Current concepts in acute vestibular syndrome and video-oculography

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Purpose of review
We present here neuro-otological tests using portable video-oculography (VOG) and strategies assisting physicians in the process of decision making beyond the classical 'HINTS' testing battery at the bedside.

Recent findings
Patients with acute vestibular syndrome (AVS) experience dizziness, gait unsteadiness and nausea/vomiting. A variety of causes can lead to this condition, including strokes. These patients cannot be adequately identified with the conventional approach by stratifying based on risk factors and symptom type. In addition to bedside methods such as HINTS and HINTS plus, quantitative methods for recording eye movements using VOG can augment the ability to diagnose and localize the lesion. In particular, the ability to identify and quantify the head impulse test (VOR gain, saccade metrics), nystagmus characteristics (waveform, beating direction and intensity), skew deviation, audiometry and lateropulsion expands our diagnostic capabilities. In addition to telemedicine, algorithms and artificial intelligence can be used to support emergency physicians and nonexperts in the future.

Summary
VOG, telemedicine and artificial intelligence may assist physicians in the diagnostic process of AVS patients.

Keywords
acute vestibular syndrome, HINTS, HINTSplus, nystagmus, video-oculography

INTRODUCTION

The acute vestibular syndrome (AVS), first introduced by Hotson and Baloh [1], consists of a continuous state of dizziness (>24 h), nystagmus, gait disturbance, nausea, vomiting and motion intolerance [2,3]. The classification into episodic, acute and chronic vestibular syndromes was driven forward by the international committee of the Barany Society and is now reflected in the new ICD-12 catalogue of the WHO. Subcategories for the AVS have recently been described, including spontaneous and triggered AVS [4]. A previous approach focused on symptoms and risk stratification rules, which was associated with a high rate of misdiagnosis [5,6]. Even neuroimaging (MRIs) in the acute state miss about 20–50% of causes of AVS (such as strokes) [7] and is not cost and time-efficient if applied to all AVS patients. Therefore, current concepts suggest a targeted history (including questions about timing and triggers) [4] and a clinical examination focusing on eye movements [8]. The bedside 'HINTS' examination (head impulse test, nystagmus, test of skew) was first introduced in 2009 by Kattah *et al.* [9], and

in the hands of subspecialists, has a higher sensitivity and specificity to detect a central cause of the AVS as compared to MRI. Recent literature recommends the use of video-oculography (VOG) in order to record and quantify nystagmus and the vestibulo-

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ocular reflex (VOR, using video head impulse test) when possible. Quantification of eye movements compared with a clinical examination using Frenzel glasses has the following advantages:

(1) It is less dependent on the clinical skills of the examiner.
(2) It is cost-efficient compared with neuroimaging.
(3) It allows for subtle nystagmus to be more easily appreciated.
(4) It allows an automated analysis of test results, and finally
(5) recording the examination makes it possible for a subspecialist to review remotely.

This review intends to present current concepts in the clinical assessment and diagnostic work-up of patients with the AVS using VOG and modern algorithms in eye movement detection.

PREVALENCE AND DIFFERENTIAL DIAGNOSIS

AVS accounts for nearly 20–25% of dizzy patients visiting the emergency department (ED). Of the AVS patients, 20–25% will have a cause that is potentially life-threatening such as posterior fossa stroke. The large spectrum of diseases causing AVS (Table 1) makes it difficult for a physician to identify the patient at risk using the history and risk stratification alone. Table 1 summarizes a list of differential diagnosis causing an AVS.

