINATIONS IN AVS

After taking the patient's history, a standardized neurotological examination is required to assess a patient with an AVS. The examination begins with evaluating the head and ocular alignment using the alternate cover test. Next, any SN is characterized during visual fixation and visual blockage as well as by analyzing the nystagmus components. The patient is then instructed to follow a visual target placed eccentrically, both horizontally and vertically, to detect any GEN and any restriction in ocular motility. The bedside HIT is essential in every AVS patient. Following this, saccades and pursuit eye movements are assessed. The evaluation continues with an assessment of truncal stability and a general neurological examination. Positional testing and other triggering maneuvers can be reserved for last, especially if the patient's cooperation is uncertain. Below we provide a focused review of the clinical implications of SN and triggered nystagmus.

SN PATTERNS

Analyzing the pattern of SN can aid in differentiating AVS of central origin (cAVS) from AVS of peripheral origin (pAVS). A recent systematic review and meta-analysis assessed the diagnostic accuracy of SN patterns in 1,599 patients with either pAVS or cAVS.[14] The key findings of that study included the observations that 1) SN was observed in almost all pAVS patients but in fewer than half of cAVS patients (97.6% vs. 43.6%), 2) the SN pattern varied with the underlying cause, and 3) the directionality of any horizontal SN was predictive of the lesioned side only in pAVS cases. Specifically, while horizontal or horizontal-torsional SN was significantly more common in pAVS than cAVS patients (94.8% vs. 43.4%, $p<0.001$), isolated torsional, isolated torsional-vertical, and/or vertical SN patterns were more prevalent in cAVS than pAVS patients (15.1% vs. 2.6%, $p<0.001$) (Supplementary Video 2 in the online-only Data Supplement). When present, such patterns were highly predictive of a central cause (specificity=97.7%, 95% confidence interval [CI]=95.1%−100.0%), but such patterns of high diagnostic value were rarely seen, resulting in a low sensitivity of only 19.1% (95% CI=10.5%−27.7%).[14] It is particularly interesting that a predominance of either ipsilesional or contralesional horizontal-torsional SN was found for certain lesion locations (e.g., medulla oblongata) and certain vascular patterns (strokes in the peripheral inferior cerebellar artery [PICA] and anterior inferior cerebellar artery [AICA] vascular territory). This review identified inferior-branch AUVP (also called inferior vestibular neuritis)—resulting in a combined torsional-downbeat (con-

tralesional) SN pattern—as the most frequent cause of false-positive findings.[15,16] When combined with HINTS-plus testing, this condition is likely to be classified as central as well due to normal horizontal HIT findings. Performing a quantitative (usually video-based) HIT in all six canals is critical in such cases,[17,18] since it allows canal-specific testing of the aVOR[19] and thus is able to demonstrate isolated impairment of the posterior semicircular canal, supporting the diagnosis of pAVS due to inferior-branch AUVP (Supplementary Video 3 in the online-only Data Supplement).[15] Importantly, the absence of SN in AVS patients was highly predictive of a central cause with a specificity of 97.9% (95% CI=96.9%–98.9%) in this systematic review,[14] and should therefore be considered a central sign in this clinical situation (Tables 1 and 2).[20]

In cAVS cases, the nystagmus beating pattern may be associated with specific lesion locations. For example, a torsional component of SN was observed more frequently in AICA than PICA strokes and in lateral than medial medullary lesions.[14] Dissociated torsional-vertical nystagmus can be observed in lesions involving the medial longitudinal fasciculus, due to disruption of the vertical canal pathway (Supplementary Video 4 in the online-only Data Supplement). Predominant upbeat nystagmus (UBN) was anecdotally reported in lesions involving the medial medulla, which is probably due to damage to the nucleus intercalatus unilaterally or bilaterally (Supplementary Video 2 in the online-only Data Supplement).[21]

The diagnostic value of SN fixation suppression in distinguishing peripheral from central lesions has been controversial. While decreased or even abolished SN by fixation and enhanced SN by removal of visual fixation is considered an important finding in peripheral-vestibular causes such as AUVP,[12] the lack of an impact of fixation on the SN intensity has been described in neurology textbooks as being associated with central causes.[22] However, a previous systematic review of the literature has emphasized that evidence is lacking for the accuracy of such a clinical suppression sign.[6] It is particularly noteworthy that quantitative data now provide a more-detailed view. Complete nystagmus fixation suppression may be observed in both patients with AUVP and those with vestibular strokes at similar frequencies (40% vs. 62.5%),[23] but the magnitude of nystagmus reduction as measured quantitatively may be helpful in distinguishing peripheral from central causes. The conventional wisdom of a cutoff value of 0.5 for the fixation index originated from earlier measurements of caloric nystagmus with and without visual fixation in monkeys.[24] Patients with a reduction in the nystagmus velocity of less than 2°/s were more likely to have cAVS than pAVS (sensitivity=62.2%, specificity=84.8%) in one study.[23] Another study found that the reduction in the nystagmus slow-phase velocity (SPV) during visual fixation was smaller in patients with AUVP than in those with posterior circulation stroke (5.9±4.7°/s vs. 0.7±2.3°/s [mean± standard deviation], $p<0.001$).[20] Other researchers reported that a ratio >0.42 between the SPV during fixation and that in darkness could be considered as a failure of fixation suppression, which was found in 41% of cerebellar stroke cases.[25]

The modulation of SN by fixational varies substantially in cAVS and may show patterns other than failure of fixational suppression. For example, the direction of SN can reverse depending on the presence of visual fixation (Supplementary Video 5 in the online-only Data Supplement). Likewise, SN can emerge only during visual fixation nystagmus and be nullified in darkness (i.e., fixation nystagmus) when lesions affect the cerebellar circuits[26] that are involved in visual–vestibular interactions.[27]

The effect of visual fixation also seems to depend on the SN plane. Upon removal of visual fixation, an increase in the intensity of horizontal-torsional SN was noted in stroke patients with lateral or dorsolateral medullary lesions.[28] In another study of dorsolateral medullary stroke, the horizontal and vertical components of SN were found to be markedly suppressed by visual fixation, with less suppression of the torsional component.[29] A minor impact of visual fixation on torsional SN was also reported in patients with mesencephalic stroke and predominantly torsional SN, with no increase in SN in darkness or with Frenzel goggles.[30]

Thus, at the bedside, the impact of assessing SN fixation suppression is limited as a qualitative assessment may not be sensitive enough to depict differences in the magnitude of response to fixation and fixation removal, and quantitative assessments may not be readily available. Importantly, the presence of fixation suppression does not rule out cAVS.[23]

