

ACUTE VESTIBULAR SYNDROME

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Acute Vestibular Syndrome

Overview

The *acute vestibular syndrome* (AVS) is a clinical condition characterized by dizziness or vertigo that develops acutely (over seconds, minutes, or hours); is accompanied by nausea/vomiting, gait instability, nystagmus, and head-motion intolerance; and persists for a day or more.^{1,2} Most patients presenting with AVS are believed to suffer an acute, self-limited, presumed viral or post-viral vestibular disorder commonly called vestibular neuritis (VN). This disorder is also sometimes referred to as vestibular neuronitis, labyrinthitis, neurolabyrinthitis, or acute peripheral vestibulopathy.^{1,2} However, central causes of AVS (C-AVS), particularly brainstem and cerebellar ischemic stroke, can closely mimic benign peripheral causes (P-AVS), particularly VN.²⁻⁵ There is growing evidence that many C-AVS patients are misdiagnosed on initial presentation to the emergency department (ED) or other frontline healthcare settings⁶⁻⁹ and that physicians are eager for diagnostic guidelines.^{10,11}

Epidemiology of AVS

Dizziness/vertigo is the third most common major medical symptom reported in general medical clinics¹² and accounts for roughly 3-5% of visits across clinical settings.¹³ This translates to ~10 million ambulatory visits per year in the US for dizziness¹⁴ with ~25% of these visits to EDs.¹³ Based on calculations from published and unpublished studies, we estimate that ~10-20% of patients presenting with acute dizziness have AVS, corresponding to 250-500,000 ED visits per year in the US alone (Newman-Toker, unpublished).

Differential Diagnosis of AVS

The most common diagnoses identified in AVS patients are VN for P-AVS and posterior fossa ischemic stroke for C-AVS, but their relative prevalence is difficult to estimate because no high-quality, published diagnostic studies have enrolled completely unselected AVS presentations. Though the complete differential diagnosis is probably fairly broad^{15,16} central mimics of VN (sometimes called “pseudoneuritis”³) are predominantly cerebrovascular (82%) and demyelinating (11%) in etiology (Box 1). Considering the estimated incidence of AVS and the calculated frequency of posterior fossa stroke presenting with dizziness (~70-100,000 calculated as ~50-70% [frequency of dizziness in posterior fossa stroke]^{17,18} of ~18% [proportion of strokes located in the posterior fossa]¹⁹ of 795,000 strokes/year US²⁰), the true fraction of stroke in AVS is probably about 25 ±10%.

To meet the clinical definition of AVS, *total duration* must exceed 24 hours of continuous dizziness. This excludes most disorders in which dizziness typically presents with transient episodes lasting seconds, minutes, or hours, such as benign paroxysmal positional vertigo (BPPV), cardiac arrhythmia, transient ischemic attack (TIA). Thus, these disorders rarely remain diagnostic considerations in AVS patients beyond the first few minutes or hours. Menière disease and vestibular migraine may be exceptions, with an estimated 12%²¹ and ~27%,²² respectively, having attacks of dizziness that last longer than 24 hours.

Box 1. Differential diagnosis of AVS

<p>BENIGN* or LESS URGENT causes</p> <p><i>Common Causes (>1% of AVS)</i></p> <ul style="list-style-type: none"> • vestibular neuritis • multiple sclerosis <p><i>Uncommon or Unknown Frequency</i></p> <ul style="list-style-type: none"> • viral labyrinthitis • herpes zoster oticus (Ramsay Hunt) • acute traumatic vestibulopathy • med ototoxicity (e.g., aminoglycosides) • acute disseminated encephalomyelitis • CNS side effects (e.g., antiepileptics) • prolonged Menière syndrome attack • prolonged vestibular migraine attack • episodic ataxia syndrome attack <p><i>Presumed Possible Causes‡</i></p> <ul style="list-style-type: none"> • atypical infection (otosyphilis, lyme) • degenerative cerebellar ataxia • cerebello-pontine angle neoplasm (e.g., vestibular schwannoma, metastases) • drug intoxication (e.g., alcohol, illicit) 	<p>DANGEROUS* and MORE URGENT causes</p> <p><i>Common Causes (>1% of AVS)</i></p> <ul style="list-style-type: none"> • brainstem or cerebellar infarction • brainstem or cerebellar hemorrhage <p><i>Uncommon or Unknown Frequency</i></p> <ul style="list-style-type: none"> • labyrinthine stroke†²³ • bacterial labyrinthitis/mastoiditis • Wernicke syndrome (B1 deficiency) • Miller Fisher syndrome • brainstem encephalitis (e.g., listeria, herpes simplex/zoster, paraneoplastic) <p><i>Presumed Possible Causes‡</i></p> <ul style="list-style-type: none"> • cerebral infarction or hemorrhage§ • subarachnoid hemorrhage/aneurysm • severe anemia or hypoxia • carbon monoxide toxicity • electrolyte (e.g., hyponatremia) • endocrine (e.g., hypothyroidism) • decompression sickness • CNS medication toxicity (e.g., lithium)
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CNS = central nervous system