VIDEOOCULOGRAPHY AND HINTS

'HINTS' has a high sensitivity and specificity [2,9–11] in detecting vestibular strokes, which exceeds that of the acute MRI performed within 24–48 h after symptom onset [2,12]. Strokes smaller than 1 cm can be missed in up to 50% of the cases within the first 24 h [7]. 'HINTS' allows a fast and accurate bedside assessment of AVS patients even without

Frenzel glasses; however, its accuracy is highly dependent on the examiner's experience. The sensitivity and specificity of 'HINTS' performed by nonexperts is not clearly known. In addition, the sensitivity of 'HINTS' is reduced if AVS patients do not present with spontaneous nystagmus. It was shown that nonexperts (e.g. emergency physicians) used 'HINTS' on almost all dizzy patients regardless the underlying syndrome or the presence of nystagmus [13]. Nystagmus might serve as a biomarker for the severity of symptoms, mandatory for the application of the 'HINTS' examination. Unpublished data from a larger cross-sectional study revealed that 50% of dizzy stroke patients did not have spontaneous nystagmus even after removal visual fixation [14▪▪]. It is under debate, whether we should consider spontaneous nystagmus as a mandatory feature for the classification of AVS. Another clinical feature, such as postural or gait stability, could serve as another 'marker' of severity and predict stroke [15].

Video head impulse test

The video head impulse test (vHIT) is the most potent test to detect a vestibular stroke with an estimated sensitivity of 88% and specificity of 92% [11]. Although experts might perform HITs at the bedside accurately, the assessment (or interpretation) of a clinical HIT without the support of VOG remains difficult due to spontaneous nystagmus and covert corrective saccades [16]. vHIT allows for quantification of the VOR, including gain measurements of the slow phase of the VOR and fast phase metrics such as saccade latencies and amplitude. It was found that VOR gain cutoff more than 0.68 for lateral (horizontal) vHIT was predictive for stroke [11,17,18]. vHIT gain is normal in most PICA strokes but can be unilaterally or bilaterally abnormal in AICA strokes [11]. VOR gain asymmetry less than 20% (absolute asymmetry) or less than 8–10% (Jongkees formula using a normalization procedure) was predictive as well [18]. Further saccade analysis such as cumulative saccade amplitude could further increase the accuracy of stroke prediction [19]; however, this study was performed with scleral search coils and confirmation of study results using vHIT is still lacking. vHIT is prone to artifacts similar to any other eye tracking based system [20]. The results collected with VOG/vHIT are operator-dependent, although the quality of data can be enhanced and artifacts minimized through training. In fact, we observed a steep learning curve and significant improvement in performance after exceeding 160 HITs [21]. There is no need for a large head excursion with vHIT as is typically necessary with the clinical HIT; however, most of the vHIT operators struggle to

perform the movements at high enough head acceleration. Artifacts might still occur even in the hands of an experienced examiner; however, there is no major impact of artifacts on the final result as long as the number of valid vHITs is larger than 10–20 trials [22]. Although the various vHIT systems calculate VOR gain differently, there is no superiority in the method used provided that the vHITs are properly recorded [23].

Videonystagmography

Nystagmus can be recorded using current VOG devices. vHIT devices record eye position traces at higher frame rates (>200/s); however, binocular recordings are often not possible, as the devices must be lightweight enough for dynamic tests such as vHIT. Testing gaze holding with vHIT can be achieved either by maintaining eccentric gaze focusing on the examiner's fingers (nonstandardized gaze angle) or on a visual target projected either on a wall, screen or led light on the goggles frame (standardized gaze angle). Although nystagmus and nystagmus-like eye movements have been recently classified by the international consensus committee of the Bárány Society [24▪▪], the evaluation of nystagmus in 'HINTS' includes only a qualitative assessment of beating direction of horizontal nystagmus in primary and eccentric gaze in light. Nystagmography offers additional quantitative parameters