GEN PATTERNS

Eccentric gaze-holding is controlled by a brainstem neural velocity-to-position integrator that is thought to be inherently leaky and hence requires cerebellar input to calibrate its output precisely in proportion to the eye position.[31] Impairment of this network will reduce the ability to main a steady gaze in an eccentric horizontal or vertical position. Thus, a centripetal drift of the eyes may be observed, and a central compensatory outward-directed saccade is initiated to compensate for this type of unwanted positional change of the eyes. By definition, GEN changes with the current eye position, always beating toward the direction of gaze (Supplementary Video 1 in the online-only Data Supplement). Horizontal GEN is a central sign with an excellent specificity but only a low-to-moderate sensitivity for distinguishing

Table 1. Discrimination value of nystagmus patterns seen in acutely dizzy patients

	Discrimination value (peripheral vs. central) if observed	Observations	Limitations
SN			
Horizontal or horizontal-torsional nystagmus	None	Seen in both pAVS and cAVS.[14] Absence of SN favors cAVS	While mandatory for pAVS by definition, SN is seen only in about 50% of cAVS cases
Purely torsional, torsional-vertical nystagmus	High	Excellent specificity for central causes, but low sensitivity[14]	Torsional-downbeat nystagmus may also be seen in inferior-branch AUVP[15]
Purely vertical, horizontal-vertical nystagmus	High	Acute DBN is evidence for drug intoxication,[89] paraneoplastic syndromes,[90] or WE[91]	Might not be related to the acute symptoms, since also seen in degenerative cerebellar ataxia
GEN			
Horizontal GEN	High	Excellent specificity for central causes, but low-to-moderate sensitivity[7]	Bedside application may be challenging, gaze should be held at an eccentricity of 30° or less for at least 5 s.[92] Might not be related to the acute symptoms, since they are also seen in degenerative cerebellar ataxia
Vertical GEN	Presumably high		Inadequate data, with no diagnostic-accuracy studies being performed. Might not be related to the acute symptoms, since they are also seen in hereditary cerebellar ataxia
Triggered nystagmus			
HSN			
Horizontal nystagmus after horizontal head-shaking	Moderate	If there is early and strong direction reversal or if the nystagmus after head-shaking goes in the opposite direction to SN, this suggests a central cause[29]	
Vertical nystagmus after horizontal head-shaking	Moderate	Perverted HSN is found more often in central than peripheral vestibular disorders,[75] and pure vertical HSN in AVS is predictive of cAVS[74]	Perverted HSN may be seen in up to 50% of peripheral cases as well,[74] reducing its diagnostic accuracy
PN			
DBN during the DHM	High	Positional DBN during the DHM is strongly predictive of a central cause	Rarely this pattern can be seen in anterior-canal BPPV, but then should respond to treatment by the Yacovino maneuver[47]
Apogeotropic-horizontal nystagmus in the SRT	Moderate	Lack of treatment response for lateral-canal BPPV is suggestive for central causes.[60] Low threshold for brain MRI including DWI recommended	High risk of misdiagnosis, since seen in both peripheral and central causes
Hyperventilation-induced nystagmus	None	Triggered by discharges along the nerve or root zone with demyelination, may be peripheral or central	Diagnostic-accuracy studies on hyperventilation-induced nystagmus in AVS patients are lacking
Valsalva-induced nystagmus	Low	Associated with both peripheral (SSCDS) and central (Arnold-Chiari malformation) causes. Additional findings such as Tullio phenomenon may favor a peripheral cause (SSCDS)[82]	Diagnostic-accuracy studies on Valsalva-induced nystagmus in AVS patients are lacking
VIN	Low	VIN is rarely seen in central vestibular disorders[77]	Diagnostic-accuracy studies on VIN in AVS patients are lacking

AUVP, acute unilateral vestibulopathy; AVS, acute vestibular syndrome; BPPV, benign paroxysmal positional nystagmus; cAVS, central AVS; DBN, downbeat nystagmus; DHM, Dix-Hallpike maneuver; DWI, diffusion-weighted imaging; HSN, head-shaking nystagmus; MRI, magnetic resonance imaging; pAVS, peripheral AVS; PN, positional nystagmus; SN, spontaneous nystagmus; SRT, supine roll test; SSCDS, superior semicircular canal dehiscence syndrome; VIN, vibration-induced nystagmus; WE, Wernicke encephalopathy.

Table 2. Etiologies of various nystagmus patterns

	Peripheral	Central	Observations
SN			
Horizontal or horizontal-torsional nystagmus	AUVP, MD, vestibular paroxysmia	Vertebrobasilar stroke or hemorrhage, drug intoxication (e.g., AED, neuroleptics), WE, epileptic seizure, VM	Broad differential diagnosis, observed less frequently in central causes
DBN	Inferior-branch AUVP (if combined with down beat-torsional nystagmus)	Drug intoxication (antiepileptics, lithium), WE, paraneoplastic syndromes	Rarely seen in AVS due to vertebrobasilar stroke
Upbeat nystagmus	None	WE, vertebrobasilar stroke or hemorrhage	Usually not suppressed by fixation
Torsional nystagmus	None	Vertebrobasilar stroke or hemorrhage	Usually not suppressed by fixation
GEN			
Horizontal GEN	None	Vertebrobasilar stroke or hemorrhage, WE, drug intoxication, paraneoplastic syndromes	
Triggered nystagmus			
HSN	AUVP, MD, BPPV	Vertebrobasilar stroke (nodulus, flocculus, lateral medulla), VM, paraneoplastic syndromes	
PN	BPPV, vestibular tone imbalance after AUVP	CPA tumor, acute demyelinating lesion, VM, vertebrobasilar stroke, autoimmune cerebellar syndromes	Distinguishing between peripheral and central causes depends on the PN pattern seen (being most difficult for apogeotropic horizontal PN)
VIN	SSCDS, vestibular tone imbalance after AUVP	Paraneoplastic syndromes	
Hyperventilation-induced nystagmus	Vestibular paroxysmia	Demyelinating multiple sclerosis lesion	
Valsalva-induced nystagmus	SSCDS, perilymph fistula	Arnold-Chiari malformation	

AED, antiepileptic drugs; AUVP, acute unilateral vestibulopathy; AVS, acute vestibular syndrome; BPPV, benign paroxysmal positional nystagmus; CPA, cerebellopontine angle; DBN, downbeat nystagmus; GEN, gaze-evoked nystagmus; HSN, head-shaking nystagmus; MD, Ménière's disease; PN, positional nystagmus; SN, spontaneous nystagmus; SSCDS, superior semicircular canal dehiscence syndrome; VIN, vibration-induced nystagmus; VM, vestibular migraine; WE, Wernicke encephalopathy.

peripheral from central causes, as recently summarized in a systematic review and meta-analysis of the diagnostic accuracy of subtle oculomotor signs in patients with AVS (sensitivity=37.3%, 95% CI=27.2%–47.3%; specificity=98.2%, 95% CI=96.8%–99.7%) (Tables 1 and 2).[7] However, data on the diagnostic impact of vertical GEN are scarce.

