Acute Vestibular Syndrome:
Differentiating Stroke from Vestibular Neuritis

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How to Use This Review

This review examines acute vestibular syndrome, focusing on clinical differentiation of stroke from benign peripheral vestibular disease. Early recognition of posterior circulation stroke is critical for thrombolysis and thrombectomy eligibility.
The document follows a structured clinical format with numbered sections, integrated callout boxes for rapid reference, summary tables, and a references section. It is designed both as a learning resource and a quick-reference tool for practising clinicians.

 Key Point: *Foundational concepts and summary statements that anchor the core scientific content of each section.*

 Clinical Insight: Clinically relevant observations derived directly from the evidence — for direct application in assessment and diagnosis.

 Clinical Pearl: High-yield, memorable clinical points — the take-home messages most likely to influence management or examination performance.

Contents

I. Introduction

Acute vestibular syndrome (AVS) describes a discrete clinical presentation defined by sustained continuous vertigo or dizziness lasting more than 24 hours, accompanied by spontaneous nystagmus, nausea or vomiting, head-motion intolerance, and gait unsteadiness [4,9]. It accounts for roughly 3-5% of emergency department visits for dizziness and sits at the intersection of benign peripheral disease and potentially lethal posterior-circulation stroke [11,20].

The central diagnostic challenge in the ED is that vestibular neuritis and posterior-fossa stroke share an almost identical symptom profile at the bedside, yet their prognoses diverge sharply [1,3]. Missed cerebellar or brainstem infarction carries significant morbidity and mortality, while over-imaging of peripheral disease wastes resources and delays rehabilitation [7,19]. This review focuses on the structured bedside approach that allows frontline clinicians to stratify risk accurately before imaging.

 Clinical Pearl: One in ten AVS patients has a stroke. Early bedside differentiation guides urgent imaging and thrombolysis decisions.

II. Defining Acute Vestibular Syndrome

By consensus, AVS is diagnosed when three elements coexist: continuous vertigo or dizziness lasting more than 24 hours, spontaneous or gaze-evoked nystagmus, and gait unsteadiness or ataxia [4,9]. Additional supportive features include nausea, vomiting, and marked head-motion intolerance [6]. The temporal profile distinguishes AVS from episodic vestibular syndromes such as BPPV or vestibular migraine, which typically resolve between attacks [15].

Identifying the syndrome is only the first step - every AVS presentation mandates active exclusion of a central cause before reassurance or discharge [1,11]. The following sections describe the epidemiology of stroke within this group, the typical features of peripheral and central causes, and the structured bedside examination used to differentiate them.

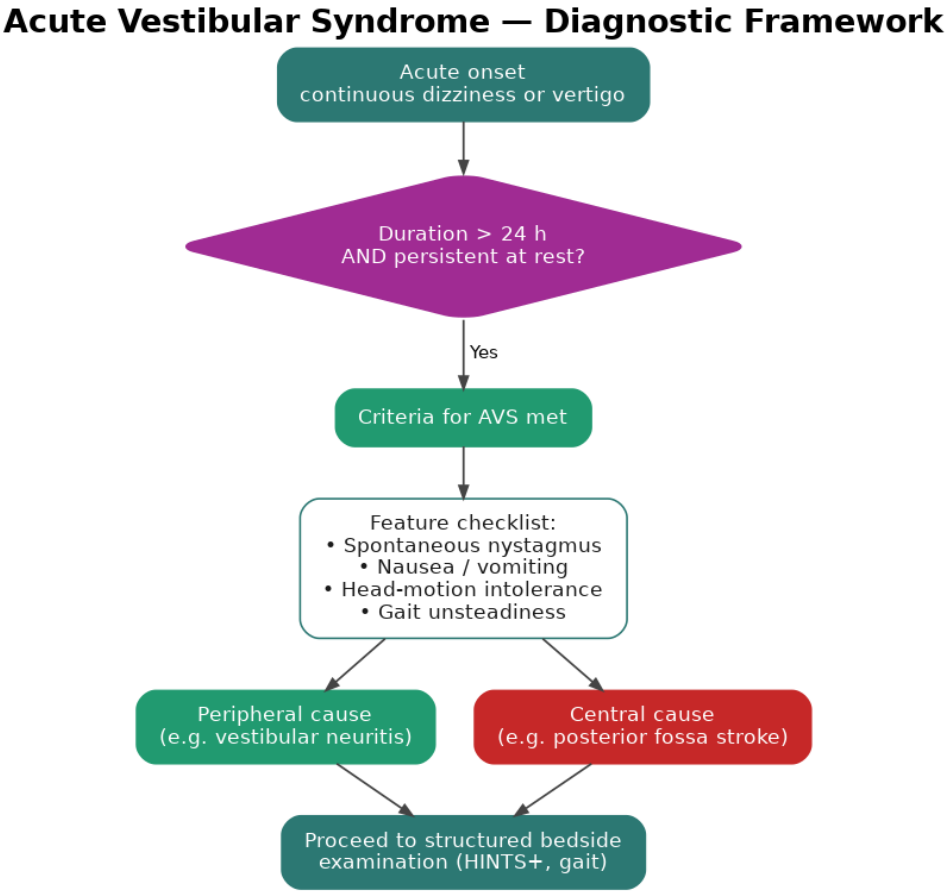

Figure 1. Diagnostic framework for Acute Vestibular Syndrome in the ED.