* Any disease causing dizziness/vertigo can be considered a ‘dangerous’ medical problem if the symptoms occur in dangerous circumstances (e.g., highway driving or free-rock climbing). Furthermore, the high vagal tone that accompanies some vestibular disorders can provoke bradyarrhythmias in susceptible individuals. Nevertheless, although they may be quite disabling during the acute illness phase, diseases classified here as ‘Benign or Less Urgent Causes’ rarely produce severe, irreversible morbidity or mortality (unlike their ‘Dangerous’ counterparts).

† The frequency of labyrinthine infarction is difficult to estimate given that the current reference standard test for confirming the diagnosis (i.e., autopsy with temporal bone histology) is rarely performed. Recent studies, however, suggest that patients with sudden deafness, with or without vertigo, are at increased risk of stroke, suggesting a possible vascular mechanism.^{24;25}

‡ Presumed possible causes are known to cause acute dizziness, but it remains unknown whether they can present with a clinically-complete or clinically-predominant AVS picture.

§ AVS secondary to cerebrovascular disease is strongly associated with posterior fossa infarcts,² although transient dizziness or vertigo may be present in up to 10% of patients with right-hemispheric stroke²⁶ and isolated cases of insular or parietal infarction.^{27;28} The precise duration of symptoms in such cases is unknown.

Predictors of Stroke in AVS – The History – Type, Timing, & Triggers

The *type* or *quality* of dizziness is often the main focus for directing diagnostic inquiry in patients with dizziness.²⁹ Classic teaching{Drachman, 1972 75 /id} and current US practice¹¹ subdivides dizziness into four types based on symptom quality, each said to predict the underlying etiology: (i) vertigo (false sense of spinning or motion), (ii) presyncope, (iii) unsteadiness, and (iv) nonspecific/other dizziness.^{30;31} Despite the fact that AVS has sometimes been referred to as “prolonged spontaneous vertigo,”³² the type of dizziness in AVS is not likely a good predictor of underlying etiology. In a population-based study of ED dizziness, patients with unsteadiness as part of their complaint were at slightly higher risk of stroke, but vertigo or other dizziness predicted stroke with equal likelihood.⁶ These results accord with disease-based studies (reviewed in Newman-Toker et al.³³) indicating different types of dizziness are present in patients with disorders known to cause AVS such as VN,³⁴ stroke,^{18;35} and hemorrhage.³⁶

Some experts suggests that *abrupt onset* of dizziness in AVS favors a vascular cause, whereas VN produces symptoms more gradual in onset, over hours,¹ and there is some evidence that partially supports this claim.^{37;38} *Transient isolated episodes of prodromal dizziness* may precede an AVS presentation. As a harbinger of stroke, these episodes presumably represent TIAs. While a *single* episode of transient dizziness within a few days prior to AVS presentation is probably non-specific (reported in ~25% of either P-AVS³⁹ and C-AVS^{37;38;40}), recurrent episodes lasting seconds to minutes over the preceding weeks or months may favor a cerebrovascular origin.^{37;38} In one series, 29% (n=12/42) of vertebrobasilar stroke patients had prodromal, isolated attacks of vertigo for up to two years leading up to the stroke.³⁷

Although *triggers* represent a key source of diagnostic information for patients with brief, episodic dizziness (e.g., for those with BPPV or orthostatic hypotension which are position-provoked⁴¹), this is probably not the case for AVS. Patients with AVS tend to be intolerant of head motion during the acute stage, regardless of the underlying localization or etiology.⁴² This occurs because head movement (a non-specific vestibular stimulus) preferentially excites the intact inner ear (or central connections), exacerbating the pathologic right-left asymmetry responsible for the dizziness or vertigo.¹⁶ Not surprisingly, exacerbation of symptoms or signs during head motion or positional testing has been reported in both VN⁴³ and stroke.⁴⁴