Thus, when acutely dizzy patients are examined at the bedside, testing for GEN needs to be combined with testing for other, subtle oculomotor signs in order to achieve high diagnostic accuracy. Implementing the HINTS approach[8] produced an excellent diagnostic accuracy, with a sensitivity of 95.3% (95% CI=92.5%–98.1%) and a specificity of 92.6% (95% CI=88.6%–96.5%).[7] Evaluating eccentric gaze-holding is also part of the STANDING algorithm, but this is done only in patients who also present with SN.[10]

A potential pitfall when assessing horizontal eccentric gaze-holding at the bedside is asking the patient to look too far left or right, since GEN may be confused with the physiological drift that is observed at extreme angles (>40°), referred to as "end-point nystagmus," which is usually of low amplitude and frequency, and is not sustained.[32,33] Therefore, an angle of eccentricity of 30° or less is recommended when assessing eccentric gaze-holding, with the gaze maintained at the position for at least 5 s.[34] This also ensures that testing is performed within the binocular field of view. While a pathological GEN is sustained, its amplitude often reduces over time. This reflects a central compensatory phenomenon, which will result in subsequent rebound nystagmus when the eyes are brought back to the primary gaze. While rebound nystagmus is a hallmark of subacute and chronic cerebellar diseas-

es such as spinocerebellar ataxias and episodic ataxia type 2, it is assumed to be rare in cAVS. However, this has not been studied systematically, which might be due to the need to first establish the central compensatory mechanisms in order to enable rebound nystagmus.

CONVERGENCE-INDUCED NYSTAGMUS

The effect of convergence on SN has been reported only rarely in cAVS. For example, convergence can induce UBN, downbeat nystagmus (DBN), or pendular nystagmus in patients with multiple sclerosis and brainstem infarctions.[35,36] The modulation pattern may differ between pAVS and cAVS; horizontal nystagmus can be evoked in a patient with a vestibular nuclei lesion, while no such effect has been found in patients with AUVP.[37] Since the neural substrates as well as modulation patterns remain speculative, future research is warranted to determine the diagnostic yield of convergence-induced nystagmus in patients presenting with AVS.

TRIGGERED NYSTAGMUS PATTERNS

Various triggered nystagmus patterns may be assessed in the acutely dizzy patient to further support distinguishing between benign, self-limited peripheral causes, and dangerous, potentially life-threatening central causes (Tables 1 and 2). Triggering maneuvers may be challenging to perform in acutely dizzy patients, and are generally more feasible once nausea and vomiting have subsided. Nevertheless, triggered nystagmus should not be omitted in the standard neurotological evaluation since it provides invaluable insights into the integrity and symmetry of vestibular afferents, as well as helping to localize the lesion. Standardized testing for positional nystagmus (PN) is implemented in the STANDING algorithm for patients who do not present with SN.[10] PN testing was implemented mainly to identify benign paroxysmal positional nystagmus (BPPV) and thus to distinguish triggered episodic vestibular syndromes from spontaneous AVS cases. However, PN testing may also be useful for identifying central causes, as discussed further below. While not included in the current bedside algorithms for assessing acutely dizzy patients, testing for head-shaking nystagmus (HSN), vibration-induced nystagmus (VIN), and hyperventilation-induced nystagmus may be valuable in reducing the number of possible alternatives during the differential diagnosis of acutely dizzy patients.

Positional nystagmus

PN testing should be performed in all patients who report intermittent (episodic) vertigo and position-dependent vertigo (PV) or dizziness. However, it may also be combined with the HINTS or STANDING algorithm in assessments of AVS patients. Motion intolerance and the resulting increases in vertigo, dizziness, and nausea may restrict or even prohibit positional testing in AVS patients. Notably, it is important to include head tilting and extension maneuvers to identify pseudo-SN observed in horizontal canal cupulolithiasis or canalolithiasis.[38] Since the HIT produces mostly normal findings bilaterally in BPPV, misinterpretation of pseudo-SN as true SN could lead to misleading conclusions, such as a patient having central vestibulopathy.

In positional testing, a distinction must be made between different forms of triggered (position-dependent) nystagmus (i.e., PN) and vertigo (i.e., PV). If the nystagmus and symptoms last for less than 60 s, they are referred to as paroxysmal PV and nystagmus, while if they persist for longer they are referred to as persistent, and the nystagmus is then classified as persistent PN.[39]

PN was found in 10.5%–21% of patients evaluated for dizziness or balance disorders.[40] In two retrospective studies, 11%–12% of the patients with PN had a central cause,[41,42] which underlines the importance of a dedicated investigation and the search for structural causes. PN is also very common in vestibular migraine (VM). In a prospective study, up to 90% of patients with VM exhibited central position-dependent nystagmus (CPN) during attacks.[43] One study that considered all patients with cAVS together found that one in three patients had CPN.[44] Applying comprehensive investigations to a cohort of patients with a suspected diagnosis of cupulolithiasis of the lateral semicircular canal resulted in 11% being diagnosed with CPN.[45]

The classification of PV and PN includes searching for BPPV related both to the posterior semicircular canal (and the contralateral anterior semicircular canal) using the Dix-Hallpike maneuver (DHM) and to the lateral semicircular canal using the supine roll test (SRT).[46] These triggering maneuvers revealing nystagmus patterns that do not match the plane of the stimulated semicircular canal represents evidence against the presence of BPPV. For example, observing a purely vertical DBN instead of the torsional-geotropic nystagmus seen in posterior-canal BPPV during the DHM should prompt further diagnostic testing. This nystagmus pattern is suggestive of a central cause, but could also be confused with BPPV related to the contralateral anterior semicircular canal. However, anterior-canal BPPV is responsible for fewer than 5% of all cases[46] and may present with isolated DBN or in combination with torsional apogeotropic nystagmus.[47] Thus, the PN patterns seen when performing the DHM usually allow peripheral (BPPV) and central causes to be clearly dis-

tinguished, with a low risk of misdiagnosis. However, if the SRT reveals horizontal geotropic or apogeotropic (paroxysmal or persistent) PN, this may be due to lateral-canal BPPV (lcBPPV) as well as having a central cause (Table 1). While the apogeotropic variant of lcBPPV is significantly rarer than the geotropic variant, apogeotropic PN is the most common form of central PV. Thus, the risk of misdiagnosis is significantly higher in this condition.