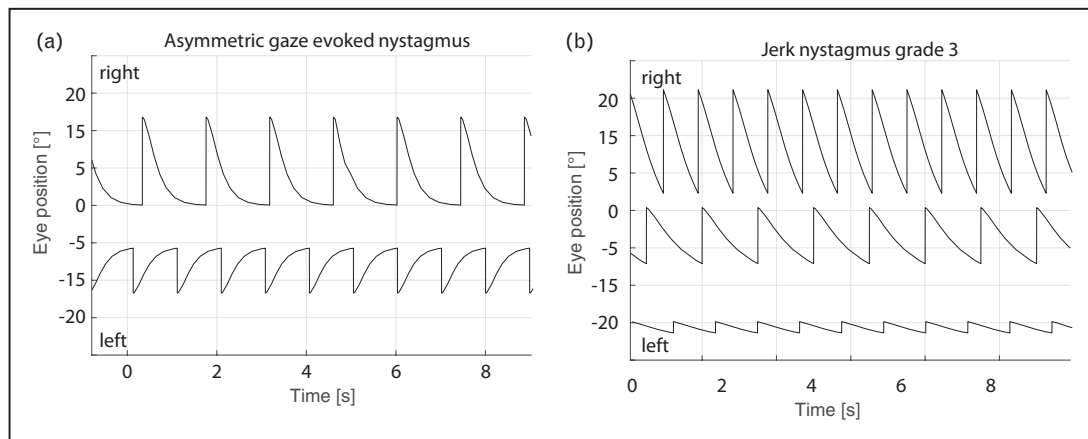| Table 1. Differential diagnosis of AVS		
Vascular	Ischemic strokes Posterior fossa haemorrhages vertebral artery dissection	Horii *et al.*, 2006, Acta Otolaryngol [39]
Inflammatory/Infectious	Acute unilateral vestibulopathy (AUVP) / vestibular neuritis / Labyrinthitis	Mantokoudis *et al.*, 2015, Otol Neurotol [11]
	Brainstem encephalitis	Smiatacz *et al.*, 2006, J Infect [40]
	Ramsay Hunt syndrome	Palchun *et al.*, 2019, Vestn Otorinolaringol [41], Proctor *et al.*, 1979, Ann Otol Rhinol Laryngol [42]
	Mumps virus	Tsubota *et al.*, 2008, Acta Otolaryngol [43]
	Neuroborelliosis	Ishizaki *et al.*, 1993, Acta Otolaryngol Suppl [44]
	Neurosyphilis	Young *et al.*, 2017, J Neurol Sci [45]
	COVID 19	Malayala *et al.*, 2021, Infez Med [46], Mat *et al.*, 2021, Ear Nose Throat J [47], Halalau *et al.*, 2021, SAGE Open Med Case Rep [48]
Neoplastic	Chronic myeloid leukaemia	Martín-Hernández *et al.*, 2013, Case Rep Otolaryngol [49]
	Primary central nervous system lymphoma	Lee *et al.*, 2018, J Neurol [50]
	Paraneoplastic	Moreno-de-Jesús *et al.*, 2021, Acta Otorrinolaringol Esp (Engl Ed) [51]
	Vestibular schwannoma	Sunara *et al.*, 2021, Auris Nasus Larynx [52]
	Temporal bone metastasis	Grubbe Gregersen *et al.*, 2013, Ugeskr Laeger [53]
Degenerative / Deficiency / Drugs	Multiple sclerosis	Barona-Lleo *et al.*, 2014, Am J Otolaryngol [54], Pula *et al.*, 2013, J Neurol [55], Valente *et al.*, 2020, Int J Pediatr Otorhinolaryngol [56]
	Wernicke encephalopathy	Kattah *et al.*, 2013, Neurol Clin Pract [57]
	Creutzfeldt-Jakob Disease	Mantokoudis *et al.*, 2015, Neurologist [58]
	Leukoencephalopathy with subcortical infarction	Rufa *et al.*, 2008, J Neurol Sci [59]
Autoimmune / Allergic / Anatomic	anti-GQ1b antibody syndrome	Lee *et al.*, 2019, Neurology [60]
	Behcet's disease	Tsunoda *et al.*, 1994, Auris Nasus Larynx [61]
	Antiphospholipid syndrome	Vyse *et al.*, 1994, J Laryngol Otol [62]
	Cogan Syndrome	Morgan *et al.*, 1984, Am J Otolaryngol [63]
	internal auditory canal osteoma	Brake *et al.*, 2014, Ear Nose Throat J [64]
	Superior canal dehiscence syndrome	Manzari, *et al.*, 2015, Acta Otolaryngol [65]
	Medullary cavernous malformation	Lee *et al.*, 2017, Neurol Sci [66]
Traumatic	Concussion / whiplash injuries	Vibert *et al.*, 2003, Ann Otol Rhinol Laryngol [67]
	inner ear decompression sickness /barotrauma	Gempp *et al.*, 2014, Eur Ann Otorhinolaryngol Head Neck Dis [68]