Predictors of Stroke in AVS – The History – Associated Symptoms & Risk Factors

Neurovegetative (nausea with or without vomiting) and gait or postural symptoms are core features of AVS, regardless of the underlying cause. *Proportionality* of dizziness, neurovegetative, and gait/postural symptoms is thought to be typical of peripheral vestibular disorders, while central syndromes are said to sometimes be associated with disproportionate symptoms.^{16;17} Vomiting^{35;45;46} or imbalance/gait unsteadiness³⁵ out of proportion to the degree of dizziness or nystagmus may be a marker for brainstem or cerebellar pathology, including stroke. The presence of other *neurologic symptoms* (e.g., diplopia) or signs (e.g., internuclear ophthalmoplegia) indicates a central cause of AVS (C-AVS), but their absence is a relatively poor predictor of P-AVS.^{2;24;47;48}

Auditory symptoms usually point to a peripheral localization in patients with dizziness (reviewed in Newman-Toker¹⁶), but this sometimes leads to the mistaken diagnosis of a more benign cause.⁴⁹ The vascular supply to the inner ear arises from the posterior cerebral circulation, and combined audio-vestibular presentations may be common among those with cerebrovascular disease.⁵⁰ Unfortunately, patients with auditory symptoms have generally been excluded from major studies of AVS or VN. Nevertheless, mounting evidence suggests that auditory symptoms in AVS patients can and do result from inner ear ischemia, often in association with TIA or stroke in the anterior inferior cerebellar artery (AICA) territory.^{17;24} Transient dizziness, hearing loss, and/or tinnitus may occur as a premonitory TIA preceding acute audio-vestibular loss due to AICA infarction in up to 42% of cases.^{24;40} Patients with sudden hearing loss (in some cases presumably co-occurring with vertigo or vestibular symptoms) have been found to be at increased stroke risk (OR 1.64, 95% CI, 1.31 to 2.07) relative to a general hospital population control group during the five year period following hospitalization.²⁵

Cranio-cervical pain may accompany dizziness in posterior fossa strokes as a result of the stroke itself (mass effect or direct involvement of pain-sensitive structures) or its underlying cause (e.g., vertebral artery dissection or aneurysm).¹⁷ It is unclear whether headache or neck pain does² or does not⁵ predict a central cause for AVS. Recent head or neck *trauma* is a known risk factor for vertebral artery dissection (estimated odds ratio 3.8 for minor trauma⁵¹). Since vertebral artery dissection is a known potential cause of AVS,² a history of trauma should spark concern for underlying dissection.⁵¹ However, since roughly half of symptomatic vertebral artery dissections occur *without* an identifiable history of trauma,⁵² the absence of trauma is insufficient to exclude the possibility.

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Typical vascular risk factors (e.g., chronic hypertension, diabetes mellitus, hypercholesterolemia, cigarette use) should obviously be considered, since the presence of even one stroke risk factor appears to increase the risk of stroke in AVS by several-fold (e.g., ~69%² vs. 25%). Though age is a known risk factor for stroke, there is evidence that stroke should also be considered in younger patients. In one prospective study of AVS patients, 25% (n=15/69) with ischemic stroke (and three of four patients with vertebral artery dissection as the underlying cause) were under 50 years of age.² Younger patients with AVS may be more likely to be misdiagnosed than their older counterparts, with a small case series of missed cerebellar infarctions finding that 50% (n=7/14) who presented with dizziness/vertigo were under age 50, 6/7 of whom had vertebral artery dissection or occlusion.⁹

When obtaining the medical history from a patient with AVS, one should also strive to identify any major known, *concurrent illnesses* (e.g., multiple sclerosis, HIV-AIDS, metastatic malignancy) or *potentially-relevant exposures* (e.g., ear surgery, bacterial otitis media, ototoxic drugs, chemotherapy, or viral syndrome). However, virtually nothing is known about the predictive value of these historical elements for the underlying cause of AVS.

Predictors of Stroke in AVS – The General Neurologic Examination

The presence of *general neurologic findings* has occasionally been touted as the principal differentiating feature between P-AVS (usually VN) and C-AVS (usually stroke).^{53;54} However, case reports dating back over 30 years indicate that some patients with stroke mimic VN in almost all clinical attributes.⁵⁵ The proportion of AVS patients with focal neurological signs in AVS is difficult to estimate with confidence, since most studies of AVS exclude subjects with other signs/symptoms of brainstem/cerebellar disorder. Focal neurological signs have been reported in approximately 80% (n=201/251) of AVS stroke patients,^{2;24;40;47;56-59} but diagnostic ascertainment bias likely makes this proportion an overestimate. Current best data come from a prospective study of 101 consecutive, high-risk-for-stroke AVS patients.² Not counting truncal ataxia, authors found “obvious” general neurological (e.g., facial palsy, sensory loss, limb ataxia, hemiparesis) or oculomotor (e.g., internuclear ophthalmoplegia, gaze palsy, vertical nystagmus) signs in 42% of 76 patients with C-AVS, but none of those with P-AVS.²