Various peripheral and central causes can mimic the clinical picture of BPPV. The time course is decisive for the differential diagnosis. If there is a (sub)acute onset, the differential diagnosis differs significantly from cases in which PV has been present for weeks or months. In (sub)acute onset, inflammatory as well as neoplastic and ischemic or hemorrhagic lesions are the most-likely causes of central positional vertigo (CPV).[48-52] In addition, VM is considered the most common nonlesional cause of CPV, characteristically with a persistent geotropic or, more rarely, apogeotropic CPN.[43,53,54] However, paraneoplastic syndromes or intoxication with substances such as antiepileptic drugs can also lead to CPN.[55,56] Among patients presenting in the ER, VM can be considered in young females, particularly those younger than 60 years and those with a history of motion sensitivity.[57] In such cases the nystagmus intensity is usually significantly lower than in BPPV, and there is no clear asymmetry when examining both lateral semicircular canals.[43,53]

Lesional causes include circumscribed lesions in the cerebellar peduncle, the cerebellar nodule, and/or the uvula, the tonsils, or the brainstem (pontine and medullary regions).[49,50,58-60] In addition, tumors in the brainstem or cerebellum can manifest as purely position-dependent symptoms. Inflammatory lesions, such as in the context of multiple sclerosis, can also cause CPV.[48] Ménière's disease can manifest as position-dependent geotropic or apogeotropic nystagmus and dizziness during an attack.[53,61]

Head-shaking nystagmus

HSN allows that dynamic imbalance of vestibular function to be assessed at the bedside. With fixation removed (i.e., with the patient wearing Frenzel goggles), the head is rotated at 2–3 Hz in the horizontal or vertical plane for 10–20 s. Any induced nystagmus pattern is then assessed, still with fixation removed. For patients with unilateral or asymmetric peripheral vestibular dysfunction, nystagmus beating toward the less-affected or unaffected ear can be observed after horizontal head-shaking, which may be monophasic or biphasic (i.e., where the beating direction alternates over time).[62] Such post-HSN was explained by a central velocity-storage mechanism and asymmetric vestibular input following Ewald's second law, which states that at high head accelerations, excitatory vestibular inputs are more effective than inhibitory vestibular inputs.[29]

Thus, horizontally beating HSN may confirm an acute vestibular imbalance but may also help unmasking a centrally compensated unilateral (or asymmetric bilateral) loss of vestibular function at follow-up even when canal paresis induced by caloric irrigation has normalized.[63] However, the diagnostic value of HSN has been questioned, including since HSN may also be a physiological sign.[64] It is particularly noteworthy that horizontal nystagmus after horizontal head-shaking was also observed in 43% of patients with lcBPPV (apogeotropic variant), and so HSN does not necessarily indicate vestibular dysfunction.[65] Vertical head-shaking is less-well characterized, and may demonstrate minor horizontal nystagmus patterns in cases of unilateral vestibular loss only.[66]

HSN may also be observed in patients with cAVS, especially if lesions are located within the medulla or cerebellum. If horizontal head-shaking results in HSN in the vertical plane (i.e., cross-coupled [also referred to as "perverted"] HSN), this is considered predictive of a central lesion located in the vestibulocerebellum (i.e., flocculus or paraflocculus, nodulus, or ventral uvula) or, less frequently, in the brainstem involving the pathways for the vertical aVOR (e.g., the crossed ventral tegmental tract). Abnormal or asymmetric stimulation of the vertical aVOR pathway has been proposed for explaining such cross-coupling.[67,68] HSN may be observed in most patients with (hemi)nodular strokes, beating either horizontally or vertically.[69,70] HSN was found to be frequent (88%) in patients with lateral medullary stroke (sparing the rostral portion), and the horizontal component was always beating toward the lesioned side, and sometimes was unusually strong.[29] About half of patients demonstrated horizontal HSN after horizontal head-shaking, beating contralateral to the side of their SN. Profound fixation suppression and unusually strong HSN have been considered central (medullary) signs of HSN.[29] Unilaterally impaired nodulouvular inhibition of the velocity storage mechanism has been proposed.[71] HSN has been a frequent finding in AICA stroke presenting with acute audiovestibular loss, with various planes being affected including purely horizontal contralesional or downbeat or upbeat HSN after horizontal head-shaking.[72] Overall, 50% of patients demonstrated central-type HSN, with most being the perverted type, but also in the opposite direction to SN or toward the lesioned side as seen in the HIT. These patients have frequently demonstrated floccular lesions in brain MRI. It is particularly noteworthy that in one patient with isolated audiovestibular loss, central-type HSN was the only other bedside finding supporting a central cause.[72] Perverted HSN has also been reported in a patient with an acute-

onset paraneoplastic syndrome.[73]

However, perverted HSN (upbeat or downbeat) was also found in about half of patients with peripheral-vestibular lesions including AUVP and Ménière's disease in one study[74] and in 19.6% of patients with various peripheral-vestibular disorders in another study.[75] This included pure vertical HSN after horizontal head-shaking in about 5% of patients with Ménière's disease and in 4% of patients with VM, but in none of the AUVP patients.[74]

While the presence of pure vertical HSN after horizontal head-shaking seems predictive of a central cause in patients with AVS, allowing for a distinction from AUVP cases at the bedside, overlap exists for patients with episodic or chronic vestibular syndromes, which reduces the predictive value of perverted HSN.[74,75] Furthermore, HSN with horizontal-vertical components in AVS may be seen in both pAVS and cAVS, indicating the need to combine HSN with other subtle oculomotor signs in this setting as well.

Vibration-induced nystagmus

VIN can be considered a triggered type of nystagmus in which both the otolith organs and the semicircular canals are stimulated, and it indicates the presence of vestibular tone imbalance, such as in persistent unilateral vestibulopathy. VIN points toward the side with better excitability, and so usually to the healthy ear. Deciding upon an appropriate stimulation frequency and restrictions in the availability of suitable stimulators are important limitations. The strongest responses have been observed for stimulation at 40 Hz.[76] VIN is rarely seen in central vestibular disorders[77] other than paraneoplastic syndromes.[73] Its applicability in the acute ER setting may be restricted by the nonavailability of suitable vibrators. Moreover, VIN is primarily observed in episodic or chronic vestibular disorders, such as superior semicircular canal dehiscence syndrome (SSCDS), and is rarely observed in patients presenting with AVS.

Hyperventilation-induced nystagmus

Hyperventilation-induced nystagmus is a valuable indicator of latent vestibular asymmetry in both peripheral and central pathologies. It works via metabolic mechanisms that have an excitatory effect on nerve tissues[78] or (much less frequently) by changes in the cerebrospinal fluid pressure. This type of nystagmus is usually induced over 60–70 s with the subject in a sitting position. Eventually both paretic nystagmus and excitatory nystagmus may be observed. The presence of AUVP may enhance a paretic or excitatory pattern, leading to changes in SN in about 75% of cases. SN usually does not appear in cases with unilateral vestibular schwannoma, and so a paretic or excitatory nystagmus pattern may be elicited. At the bedside, the hyperventilation-induced nystagmus test may be valuable in distinguishing vestibular schwannoma from idiopathic sudden-onset unilateral hearing loss. However, hyperventilation may also lead to an inhibitory (downbeat-torsional) nystagmus pattern in patients with SSCDS.[78] Demyelinating disease is highly likely when hyperventilation inhibits a central type of SN such as DBN or UBN.[79] In patients with cerebellar disease, hyperventilation may induce or augment DBN.[80] Thus, by suppressing inhibition by the cerebellum, hyperventilation may unmask cerebellar loss of function.[78] It was particularly interesting that the diagnostic criteria for VM were met by half of 22 cases of an episodic vestibular syndrome with DBN that were enhanced or induced by hyperventilation.[81]

In summary, when evaluating acutely dizzy patients, hyperventilation may prove useful in the differential diagnosis of patients presenting with AVS showing vertical nystagmus, as well as among those presenting with sudden-onset unilateral hearing loss.