FIGURE 1. Video-oculography. (a) A simulated example for asymmetric gaze evoked nystagmus with two horizontal eye positional traces for right and left gaze. There is a decreasing negative exponential eye drift (waveform of slow phase velocity). The beating direction is changing for left and right gaze. Frequency of nystagmus is often lower and the amplitude of the quick phases higher on one side. (b) An example of a vestibular nystagmus (jerk nystagmus with a linear slow phase velocity wave form, which is considered grade 3, as there is spontaneous nystagmus on all horizontal gaze directions. Nystagmus slow phase velocity (SPV) increases with gaze towards the beating direction (e.g. right-beating nystagmus that increases in right gaze) following Alexander's law. Despite the fact that the SPV increases, the waveform itself remains linear.

including its slow phase 'waveform', which is one metric that can be used to distinguish a peripheral from a central lesion in AVS patients. For example, a patient with a stroke can have gaze-evoked nystagmus with a decreasing slow phase velocity waveform (Fig. 1a), whereas a patient with vestibular neuritis will have (vestibular) nystagmus with a linear slow phase velocity waveform (Fig. 1b) [25]. Gaze-evoked nystagmus can often be asymmetric with a more intense nystagmus towards one gaze direction. These patients often have a spontaneous nystagmus at straight gaze as well with a shift of their 'null point', which is a gaze position wherein no eye drift occurs (Fig. 1a) [26■], which is similar to 'Bruns' nystagmus (e.g. cerebellopontine angle tumour or certain posterior fossa syndromes). When present, Bruns nystagmus may allow for determination of the lesion side, for example gaze-evoked nystagmus is typically ipsilesional and vestibular nystagmus is typically contralesional [26■]. It has also been demonstrated that patients with the AVS due to stroke have lower intensity nystagmus ($<12°$/s) compared with patients with an acute unilateral vestibulopathy [14■■]. Although traditional teaching was that spontaneous nystagmus due to an acute peripheral vestibulopathy should be suppressed or partially suppressed by visual fixation, fixation often suppresses spontaneous nystagmus due to stroke as well. However, the magnitude of suppression is lower in stroke patients compared with those with vestibular neuritis [14■■]. A reduction in nystagmus of less than $2°$/s in light (using VOG) is predictive of stroke [13]. However, such subtle changes could be easily overlooked on

bedside examination, although the absence or presence (and to what degree) of fixation suppression can be clinically useful.

Video test of skew

A vertical ocular misalignment, or skew deviation, can be seen in both peripheral [27] and central causes of AVS, as lesions at various levels of the otolithic (utricular) pathway may lead to an ocular tilt reaction (OTR), including head tilt, ocular counterroll and skew. Typical central lesions producing pathologic OTR occur in the vestibular nuclei (lateral medullary or Wallenberg syndrome, caudal to the decussation of utriculo-ocular motor fibres causing an ipsiversive OTR), medial longitudinal fasciculus (usually pontine – rostral to the decussation causing a contraversive OTR) and the interstitial nucleus of Cajal (midbrain – contraversive OTR) [28].

A recent cross-sectional study of AVS patients found a skew deviation prevalence of 26% [29]; however, this included some very small skews that were detected only with VOG and not with bedside ocular alignment testing. Skews larger than 3.3 degrees (5.8 diopters) were indicative of stroke and typically caused vertical diplopia, while 'peripheral' skews were smaller in magnitude and rarely caused vertical diplopia. Because the very small 'peripheral' skews could not be appreciated in these studies without VOG or Maddox rod testing, alternate cover testing as part of the originally described HINTS examination ('Test of Skew') was sufficient to recognize larger skews (>3 degrees) that were likely to be central in

origin. We observed a difficulty in recording skew with VOG due to the following reasons:

(1) Small skews are not detectable in noisy signals due to artifacts or improper eye tracking.
(2) There can be a lack of synchronization between visual stimulus (right versus left eye viewing occurring during alternate cover) and the detection of saccades in time dependency of the stimulus.
(3) There might be an overestimation of skew in patients with spontaneous nystagmus with a vertical component or a potential crosstalk between horizontal and vertical eye movements.