It has been suggested that *severe difficulty walking or inability to sit or stand* unaided is a sign of central pathology in AVS,^{17;60} and the assertion is supported by some evidence. The prospective study mentioned above found severe truncal ataxia (inability to sit with arms crossed unaided) more frequent in stroke (33%) than VN (0%) ($p < 0.001$).² A study of cerebellar infarctions presenting with pseudo-neuritis demonstrated a high proportion with severe imbalance (71%, n=17/24)⁴ and a retrospective, population-based study found imbalance/gait unsteadiness to be associated with stroke in acute dizziness (OR 3.71 95% CI 1.30-10.65).⁶

Predictors of Stroke in AVS – The Neuro-vestibular/Oculomotor Examination

Given the relatively low sensitivity of the general neurological examination for detecting stroke in AVS, a number of studies have focused on bedside tests of vestibular and oculomotor function as assessed through careful *eye movement examination*. Among the tests reported in these studies, a normal horizontal head impulse test (h-HIT) of vestibulo-ocular reflex function⁶¹ is the single best bedside predictor of the final diagnosis (VN versus stroke), based on the results of a recent systematic review (Newman-Toker, unpublished) (Table 1). By itself, the h-HIT result probably outperforms MRI with diffusion-weighted imaging (DWI) sensitivity for detecting posterior fossa stroke (~80%^{62;63}), with comparable specificity.² In AVS, an abnormal h-HIT response usually indicates a peripheral vestibular lesion while a *normal* response virtually confirms a stroke.⁴⁸ However, ~15% of patients with stroke or other central etiology might be misdiagnosed as having VN due to an abnormal h-HIT if other eye findings are not considered. Falsely abnormal (i.e. suggesting a peripheral vestibular lesion) h-HIT findings are almost exclusively associated with AICA infarctions. AICA strokes often result in an ischemic lesion of the labyrinth or eighth nerve root entry zone that mimics VN anatomically and physiologically.^{17;50} By contrast, the h-HIT is almost a perfect predictor when considering the more common posterior-inferior cerebellar artery (PICA) strokes, which generally affect only the cerebellum or lateral medulla.^{2;17}

Another bedside predictor of C-AVS is direction-changing horizontal nystagmus on lateral gaze,¹ which is believed to represent failure of so-called “gaze-holding” structures located in the brainstem and cerebellum. Direction-changing horizontal nystagmus correctly identifies central causes with high specificity (92%) but low sensitivity (38%) (Table 1). Vertical ocular misalignment of vestibular cause (so-called “skew deviation” or “skew”) during the alternate cover test is a third bedside predictor for a C-AVS. Skew results from a right-left imbalance in otolithic/graviceptive inputs from the vestibular system to the oculomotor system, and, with rare exceptions, is generally central in origin.⁶⁴ Skew, similar to direction-changing nystagmus, correctly identifies central causes with high specificity (98%), but low sensitivity (30%) (Table 1).

Table 1. Test properties of bedside oculomotor predictors of stroke in AVS (Newman-Toker, unpublished*)

Test	Sensitivity (95% CI)	Specificity (95% CI)	NLR (95% CI)	PLR (95% CI)
Normal h-HIT	85% (79-91)	95% (90-100)	0.16 (0.11-0.23)	18.4 (6.08-55.6)
▪ PICA only	▪ 99% (96-100)	▪ data not available	▪ 0.01 (0.00-0.10)	▪ data not available
▪ AICA only	▪ 62% (35-88)	▪ data not available	▪ 0.40 (0.20-0.80)	▪ data not available
Direction-changing nystagmus	38% (32-44)	92% (86-98)	0.68 (0.60-0.76)	4.51 (2.18-9.34)
Skew deviation	30% (22-39)	98% (95-100)	0.71 (0.63-0.80)	19.7 (2.76-140)

CI = confidence interval; NLR = negative likelihood ratio; PLR = positive likelihood ratio

* Results based on a recent systematic review of the medical literature (data compiled from 7 primary studies)