Valsalva-induced nystagmus

Testing for nystagmus induced by the Valsalva maneuver is more established in outpatients presenting with episodic vestibular syndromes than in acutely dizzy patients seen in the ER. This is mainly due to the differential diagnosis of Valsalva-induced nystagmus and vertigo being associated with either the third mobile window syndrome and Tullio phenomenon (i.e., sound-induced vertigo and/or nystagmus) in the case of SSCDS (presenting as vertical-torsional nystagmus),[82] perilymph fistula, or in the presence of craniocervical junction anomalies such as Arnold-Chiari malformation.[32] Due to a pathophysiological condition needing to be present for triggering nystagmus by applying the Valsalva maneuver (i.e., a third mobile window or an obstructive intracranial mass), it is unlikely that there will be a significant diagnostic yield in acutely dizzy patients. To our knowledge the value of the Valsalva maneuver has not been systematically studied in acutely dizzy patients presenting in the ER.

CHALLENGES AND FUTURE DIRECTIONS

While nonspecialists with a moderate amount of training can apply the HINTS-plus algorithm or the STANDING algorithm to achieve a high diagnostic accuracy in distinguishing peripheral from central causes in acutely dizzy patients in the ER,[7,83] the interpretation of nystagmus patterns at the bedside is less straightforward and has not been systematically validated. While it is mostly low sensitivity that restricts the usefulness of SN patterns and GEN, the usefulness of trig-

gered nystagmus patterns is also reduced by their moderate specificity. This is especially true for perverted HSN, which may also be observed in peripheral vestibular syndromes. Furthermore, the correct identification, classification, and interpretation of subtle nystagmus patterns requires considerable experience and skill.

Combining different bedside examination techniques and providing sufficient training to frontline healthcare providers in both applying and interpreting these tests may be an effective strategy for overcoming these limitations and increasing their application in daily clinical practice. Alternatively, implementing automated quantitative assessment algorithms[84] and telemedicine[85-88] for providing appropriate diagnoses may be another useful strategy. However, the lack of expertise in interpreting findings, infrastructure including suitable video-oculography devices, and technicians for data collection may impede attempts to implement such interventions.

CONCLUSIONS

Interpreting SN or triggered nystagmus patterns observed in acutely dizzy patients is less straightforward than for other subtle oculomotor bedside findings, such as testing for GEN, vertical divergence, the integrity of the aVOR, and new-onset unilateral hearing loss, which allow peripheral and central causes to be differentiated with high diagnostic accuracy (HINTS-plus: sensitivity=99.2%, 95% CI=96.1%−100.0%; specificity=97.0%, 95% CI=90.4%−99.5%[9]). While the specificity is excellent for predicting central causes in patients with (isolated) vertical and/or torsional SN and the absence of horizontal SN, such patterns are seen in only 15% of patients with cAVS, and the presence of horizontal or horizontal-torsional SN does not allow peripheral and central causes to be distinguished.[14] Likewise, the presence of visual fixation suppression does not rule out cAVS. While several HSN patterns have been considered to have central causes, such as vertical nystagmus after horizontal head-shaking (perverted nystagmus) or nystagmus whose direction reverses after horizontal head-shaking or beating toward the aVOR deficit, perverted HSN may also be observed in peripheral vestibular syndromes, which restricts its diagnostic accuracy. For PN, the performed maneuver and the pattern observed have significant impacts on their diagnostic accuracy. While specificity is high for DBN during the DHM, persistent apogeotropic horizontal nystagmus in the SRT may be of peripheral or central origin. The lack of a treatment response and MRI abnormalities favor a central cause. The diagnostic value of VIN, hyperventilation-induced nystagmus, and Valsalva-induced nystagmus data in the acutely dizzy patient is either preliminary only or completely lacking.

In conclusion, it is essential to perform an initial detailed characterization and documentation of the observed nystagmus pattern. Since most such patients present in the ER, it is of great importance to provide dedicated training to frontline healthcare providers including ER physicians and neurology or otolaryngology residents in describing and interpreting nystagmus patterns. Combining these approaches with other subtle oculomotor findings is mandatory.

Supplementary Video Legend

Video 1. HINTS (Head-Impulse—Nystagmus—Test-of-Skew) examination in a 68-year-old male with left anterior inferior cerebellar artery hemorrhage. The patient shows gaze-evoked nystagmus during eccentric gaze, which is more prominent during rightward gaze. The head-impulse test is positive bilaterally, but more conspicuous to the left. The patient shows right concomitant hypertropia in the alternate cover test, which is compatible with skew deviation to the left.
Video 2. Upbeat nystagmus (UBN) and bowtie nystagmus in a 61-year-old male with a medial medullary infarction. The patient shows UBN during primary gaze. Vigorous gaze-evoked nystagmus is seen during eccentric gaze. The UBN changes to bowtie nystagmus during upward gaze. The video was provided by Dr. Hong-Kyun Park.
Video 3. A 40-year-old female with inferior left vestibular neuritis. The bedside head-impulse test is positive for the left posterior canal. Video-oculography (SensoMotoric Instruments [SMI], Germany) shows downbeat nystagmus with a clockwise component with and without visual fixation. The video was modified from Kim and Kim[15] and provided by Dr. Ji-Soo Kim.
Video 4. A 62-year-old female with a left medial medullary infarction. The patient shows dissociated torsional upbeat nystagmus (UBN), showing predominant UBN in the right eye and counterclockwise torsional nystagmus in the left eye. The video was modified from Lee et al.,[93] and provided by Dr. Ji-Soo Kim.
Video 5. A 46-year-old female with cerebellitis due to neuro-Behçet's disease presenting with an acute vestibular syndrome. The patient shows paroxysmal alternating nystagmus with visual fixation, which changes to left-beating nystagmus when wearing video-Frenzel goggles. The video was modified from Park et al.[94]

Supplementary Materials

The online-only Data Supplement is available with this article at https://doi.org/10.3988/jcn.2025.0105.

Ethics Statement

The patients provided permission for publication and appearance in the videos.

Availability of Data and Material

Data sharing not applicable to this article as no datasets were generated or analyzed during the study.

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Conflicts of Interest

The authors have no potential conflicts of interest to disclose.