Crosstalk occurs when we observe simultaneous and synchronous vertical eye movements (refixation movements during alternate cover) during pure horizontal eye movements (spontaneous horizontal nystagmus). It can be the result of an improper head/eye calibration when VOG goggles or the cameras on the head are tilted.

NYSTAGMUS DETECTION ALGORITHMS AND ARTIFICIAL INTELLIGENCE

A classical pipeline of automatic nystagmus analysis starts with raw eye position recordings obtained with any VOG or eye tracking device [30]. The ultimate objective is to classify the recording into categories of interest, for example into nystagmus present or absent or into nystagmus that is pathological or physiological. To accomplish this, there are several necessary steps to analyse the data (Fig. 2).

First, data must be cleaned of blinks and other artifacts that may appear in the data, for example due to errors in tracking. If not properly removed, these artifacts may produce spurious instances of apparent high slow-phase velocity and potentially cause a misclassification of the recording.

Second, different portions of the eye movement recording need to be identified. Although quick-phases do relate to nystagmus intensity, we often focus the analysis on the slow-phases, as they more directly correspond with vestibular sensation and vestibular neural pathways. Thus, the analysis must identify the slow phases within the recordings and eliminate the quick phases. Other portions that may need to be removed are periods of time when the head is actually moving, as slow phases will appear as part of the normal vestibular ocular reflex of the individual.

Third, after data are cleaned and segmented, it is usually desirable to obtain an estimation of the instantaneous slow-phase velocity. This signal will measure the intensity of the nystagmus over time

with a temporal precision ideally between a few hundred milliseconds to one second. From this signal, the relevant features for classification can be calculated such as maximum slow-phase velocity, mean slow-phase velocity, time to peak and so on. The relevant features will vary depending on the particular test being analysed. For example, during gaze testing, we may just be interested in mean slow-phase velocity during the entire recording, but during a positional test such as the Dix Hallpike manoeuvre, we want to capture the characteristics of the temporal evolution of the signal, that is how fast it raises, how fast it decays and if it ever changes direction.

Finally, in the simplest case, recordings will be classified according to a threshold and a single feature. For example, whether or not the maximum slow-phase velocity is above a particular speed such as 5 or 10 degree/s. In a more complicated example, we may want to use several features to determine whether the waveform corresponds with a typical crescendo and decrescendo nystagmus waveform from benign paroxysmal positional vertigo (BPPV) during positional testing.

With the advent of machine learning, these steps can be combined into a single machine learning instance that is trained to directly classify eye movement recordings [31]. However, there are two steps that need to occur prior to trying to classify any new recording. First, a large dataset must be collected and labelled by a set of experts. This labelling must correspond with the indented classification to be performed by the machine. For example, a recording in a dataset could be labelled as having nystagmus present or absent according to the experts. Then, the machine is trained with this dataset and becomes ready to classify new recordings. In some cases, machine learning may be used to directly diagnose the patient by combining results and features obtained from multiple tests [32▪▪]. In others, machine learning can be used to only replace an individual step or group of steps in the classical analysis pipeline. For example, machine learning could be used to detect and remove the quick phases in the recordings. Multiple machine learning methods have been developed to detect saccades, which is a similar problem to quick-phase detection. But as shown in a recent study, they will fail in detecting quick-phases of nystagmus because their model was trained with data wherein the eye was relatively still in between saccades [33].