Taken together, the combination of the three bedside oculomotor tests described above as a battery has been used to define a clinical prediction rule for stroke called “H.I.N.T.S.” (h-HIT, evaluation of Nystagmus, and Test of Skew).² Test results predicting stroke on the H.I.N.T.S. battery can be remembered using the acronym/mnemonic “I.N.F.A.R.C.T.” (Impulse Normal *or* Fast-phase Alternating *or* Refixation on Cover-Test). Authors found that the presence of any one “dangerous” sign was 100% sensitive and 96% specific for stroke (negative likelihood ratio [NLR] 0.00, 95% CI 0.00–0.12), surpassing MRI with DWI in the ability to urgently rule out stroke in AVS patients presenting in the first 24-48 hours after symptom onset (estimated NLR 0.21, 95% CI 0.16-0.26; *calculations below*). These clinical tests can be performed in approximately one minute at the bedside (see sections below for description of technique). The H.I.N.T.S. battery has not been fully validated by accepted standards for clinical prediction rule development⁶⁵ and its generalizability to routine clinical practice (with non-expert examiners) remains to be demonstrated. Nevertheless, since it was developed based on well-established physiologic principles rather than post-hoc empiric statistical data mining, and it appears to outperform the current gold standard (MRI with DWI), strong consideration should be given to taking this approach in clinical practice.

The vector, pattern, and fixation characteristics of spontaneous nystagmus may be able to help distinguish P-AVS from C-AVS. Mixed horizontal-torsional nystagmus that (a) is suppressed by visual fixation and (b) obeys Alexander’s law (i.e., worsens with gaze in the direction of the fast phase and lessens with gaze in the direction of the slow phase) is said to be typical of VN and diagnostic of P-AVS.^{1,66} The “opposite” features are said to predict C-AVS (usually stroke). However, there is no high-quality, empiric evidence to support or refute these assertions. Studies comparing spontaneous nystagmus characteristics (and bedside means to assess them such as Frenzel goggles⁶⁷ or penlight-cover test⁶⁶) in AVS patients are needed to determine their diagnostic utility in differentiating P-AVS from C-AVS. The same is true of bedside measures of smooth pursuit breakdown, saccade dysmetria, impaired optokinetic nystagmus, or impaired VOR cancellation, all of which are said to predict central lesions.

As alluded to above, experts agree that specific provocative testing procedures such as the Dix-Hallpike (Nylén-Bárány) positional maneuver are crucial in the evaluation of *transiently* dizzy patients to elicit symptoms (i.e., dizziness/vertigo) and signs (i.e., nystagmus),^{41,60} but generally unhelpful diagnostically in patients with AVS as they fail to differentiate between central and peripheral causes.^{42,60} Other forms of provocative testing to elicit nystagmus for diagnostic purposes (e.g., mastoid vibration, valsalva, head shaking⁶⁷) have also been described for use in patients with transient, mild, or residual symptoms, but have not been sufficiently studied as diagnostic predictors of underlying etiology. Based on evidence of frequent misdiagnosis and misconceptions in patients presenting with acute dizziness,^{6,8;9;11;42;68} it seems prudent to recommend that none of these provocative maneuvers (including the Dix-Hallpike) be routinely applied to patients with AVS in the hyperacute setting. Both physiologic and empiric evidence indicates they will only lead to a non-specific worsening of complaints, including nausea and vomiting, which could be misinterpreted as diagnostic of a peripheral vestibular disorder (either VN or BPPV),^{8;11} before stroke has been adequately excluded.

Neuroimaging in AVS

Although the predominant form of neuroimaging for patients with dizziness in the ED remains CT,¹³ substantial evidence indicates this approach is inadequate.⁸ A well-designed study has shown that the sensitivity of CT for all acute ischemic stroke is only 16% compared to 83% for MRI with DWI with masked expert readers.⁶⁹ Imaging in AVS must assess acute ischemic or hemorrhagic lesions in the posterior fossa, where CT is known to perform

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particularly poorly due to bone-related artifacts.⁷⁰ Although CT identifies acute intracranial hemorrhage with high sensitivity,⁶⁹ only ~2% of AVS presentations are due to hemorrhage (Newman-Toker, unpublished). Not surprisingly, CT has been shown to have a low diagnostic yield in the assessment of patients with dizziness.⁷¹

As alluded to above, although frequently viewed as the gold standard and final arbiter for stroke diagnosis, MRI with DWI has been shown to be falsely normal as late as ~48 hours after symptom onset in patients with ischemic stroke causing AVS. In one study where patients with AVS, negative DWI, and clinical signs of infratentorial stroke underwent repeat MRI with DWI, the initial MRI was found to be falsely negative in 12% (n=8/69) with follow-up MRI subsequently confirming stroke.² Combined with figures reported in two prior studies of MRI with DWI in acute vertebrobasilar strokes (23% false negatives; n=47/206),^{62;63} the aggregate posterior-fossa MRI with DWI sensitivity is estimated at 80% (n=220/275). Using the aggregate “all ischemic stroke” measured specificity of 97% (n=198/205),^{2;69} this gives an estimated NLR of 0.21 (95% CI 0.16-0.26), making MRI less potent as a “rule out” tool than the three bedside oculomotor predictors described above.²