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REFERENCES

1. Kerber KA, Meurer WJ, West BT, Fendrick AM. Dizziness presentations in U.S. emergency departments, 1995-2004. *Acad Emerg Med* 2008;15:744-750.
2. Newman-Toker DE, Hsieh YH, Camargo CA Jr, Pelletier AJ, Butchy GT, Edlow JA. Spectrum of dizziness visits to US emergency departments: cross-sectional analysis from a nationally representative sample. *Mayo Clin Proc* 2008;83:765-775.
3. Goeldlin M, Gaschen J, Kammer C, Comolli L, Bernasconi CA, Spiegel R, et al. Frequency, aetiology, and impact of vestibular symptoms in the emergency department: a neglected red flag. *J Neurol* 2019;266:3076-3086.
4. Ljunggren M, Persson J, Salzer J. Dizziness and the acute vestibular syndrome at the emergency department: a population-based descriptive study. *Eur Neurol* 2018;79:5-12.
5. Edlow JA, Tarnutzer AA. Intravenous thrombolysis in patients with acute dizziness or imbalance and suspected ischemic stroke-systematic review. *J Neurol* 2025;272:91.
6. Tarnutzer AA, Berkowitz AL, Robinson KA, Hsieh YH, Newman-Toker DE. Does my dizzy patient have a stroke? A systematic review of bedside diagnosis in acute vestibular syndrome. *CMAJ* 2011;183:E571-E592.
7. Tarnutzer AA, Gold D, Wang Z, Robinson KA, Kattah JC, Mantokoudis G, et al. Impact of clinician training background and stroke location on bedside diagnostic test accuracy in the acute vestibular syndrome - a meta-analysis. *Ann Neurol* 2023;94:295-308.
8. Kattah JC, Talkad AV, Wang DZ, Hsieh YH, Newman-Toker DE. HINTS to diagnose stroke in the acute vestibular syndrome: three-step bedside oculomotor examination more sensitive than early MRI diffusion-weighted imaging. *Stroke* 2009;40:3504-3510.
9. Newman-Toker DE, Kerber KA, Hsieh YH, Pula JH, Omron R, Saber Tehrani AS, et al. HINTS outperforms ABCD2 to screen for stroke in acute continuous vertigo and dizziness. *Acad Emerg Med* 2013;20:986-996.
10. Vanni S, Nazerian P, Casati C, Moroni F, Risso M, Ottaviani M, et al. Can emergency physicians accurately and reliably assess acute vertigo in the emergency department? *Emerg Med Australas* 2015;27:126-131.
11. Martinez C, Wang Z, Zalazar G, Carmona S, Kattah J, Tarnutzer AA. Systematic review and meta-analysis of the diagnostic accuracy of a graded gait and truncal instability rating in acutely dizzy and ataxic patients. *Cerebellum* 2024;23:2244-2256.
12. Strupp M, Bisdorff A, Furman J, Hornibrook J, Jahn K, Maire R, et al. Acute unilateral vestibulopathy/vestibular neuritis: diagnostic criteria. *J Vestib Res* 2022;32:389-406.
13. Eggers SDZ, Bisdorff A, von Brevern M, Zee DS, Kim JS, Perez-Fernandez N, et al. Classification of vestibular signs and examination techniques: nystagmus and nystagmus-like movements. *J Vestib Res* 2019;29:57-87.
14. Wüthrich M, Wang Z, Martinez CM, Carmona S, Mantokoudis G, Tarnutzer AA. Systematic review and meta-analysis of the diagnostic accuracy of spontaneous nystagmus patterns in acute vestibular syndrome. *Front Neurol* 2023;14:1208902.
15. Kim JS, Kim HJ. Inferior vestibular neuritis. *J Neurol* 2012;259:1553-1560.
16. Lee JY, Park JS, Kim MB. Clinical characteristics of acute vestibular neuritis according to involvement site. *Otol Neurotol* 2019;40:797-805.
17. Nham B, Reid N, Bein K, Bradshaw AP, McGarvie LA, Argaet EC, et al. Capturing vertigo in the emergency room: three tools to double the rate of diagnosis. *J Neurol* 2022;269:294-306.
18. Carmona S, Grinstein G, Weinschelbaum R, Zalazar G. Topodiagnosis of the inner ear: illustrative clinical cases. *Ann Otolaryngol Rhinol* 2018;5:1201.
19. Macdougall HG, McGarvie LA, Halmagyi GM, Curthoys IS, Weber KP. The video head impulse test (vHIT) detects vertical semicircular canal dysfunction. *PLoS One* 2013;8:e61488.
20. Nham B, Akdal G, Young AS, Özçelik P, Tanrıverdizade T, Ala RT, et al. Capturing nystagmus in the emergency room: posterior circulation stroke versus acute vestibular neuritis. *J Neurol* 2023;270:632-641.
21. Jeong SH, Kim EK, Lee J, Choi KD, Kim JS. Patterns of dissociate torsional-vertical nystagmus in internuclear ophthalmoplegia. *Ann N Y Acad Sci* 2011;1233:271-278.
22. Baloh RW, Honrubia V, Kerber KA. *Baloh and Honrubia's clinical neurophysiology of the vestibular system*. 4th ed. New York: Oxford University Press, 2011.
23. Mantokoudis G, Wyss T, Zamaro E, Korda A, Wagner F, Sauter TC, et al. Stroke prediction based on the spontaneous nystagmus suppression test in dizzy patients: a diagnostic accuracy study. *Neurology* 2021;97:e42-e51.
24. Takemori S, Cohen B. Visual suppression of vestibular nystagmus in rhesus monkeys. *Brain Res* 1974;72:203-212.
25. Kim HA, Yi HA, Lee H. Failure of fixation suppression of spontaneous nystagmus in cerebellar infarction: frequency, pattern, and a possible structure. *Cerebellum* 2016;15:182-189.
26. Lee H, Kim HA. Reversal of spontaneous nystagmus during visual fixation in dorsal medullary infarction. *J Neurol* 2020;267:1547-1549.
27. Lee SU, Kim HJ, Choi JY, Choi JH, Zee DS, Kim JS. Nystagmus only with fixation in the light: a rare central sign due to cerebellar malfunction. *J Neurol* 2022;269:3879-3890.
28. Lee SU, Park SH, Park JJ, Kim HJ, Han MK, Bae HJ, et al. Dorsal medullary infarction: distinct syndrome of isolated central vestibulopathy. *Stroke* 2015;46:3081-3087.
29. Choi KD, Oh SY, Park SH, Kim JH, Koo JW, Kim JS. Head-shaking nystagmus in lateral medullary infarction: patterns and possible mechanisms. *Neurology* 2007;68:1337-1344.
30. Helmchen C, Rambold H, Kempermann U, Büttner-Ennever JA, Büttner U. Localizing value of torsional nystagmus in small midbrain lesions. *Neurology* 2002;59:1956-1964.
31. Romano F, Bockisch CJ, Schuknecht B, Bertolini G, Tarnutzer AA. Asymmetry in gaze-holding impairment in acute unilateral ischemic cerebellar lesions critically depends on the involvement of the caudal vermis and the dentate nucleus. *Cerebellum* 2021;20:768-779.
32. Leigh RJ, Zee DS. *The neurology of eye movements*. 5th ed. New York: Oxford University Press, 2015.
33. Eizenman M, Cheng P, Sharpe JA, Frecker RC. End-point nystagmus and ocular drift: an experimental and theoretical study. *Vision Res* 1990;