It may be also relevant to compare the challenges of analysing nystagmus versus head impulse tests. One could argue that nystagmus is easier to analyse by the human eye than by a machine, while the opposite is true for head impulse test.
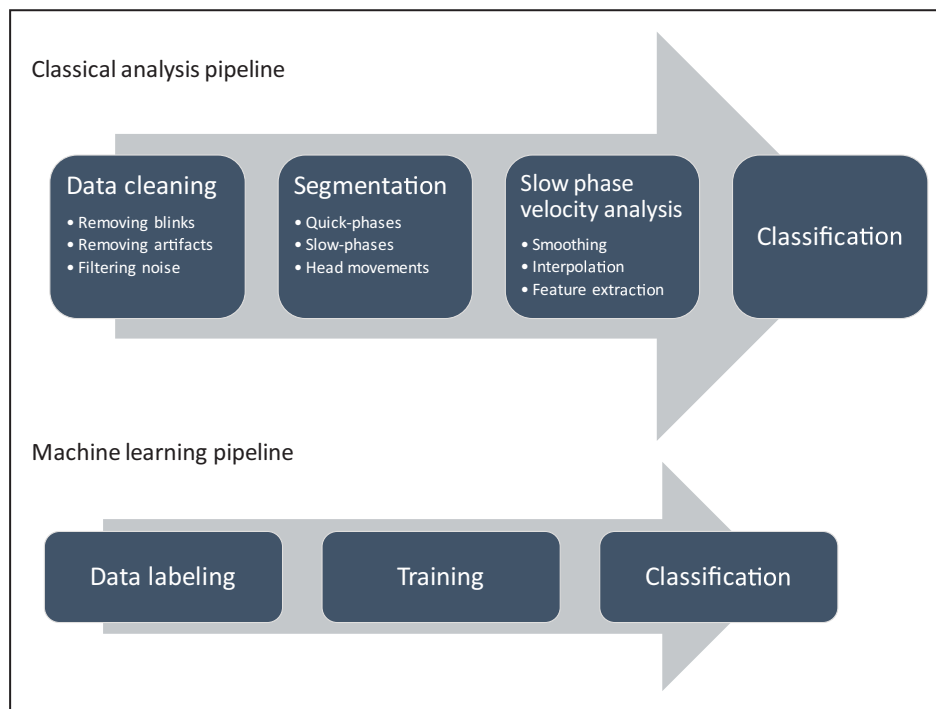
FIGURE 2. Classical and machine learning pipelines for the analysis of nystagmus.

Nystagmus data collection is typically done just once, if something goes wrong and data are contaminated by artifacts it may be hard to recover the relevant features or measurements. In the HIT, on the contrary, an expert can select the appropriate times to do each impulse and coordinate with the individual to have the eyes open and optimize the quality of the data thus minimizing the chance of artifacts. Moreover, data from all impulses are averaged, further reducing the effect of noise or artifacts in single impulse.

Nystagmus can be evaluated clinically and qualitatively either by looking at the patients' eyes, videos or raw eye position traces. Artifacts that are easily ignored by the expert eye can be a challenge for the automatic processes. On the contrary, for HIT, it is easier for the computer algorithms to precisely determine the result by measuring either gain or catch-up saccade frequency, although the clinical assessment must rely exclusively on the presence of catch-up saccades. This reliance in subjective assessment of nystagmus may have resulted in a reduced number of studies analysing the data quality and effect of artifacts in automatic nystagmus analysis while they have been carefully studied for HIT [22]. As we move towards more automated analysis of nystagmus, the lack of input from an expert to identify artifacts or problems with the recording must be recognized as new algorithms are developed and refined.

HINTS PLUS

'HINTS' plus includes a fourth step, which is intended to diagnose acute hearing loss in AVS patients [10]. Hearing is often tested with finger rubbing at the bedside [10,34,35], although this test has a low specificity since the produced sound pressure level is very low (<60 dB SPL). Audiometric assessment under controlled conditions, however, are often not available in EDs. Hearing loss and simultaneous vestibular loss could be the result of an inner ear inflammation such as a labyrinthitis in 50–60% of AVS patients. These inflammatory conditions are assumed to arise from viral or postviral causes akin to patients presenting with isolated loss of vestibular (i.e. vestibular neuritis) or auditory (i.e. idiopathic sudden sensorineural hearing loss) function. In these cases, otoscopy is typically normal unless there is an underlying infection such as an otitis media. The range of hearing loss severity is often variable from mild to severe or even complete deafness.