Prognosis of AVS and Consequences of Diagnostic Failure

While details of management and treatment of AVS are beyond the scope of this course and are considered in detail elsewhere,^{17;72} a brief discussion of prognosis and the relevance of diagnostic failure is warranted. Patients with posterior fossa stroke in AVS are at risk for additional strokes and secondary complications of the initial stroke, particularly ischemic swelling following large cerebellar infarction. Ischemic stroke of the cerebellum⁷³ and cerebellar hemorrhage⁷⁴ can be fatal without close monitoring and urgent surgical intervention (ventriculostomy or posterior fossa decompression) at the time of clinical deterioration. A critical literature review on cerebellar stroke found that 10-20% of patients deteriorate in the days following the event and swelling peaks on the third day post infarction.¹⁷ Thus, C-AVS (stroke) patients mistakenly thought to have P-AVS (VN) may appear clinically stable at the time of discharge from the ED but be at risk for life-threatening complications days later.

Dizziness is the ED symptom most often linked to missed diagnosis of stroke,⁶⁸ with 35% of cerebrovascular events in patients with *any* dizziness (and 44% in those with *isolated* dizziness) missed at first medical contact.⁶ While the real-world impact of these errors on patient outcomes is incompletely known, available data suggest those misdiagnosed are probably at particularly high risk for adverse outcomes. For example, the only published series of misdiagnosed cerebellar strokes (14 of 15 of whom presented with with dizziness, vertigo, or imbalance) found 40% (n=6/15) ended in death⁹ compared to 5% (n=15/282) in the largest series of cerebellar infarctions⁷⁵ (p<.0001, Fisher’s exact). This discrepancy is present despite the fact that almost all the misdiagnosed patients presented with milder symptoms (i.e., normal alertness in 93% (n=14/15)⁹ versus 69% (n=195/282)⁷⁵ of all cerebellar strokes, p=0.046, Fisher’s exact), and milder presentations are associated with a substantially better prognosis (among 282 patients, the rate of being bedridden, vegetative, or dead was 2% in alert patients vs. 13% in confused patients, and 57% in obtunded or comatose patients,⁷⁵ p<0.0001, Cochran-Armitage trend test).

Summary of AVS

AVS is a common presentation to the ED, and potential etiologies range from benign to deadly. Initial evaluation should include history emphasizing timing of dizziness, prodromal and accompanying symptoms, and relevant past medical history (especially vascular risk factors). Examination should be focused on focal brainstem or cerebellar signs, gait/postural reflexes, and specific eye movement abnormalities. Imaging, if readily available, should include brain MRI with DWI, since CT scans are grossly inadequate for ischemic stroke detection in AVS. Multiple transient prodromal episodes suggest TIAs followed by stroke; auditory symptoms should spark concern for inner ear ischemia; pain or recent trauma should prompt consideration of vertebral dissection (especially in younger patients); and positional test results (Dix-Hallpike) should generally be disregarded.

The main differential diagnosis is between vestibular neuritis (P-AVS) and ischemic stroke (C-AVS) presenting a “pseudo-neuritis” clinical picture. Best evidence suggests that nearly two-thirds of C-AVS patients present *without* neurological signs that would be readily apparent to a non-neurologist (and roughly one-third present without signs readily apparent to a typical general neurologist). In such cases, a 3-component bedside oculomotor examination (H.I.N.T.S. to I.N.F.A.R.C.T.) can rule stroke in and out with high accuracy that probably exceeds that of urgent MRI with DWI. Training ED and other frontline physicians (as well as general neurologists) in the H.I.N.T.S. exam (or providing a device-based solution⁷⁶ as a substitute) may be necessary if potentially lethal causes of AVS are to be promptly and accurately diagnosed. Negative cranial MRI with DWI obtained in the first 48-72 hours after symptom onset should not be considered final in the presence of any dangerous “H.I.N.T.S.” or a high baseline prior probability for a vascular etiology of AVS.

H.I.N.T.S. Test Battery

URLs to Access Videos Demonstrating H.I.N.T.S. Findings

Published in association with...