30:863-877.
34. Tarnutzer AA, Dieterich M. Bedside examination of the vestibular and ocular motor system in patients with acute vertigo or dizziness. *Clin Transl Neurosci* 2019;3:2514183X19886158.
35. Oliva A, Rosenberg ML. Convergence-evoked nystagmus. *Neurology* 1990;40:161-162.
36. Sharpe JA, Hoyt WF, Rosenberg MA. Convergence-evoked nystagmus. Congenital and acquired forms. *Arch Neurol* 1975;32:191-194.
37. Ujjainwala AL, Dewar CD, Fifield L, Rayburn C, Buenting E, Boyle J, et al. Effect of convergence on the horizontal VOR in normal subjects and patients with peripheral and central vestibulopathy. *Neurol Sci* 2022;43:4519-4529.
38. Asprella-Libonati G. Pseudo-spontaneous nystagmus: a new sign to diagnose the affected side in lateral semicircular canal benign paroxysmal positional vertigo. *Acta Otorhinolaryngol Ital* 2008;28:73-78.
39. Bisdorff A, Von Brevern M, Lempert T, Newman-Toker DE. Classification of vestibular symptoms: towards an international classification of vestibular disorders. *J Vestib Res* 2009;19:1-13.
40. Roberts RA, Bittel SN, Gans RE. Positional nystagmus in patients evaluated for dizziness and imbalance. *Adv Otolaryngol* 2016;2016:6974836.
41. Bertholon P, Tringali S, Faye MB, Antoine JC, Martin C. Prospective study of positional nystagmus in 100 consecutive patients. *Ann Otol Rhinol Laryngol* 2006;115:587-594.
42. Lawson J, Bamiou DE, Cohen HS, Newton J. Positional vertigo in a falls service. *Age Ageing* 2008;37:585-589.
43. Young AS, Nham B, Bradshaw AP, Calic Z, Pogson JM, D'Souza M, et al. Clinical, oculographic, and vestibular test characteristics of vestibular migraine. *Cephalalgia* 2021;41:1039-1052.
44. Lemos J, Martins AI, Duque C, Pimentel S, Nunes C, Gonçalves AF. Positional testing in acute vestibular syndrome: a transversal and longitudinal study. *Otol Neurotol* 2019;40:e119-e129.
45. Peña Navarro P, Pacheco López S, Almeida Ayerve CN, Marcos Alonso S, Serradilla López JM, Santa Cruz Ruiz S, et al. Early diagnosis of central disorders mimicking horizontal canal cupulolithiasis. *Brain Sci* 2023;13:562.
46. Bhattacharyya N, Gubbels SP, Schwartz SR, Edlow JA, El-Kashlan H, Fife T, et al. Clinical practice guideline: benign paroxysmal positional vertigo (update). *Otolaryngol Head Neck Surg* 2017;156(3 suppl):S1-S47.
47. Yang X, Ling X, Shen B, Hong Y, Li K, Si L, et al. Diagnosis strategy and Yacovino maneuver for anterior canal-benign paroxysmal positional vertigo. *J Neurol* 2019;266:1674-1684.
48. Anagnostou E, Varaki K, Anastasopoulos D. A minute demyelinating lesion causing acute positional vertigo. *J Neurol Sci* 2008;266:187-189.
49. Choi JY, Glasauer S, Kim JH, Zee DS, Kim JS. Characteristics and mechanism of apogeotropic central positional nystagmus. *Brain* 2018;141:762-775.
50. Choi JY, Kim JH, Kim HJ, Glasauer S, Kim JS. Central paroxysmal positional nystagmus: characteristics and possible mechanisms. *Neurology* 2015;84:2238-2246.
51. Johkura K. Central paroxysmal positional vertigo: isolated dizziness caused by small cerebellar hemorrhage. *Stroke* 2007;38:e26-e27.
52. Johkura K, Kudo Y, Sugawara E. Differential diagnosis of apogeotropic positional nystagmus in the emergency room. *J Neurol Sci* 2019;400:180-181.
53. Young AS, Lechner C, Bradshaw AP, MacDougall HG, Black DA, Halmagyi GM, et al. Capturing acute vertigo: a vestibular event monitor. *Neurology* 2019;92:e2743-e2753.
54. Chang TP, Hong YC, Kuo YH, Schubert MC. Persistent geotropic or apogeotropic nystagmus: recurrence and association with vestibular migraine. *J Neurol* 2025;272:184.
55. Choi SY, Park SH, Kim HJ, Kim JS. Paraneoplastic downbeat nystagmus associated with cerebellar hypermetabolism especially in the nodulus. *J Neurol Sci* 2014;343:187-191.
56. Wray SH, Martinez-Hernandez E, Dalmau J, Maheshwari A, Chen A, King S, et al. Paraneoplastic upbeat nystagmus. *Neurology* 2011;77:691-693.
57. Koohi N, Male AJ, Kaski D. Acute positional vertigo in the emergency department-peripheral vs. central positional nystagmus. *Front Neurol* 2023;14:1266778.
58. Lee SU, Kim HJ, Lee ES, Choi JH, Choi JY, Kim JS. Central positional nystagmus in inferior cerebellar peduncle lesions: a case series. *J Neurol* 2021;268:2851-2857.
59. Macdonald NK, Kaski D, Saman Y, Al-Shaikh Sulaiman A, Anwer A, Bamiou DE. Central positional nystagmus: a systematic literature review. *Front Neurol* 2017;8:141.
60. Lemos J, Strupp M. Central positional nystagmus: an update. *J Neurol* 2022;269:1851-1860.
61. Lechner C, Taylor RL, Todd C, Macdougall H, Yavor R, Halmagyi GM, et al. Causes and characteristics of horizontal positional nystagmus. *J Neurol* 2014;261:1009-1017.
62. Hain TC, Fetter M, Zee DS. Head-shaking nystagmus in patients with unilateral peripheral vestibular lesions. *Am J Otolaryngol* 1987;8:36-47.
63. Lee YJ, Shin JE, Park MS, Kim JM, Na BR, Kim CH, et al. Comprehensive analysis of head-shaking nystagmus in patients with vestibular neuritis. *Audiol Neurootol* 2012;17:228-234.
64. Asawavichiangianda S, Fujimoto M, Mai M, Desroches H, Rutka J. Significance of head-shaking nystagmus in the evaluation of the dizzy patient. *Acta Otolaryngol Suppl* 1999;540:27-33.
65. Lee SU, Kim HJ, Kim JS. Pseudo-spontaneous and head-shaking nystagmus in horizontal canal benign paroxysmal positional vertigo. *Otol Neurotol* 2014;35:495-500.
66. Kheradmand A, Zee DS. The bedside examination of the vestibulo-ocular reflex (VOR): an update. *Rev Neurol (Paris)* 2012;168:710-719.
67. Minagar A, Sheremata WA, Tusa RJ. Perverted head-shaking nystagmus: a possible mechanism. *Neurology* 2001;57:887-889.
68. Huh YE, Kim JS. Patterns of spontaneous and head-shaking nystagmus in cerebellar infarction: imaging correlations. *Brain* 2011;134(Pt 12):3662-3671.
69. Lee SH, Kim JM, Kim JT, Tarnutzer AA. Video head impulse testing in patients with isolated (hemi)nodular infarction. *Front Neurol* 2023;14:1124217.
70. Moon IS, Kim JS, Choi KD, Kim MJ, Oh SY, Lee H, et al. Isolated nodular infarction. *Stroke* 2009;40:487-491.
71. Lee SU, Choi JY, Kim HJ, Kim JS. Recurrent spontaneous vertigo with interictal headshaking nystagmus. *Neurology* 2018;90:e2135-e2145.
72. Huh YE, Koo JW, Lee H, Kim JS. Head-shaking aids in the diagnosis of acute audiovestibular loss due to anterior inferior cerebellar artery infarction. *Audiol Neurootol* 2013;18:114-124.
73. Kim HJ, Kim HJ, Lee JH, Kim JS. Pearls & Oy-sters: vibration-induced downbeat nystagmus: a new cerebellar sign observed in paraneoplastic syndrome. *Neurology* 2023;100:43-46.
74. Kim CH, Shin JE, Song CI, Yoo MH, Park HJ. Vertical components of head-shaking nystagmus in vestibular neuritis, Meniere's disease and migrainous vertigo. *Clin Otolaryngol* 2014;39:261-265.
75. Yang TH, Lee JH, Oh SY, Kang JJ, Kim JS, Dieterich M. Clinical implications of head-shaking nystagmus in central and peripheral vestibular disorders: is perverted head-shaking nystagmus specific for central vestibular pathology? *Eur J Neurol* 2020;27:1296-1303.
76. Hamann KF. Vibration-induced nystagmus: a biomarker for vestibular deficits - a synopsis. *ORL J Otorhinolaryngol Relat Spec* 2017;79:112-120.
77. Dumas G, Curthoys IS, Lion A, Perrin P, Schmerber S. The skull vibration-induced nystagmus test of vestibular function—a review. *Front Neurol* 2017;8:41.
78. Califano L, Mazzone S, Salafia F. Utility of the hyperventilation test in the evaluation of the dizzy patient. *Curr Opin Otolaryngol Head Neck Surg* 2013;21:487-491.
79. Califano L, Melillo MG, Vassallo A, Mazzone S. Hyperventilation-induced nystagmus in a large series of vestibular patients. *Acta Otorhi-*