However, hearing loss might also be a red flag due to involvement of the anterior inferior cerebellar artery (AICA, pontine structures including the cochlear nucleus and the nerve entry zone – this is rare) and the internal auditory artery (a branch of the AICA causing labyrinthine ischemia, this is much more common) [36]. Different patterns and severity of hearing loss can be classified by audiometric testing [36]. Interestingly, there is a high

recovery rate reported [37]. A moderate to severe hearing loss (>70 dB) on all frequencies would be expected due to cochlear infarction, which in fact has been demonstrated with MRI-proven posterior circulation infarctions [37]. We would further expect an isolated high frequency loss if there is isolated involvement of the vestibulocochlear artery and the cochlear ramus supplying the basal turn of the cochlea. Such isolated small emboli would not be visible in current imaging modalities and occur often after decompression sickness. Further studies quantifying hearing loss are needed in order to develop and recommend any quantitative screening tool for hearing loss in the future.

OCULAR LATERAL DEVIATION

A recent study proposed another bedside test to apply in patients with the AVS, looking for evidence of ocular lateral deviation (lateropulsion) [38■]. Ocular lateral deviation is a conjugate horizontal deviation occurring under closed eyelids, which has been shown to be present in 12% of stroke patients presenting with the AVS. Patients are asked to close their eyes for 3–5 s, and when opening the eyes, the examiner should look for corrective eye movements (saccades) back towards the centre. Ocular lateral deviation is most commonly seen in a lateral medullary (Wallenberg) syndrome, wherein saccades are typically hypermetric towards the lesion and hypometric away from the lesion. Under closed eyelids, ocular deviation occurs toward the side of the lesion (known as ipsipulsion), and when the eyes are opened, contralesional hypometric saccades will bring the eyes back to centre. This test showed a low sensitivity of 12% but a specificity of 100% for a central lesion [38■], but when present, is highly suggestive of a central lesion.

ACUTE VESTIBULAR SYNDROME AND TELEMEDICINE

In the hands of eye movement experts, HINTS is proven. However, its sensitivity and specificity in the hands of nonsubspecialists is unknown. Although dizziness/vertigo is a common complaint in the ED, there are few subspecialists to evaluate these patients. So, how can we utilize VOG/vHIT technology in the ED where getting the diagnosis wrong can be life-threatening?

Implementation of a 'tele-dizzy' clinical service that includes a standardized battery of VOG tests (mainly HINTS and Dix-Hallpike/supine roll) would enable a single expert to drastically expand their clinical reach through simultaneous coverage of multiple EDs. A tele-dizzy service could reliably,

inexpensively and quickly distinguish ear and brain causes of dizziness and vertigo, as experts are providing the interpretation. Eye movement specialists can now almost instantaneously review a VOG examination remotely in minutes, enabling rapid tele-diagnosis. Addition of a standardized dizzy questionnaire would enhance localization and the ability to generate an accurate differential diagnosis, especially in patients with transient symptoms or in those without spontaneous nystagmus wherein HINTS cannot be applied. A tele-dizzy service would enable standard expert clinical care provided by a remote means.

In fact, at The Johns Hopkins Hospital, 290 tele-dizzy consults have been performed since 2017 with a drastic measurable increase in diagnostic yield and decrease in unnecessary test utilization compared with a baseline ED population. The rate of specific vestibular diagnoses made in the ED with tele-dizzy is 57% compared with a baseline rate of 21% (+176%). Furthermore, our tele-dizzy service has recommended computed tomography (CT) scans 2% of the time compared to a baseline CT rate of 49% (-96%). Next steps include scaling this consultation service for expansion to other hospitals. We also see an important role for smartphone app VOG-based triage from home in the future. This would allow for the accurate diagnosis of BPPV, but could also detect red flag signs (e.g. spontaneous vertical/vertical-torsional nystagmus; vertical refixation with test of skew) that would prompt rapid referral to the ED.