Newman-Toker DE, Kattah JC, Alvernia JE, Wang DZ. Normal head impulse test differentiates acute cerebellar strokes from vestibular neuritis. *Neurology* 2008 Jun;70:2378-2385.
<http://stroke.ahajournals.org/cgi/content/full/STROKEAHA.109.551234/DC1>

Kattah JC, Talkad AV, Wang DZ, Hsieh YH, Newman-Toker DE. H.I.N.T.S. to diagnose stroke in the acute vestibular syndrome: three-step bedside oculomotor exam more sensitive than early MRI diffusion-weighted imaging. *Stroke* 2009 Nov;40(11):3504-3510.
http://www.neurology.org/cgi/content/full/70/24_Part_2/2378/DC1

Video 1a (abnormal HIT in a peripheral vestibulopathy)

- http://stroke.ahajournals.org/content/vol0/issue2009/images/data/STROKEAHA.109.551234/DC1/Kattah_Video1a_APV_HITabnormal.wmv
- http://www.neurology.org/content/vol70/issue24_Part_2/images/data/2378/DC1/Video_e-1.wmv

Video 1b (normal HIT in a central vestibulopathy caused by stroke)

- http://stroke.ahajournals.org/content/vol0/issue2009/images/data/STROKEAHA.109.551234/DC1/Kattah_Video1b_PICASTroke_HITnormal.wmv
- http://www.neurology.org/content/vol70/issue24_Part_2/images/data/2378/DC1/Video_e-2.wmv

Video 2a (direction-fixed nystagmus in a peripheral vestibulopathy)

- http://content.lib.utah.edu/u/?ehsl-dent_1

Video 2b (direction-changing nystagmus in a central vestibulopathy caused by stroke)

- http://content.lib.utah.edu/u/?ehsl-dent_2

Video 3 (skew deviation in a central vestibulopathy caused by stroke)

- http://stroke.ahajournals.org/content/vol0/issue2009/images/data/STROKEAHA.109.551234/DC1/Kattah_Video3_LatMedullaStroke_SkewAltCover.wmv

Additional video URLs (including demonstration videos on examination technique) may be found at the Neuro-Ophthalmology Virtual Education Library (N.O.V.E.L.) at <http://library.med.utah.edu/NOVEL> in the Newman-Toker collection (<http://library.med.utah.edu/NOVEL/Newman-Toker-New/>)

Descriptions adapted from Kattah JC, Talkad AV, Wang DZ, Hsieh YH, Newman-Toker DE. HINTS to diagnose stroke in the acute vestibular syndrome: three-step bedside oculomotor exam more sensitive than early MRI diffusion-weighted imaging. Stroke 2009 Nov;40(11):3504-3510.

Video 1 a/b. Horizontal head impulse test of vestibulo-ocular reflex function*

The horizontal head impulse test (h-HIT) of vestibulo-ocular reflex (VOR) function, as originally described, is a rapid, passive head rotation from a center to lateral (10-20 degrees) position as a subject fixates at a central target (e.g., the examiner's nose). A common adaptation of the h-HIT, used in this study, is to displace the head laterally first, then rotate the head back to the center position. Some examiners find the maneuver easier to conduct using this centripetal head motion, and the results are sometimes easier to interpret (since the globes end in the primary position in the orbit, rather than a somewhat lateral position). This approach also reduces any theoretical risk of vertebral artery injury with neck over-rotation by an overzealous, inexperienced examiner.

Although not originally validated with a lateral to center rotation, there is no known reason to believe that the vestibular system should respond differently, since the VOR response should be largely independent of the starting position of the head on the neck. During videotaping, the amplitude of the h-HIT head rotation was exaggerated to enhance its visibility. It is recommended that the test be performed clinically using a smaller-amplitude movement. For the practitioner, it is crucial to remember that for the test to work, the head rotation must be *passive* (i.e., conducted by the examiner), rather than *active* (i.e., deliberate head turn by the patient).

The *normal* VOR response to a rapid, passive head rotation as a subject fixates at a central target (e.g., the examiner's nose) is an equal and opposite eye movement that keeps the eyes stationary in space (i.e., still looking straight at the target) (*negative* h-HIT). An *abnormal* response occurs when the head is rapidly rotated toward the side of a vestibular lesion affecting the primary VOR pathway from the labyrinth to the lateral pons (not traversing the cerebellum). The loss of VOR input results in the subject's inability to maintain fixation during the head rotation, requiring a corrective gaze shift once the head stops moving (*positive* h-HIT). Note that in the patient with an acute vestibular syndrome, there is often spontaneous nystagmus. The refixation saccade of a positive h-HIT must be differentiated from the quick phases of any spontaneous nystagmus.