nolaryngol Ital 2011;31:17-26.
80. Walker MF, Zee DS. The effect of hyperventilation on downbeat nystagmus in cerebellar disorders. *Neurology* 1999;53:1576-1579.
81. Oh EH, Shin JH, Cho JW, Choi SY, Choi KD, Rhee JK, et al. Episodic vestibular syndrome with hyperventilation-induced downbeat nystagmus. *Cerebellum* 2021;20:796-803.
82. Ward BK, van de Berg R, van Rompaey V, Bisdorff A, Hullar TE, Welgampola MS, et al. Superior semicircular canal dehiscence syndrome: diagnostic criteria consensus document of the committee for the classification of vestibular disorders of the Bárány Society. *J Vestib Res* 2021;31:131-141.
83. Edlow JA, Carpenter C, Akhter M, Khoujah D, Marcolini E, Meurer WJ, et al. Guidelines for reasonable and appropriate care in the emergency department 3 (GRACE-3): acute dizziness and vertigo in the emergency department. *Acad Emerg Med* 2023;30:442-486.
84. Wagle N, Morkos J, Liu J, Reith H, Greenstein J, Gong K, et al. aEYE: a deep learning system for video nystagmus detection. *Front Neurol* 2022;13:963968.
85. Parker TM, Badihian S, Hassoon A, Saber Tehrani AS, Farrell N, Newman-Toker DE, et al. Eye and head movement recordings using smartphones for telemedicine applications: measurements of accuracy and precision. *Front Neurol* 2022;13:789581.
86. Green KE, Pogson JM, Otero-Millan J, Gold DR, Tevzadze N, Saber Tehrani AS, et al. Opinion and special articles: remote evaluation of acute vertigo: strategies and technological considerations. *Neurology* 2021;96:34-38.
87. Vinolo-Gil MJ, García-Campanario I, Estebanez-Pérez MJ, Góngora-Rodríguez J, Rodríguez-Huguet M, Martín-Valero R. Telehealth assessment of diagnostic and therapeutic efficacy in peripheral vestibular symptoms: a systematic review and meta-analysis. *Telemed J E Health* 2025 Jan 6 [Epub]. https://doi.org/10.1089/tmj.2024.0301.
88. von Martial R, Leinweber C, Hubert N, Rambold H, Haberl RL, Hubert GJ, et al. Feasibility of telemedical HINTS (head impulse-nystagmus-test of skew) evaluation in patients with acute dizziness or vertigo in the emergency department of primary care hospitals. *Front Neurol* 2022;12:768460.
89. Halmagyi GM, Lessell I, Curthoys IS, Lessell S, Hoyt WF. Lithium-induced downbeat nystagmus. *Am J Ophthalmol* 1989;107:664-670.
90. Kherallah B, Samaha E, Bach SE, Guede C, Kattah JC. Case report: acute vestibular syndrome and cerebellitis in anti-Yo paraneoplastic syndrome. *Front Neurol* 2022;13:960584.
91. Kattah JC, Tehrani AS, du Lac S, Newman-Toker DE, Zee DS. Conversion of upbeat to downbeat nystagmus in Wernicke encephalopathy. *Neurology* 2018;91:790-796.
92. Garces P, Antoniades CA, Sobanska A, Kovacs N, Ying SH, Gupta AS, et al. Quantitative oculomotor assessment in hereditary ataxia: systematic review and consensus by the Ataxia Global Initiative Working Group on digital-motor biomarkers. *Cerebellum* 2024;23:896-911.
93. Lee SU, Park SH, Jeong SH, Kim HJ, Kim JS. Evolution of torsional-upbeat into hemi-seesaw nystagmus in medial medullary infarction. *Clin Neurol Neurosurg* 2014;118:80-82.
94. Park E, Lee SU, Kim HJ, Choi JY, Im GJ, Yu S, et al. Neuro-Behçet's disease presenting as isolated vestibular syndrome. *J Clin Neurol* 2020;16:499-501.