IMPLICATIONS

The systematic use of VOG devices is a first step in the process of quantification and objective classification of clinical signs in AVS patients such as abnormal head impulses, spontaneous nystagmus, skew, pathologic saccade metrics and other eye movements. There is, however, a lack of an automated VOG interpretation. We suggest a similar approach to that seen in patients with acute chest pain and suspected heart attack. A point-of-care examination such as an ECG performed by nonexperts in the ED allows an automated computerized ECG interpretation. Analogous to an ECG, an automated VOG exam in conjunction with artificial intelligence could pave the way for a broadly available decision support system.

CONCLUSION

There is no doubt that the HINTS examination is highly effective in detecting central causes of the AVS *in the hands of subspecialists*. Dissemination of the HINTS examination is still limited in nonsubspecialists, perhaps due to its absence in medical school and residency programme curriculums, or that its

importance is under-emphasized for neurology, oto-laryngology and emergency medicine trainees. When the HINTS examination is performed by non-subspecialists, it is commonly applied incorrectly (e.g. HINTS examination in a patient with positional vertigo), performed incorrectly (e.g. the HIT) or interpreted incorrectly (e.g. a 'peripheral' HINTS is diagnosed in a patient who actually has a stroke). Ideally, the HINTS examination would always be performed by a subspecialist, although this is practically impossible due to a paucity of eye movement/vestibular experts. A battery of recorded VOG/vHIT testing in the emergency setting using a store-and-forward methodology (akin to a radiologist reading a study) would allow for one expert to remotely review and interpret patient data from multiple locations. Practically speaking, leveraging technology in this way allows the subspecialist to participate in the care of the patient early in their course with benefits, including decreasing (unnecessary) test utilization, and increasing diagnostic accuracy (see 'AVS and telemedicine' section above).

However, even subspecialists may struggle to distinguish central from peripheral causes in select cases. As discussed above, there are quite a few caveats and exceptions to the HINTS examination rules, and VOG/vHIT can be a powerful tool to enhance diagnostic accuracy.

(1) Head impulse test – there are a variety of central localizations and stroke syndromes that are capable of producing an abnormal HIT. Video HIT allows for quantification of the gain, and the lower the gain, the greater the likelihood that a peripheral vestibulopathy is the culprit. The function of all six semicircular canals can be quantified with vHIT, allowing for identification of superior (horizontal and anterior canal) and/or inferior (posterior canal) vestibular nerve involvement.

(2) Nystagmus – with VOG, slow phase velocity waveforms can be analysed; subtle changes in slow phase velocity can be measured in fixation and fixation-removed conditions (greater suppression with fixation in peripheral vestibulopathy); horizontal, vertical, torsional components of nystagmus can be analysed and quantified.

(3) Test of skew – with VOG, small skews that are not enough to cause diplopia may be detected, which could be helpful for the subspecialist (who can localize and diagnose a 'peripheral' skew due to utricle involvement in vestibular neuritis).

Acute VOG/vHIT testing in the emergency room has the potential to revolutionize the care of dizzy patients. The VOG/vHIT-enhanced HINTS examination has many benefits over the clinical/bedside HINTS, but perhaps most importantly it would allow a single subspecialist to impact many patients due to remote review and interpretation capabilities. The development of algorithms would enable further scalability through rapid automated diagnosis of the clearly benign (e.g. BPPV, peripheral vestibulopathy) and dangerous (e.g. stroke) disorders, allowing the subspecialist to focus in on the most challenging and ambiguous cases. We believe that VOG/vHIT-based diagnosis and triage of acutely dizzy patients should be (and will be) the standard of care, although a variety of technological, logistical and financial/billing barriers must first be surmounted.

Conflicts of interest

None of the investigators has any relevant financial interests, activities, relationships or affiliations that represent a relevant financial conflict of interest with respect to this article.

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■■ of outstanding interest

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