Video 1a: Shown is a typical acute peripheral vestibulopathy with left-beating, unidirectional nystagmus and abnormal rightward h-HIT. A 54-year-old man with a history of diabetes mellitus on diet-control presented with a 24-hour history of vertigo, falling to the right, nausea and vomiting, without auditory symptoms. He displayed a primary gaze, unidirectional, left-beating nystagmus that increased when looking in the direction of the nystagmus fast phase (i.e., in left gaze), and with fixation removal, both findings typical for a (right) peripheral vestibular lesion. He had an abnormal (positive) h-HIT to the right, and a normal (negative) h-HIT to the left, as anticipated. In the video, the rightward h-HIT is demonstrated first, with a pathologic, leftward, re-fixation saccade evident at the end of the head rotation, indicating a failure of the normal VOR response to keep the eyes steady on the target (i.e., the video camera lens). The leftward h-HIT is demonstrated next, with no refixation saccade evident at the end of the head rotation, indicating an intact VOR response. Brain MRI showed an incidental, 4 millimeter area of increased signal in the periventricular white matter, but no acute infarct by DWI. His clinical course was typical for vestibular neuritis. Note the subtle flattening of the left nasolabial fold apparent on the video was old (lifelong) and unrelated to his acute vestibular syndrome.

Video 1b: Shown is an acute peripheral vestibulopathy mimic, with pseudo-labyrinthine nystagmus, but normal h-HIT, suggesting stroke. A 71-year-old hypertensive man presented with a two-hour history of ataxia, nausea and vomiting without auditory symptoms. He fell to the left when standing. He had right-beating nystagmus in right gaze, but no nystagmus in primary or left gaze. Fixation removal showed a unidirectional, primary gaze, right-beating nystagmus that increased in right gaze, compatible with a peripheral-type nystagmus. However, the h-HIT was normal (negative), decreasing the likelihood of APV substantially, and suggesting a pseudo-labyrinthine presentation of stroke. The video, obtained 12 hours later, demonstrates saccadic rightward horizontal pursuits, but relatively smooth leftward pursuits. Fixation removal revealed a subtle oblique/down-beating component to the nystagmus, but the dominant vector remained horizontal and right-beating. Head CT scan showed a right inferior cerebellar stroke, associated with moderate mass effect and fourth ventricular compression. An open MRI obtained one month later showed an area of encephalomalacia involving the right inferior cerebellum, confirming the prior infarct evident by CT acutely.

Video 2 a/b. Examination for nystagmus in different gaze positions

Typical spontaneous nystagmus associated with acute peripheral vestibular lesions is dominantly horizontal in vector (often with a subtle torsional component but no apparent vertical vector) and generally beats in one direction, regardless of the eye position within the orbits. The nystagmus beats in the direction opposite to the side of a destructive peripheral vestibular lesion. The nystagmus is usually present in the primary position, increases in gaze towards the direction of the fast phase, and decreases or disappears completely in gaze towards the direction of the slow phase. This pattern of vestibular nystagmus is said to obey “Alexander’s law” (Video 2a – direction-fixed left-beating nystagmus in a patient with acute peripheral vestibulopathy).

With central causes of acute vestibular syndrome, it is not uncommon for the nystagmus to have a gaze-evoked component due to failure of gaze-holding circuits in the cerebellum or brainstem. In such instances, the horizontal nystagmus may reverse direction when the patient looks in the direction of the slow phase (Video 2b – direction-changing nystagmus; spontaneous left-beating nystagmus in primary and left gaze with reversal in right gaze in a patient with acute cerebellar infarction). This change in direction suggests a central lesion.

Video 3. Alternate cover test for vertical ocular misalignment (skew deviation)

With a patient fixating on a central target, the *normal* response to alternately occluding each eye (alternate cover test) is for the eyes to remain motionless, since the eyes have little or no propensity towards vertical misalignment (except in cases of congenital vertical strabismus or oculomotor disease). An *abnormal* response is indicated by the presence of a refixation saccade after transfer (removal) of the cover. A refixation saccade indicates either frank ocular misalignment (heterotropia) or a propensity for such misalignment when binocular cues to oculomotor fusion are eliminated (heterophoria). The degree of any such manifest or latent deviation can be measured using prismatic correction to neutralize the defect. Shown is a patient with acute vestibular syndrome due to lateral medullary infarction with an obvious vertical ocular misalignment of vestibular cause (i.e., skew deviation). The right eye is hypotropic (refixation saccade upward), while the left eye is hypertropic (refixation saccade downward), consistent with a right lateral medullary syndrome.

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