The 24-hour symptom threshold is not arbitrary: it excludes brief positional paroxysms (BPPV), short pre-syncopal episodes, and migrainous attacks that typically resolve within hours. Patients who fit AVS are therefore a high-stakes cohort — every presentation must be triaged as potential posterior-circulation stroke until proven otherwise [2,5,12].

Epidemiologically, AVS accounts for roughly 4% of all emergency presentations for dizziness, but contributes disproportionately to missed-stroke litigation and 30-day readmission [2,4]. The mean age in posterior-stroke AVS is younger than in anterior-circulation events, often 50–65, and traditional vascular risk factors are present in fewer than half of cases [3,11].

 Key Point: *AVS = sustained vertigo + nystagmus + gait instability. This triad mandates stroke exclusion.*

III. Stroke Prevalence in AVS

Prospective emergency-department cohorts have reported stroke in 8-20% of patients meeting AVS criteria, with posterior-circulation territories predominating [11,13]. Within the stroke subgroup, the anterior inferior cerebellar artery (AICA) accounts for approximately 40-50% of lesions, the posterior inferior cerebellar artery (PICA) for 30-40%, and basilar or superior cerebellar artery events for the remainder [3,19]. Vertebrobasilar dissection is an important cause in younger patients without vascular risk factors [13].

Traditional vascular risk factors - age over 60, hypertension, diabetes, atrial fibrillation, and prior stroke - shift pre-test probability upward but do not exclude stroke in their absence [11,12]. A structured bedside approach is therefore mandatory in every AVS patient regardless of risk profile [1].

Table 1. Stroke Risk in AVS

Risk Profile	Stroke Prevalence
Age <50, no vascular disease	2–5%
Age 50–60, 1+ risk factor	8–12%
Age >60 or multiple risk factors	15–20%

However, absence of risk factors does NOT exclude stroke. All AVS patients require rapid bedside assessment.

IV. Vestibular Neuritis

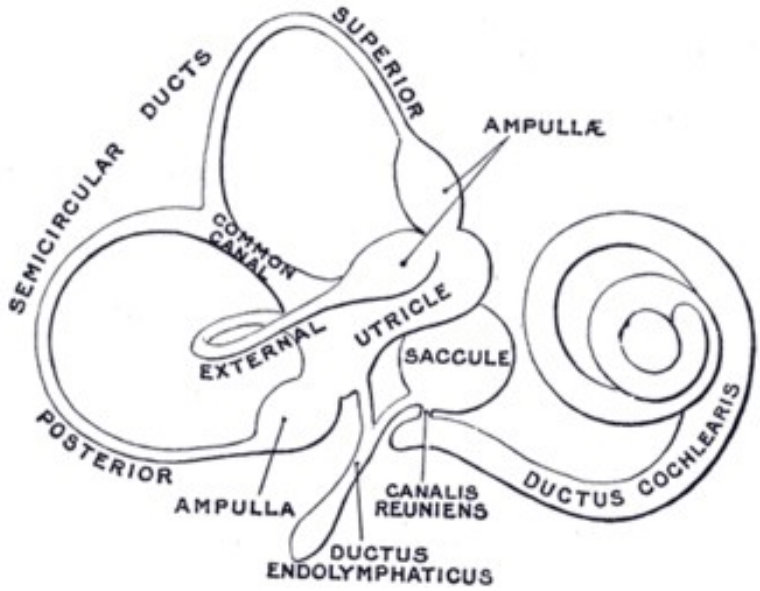

Figure A. Membranous labyrinth — three semicircular ducts with ampullae, utricle, saccule, and cochlear duct (ductus cochlearis). The peripheral sensory organ involved in vestibular neuritis.
Source: Wikimedia Commons

Vestibular neuritis is thought to result from acute inflammation or reactivation of latent viral infection within the vestibular nerve [5,10]. It typically presents with sudden vertigo, nausea, vomiting, and horizontal-torsional spontaneous nystagmus beating away from the affected side [4,16]. Symptoms usually peak within 24 hours and settle gradually over days to weeks as central compensation develops [10].
Hearing is preserved in pure vestibular neuritis; when acute hearing loss accompanies the vertigo, AICA-territory stroke or labyrinthitis must be considered [3,7]. The head impulse test is typically abnormal on the side of the lesion, and the patient can usually stand with support - distinguishing features from central causes [1,16].

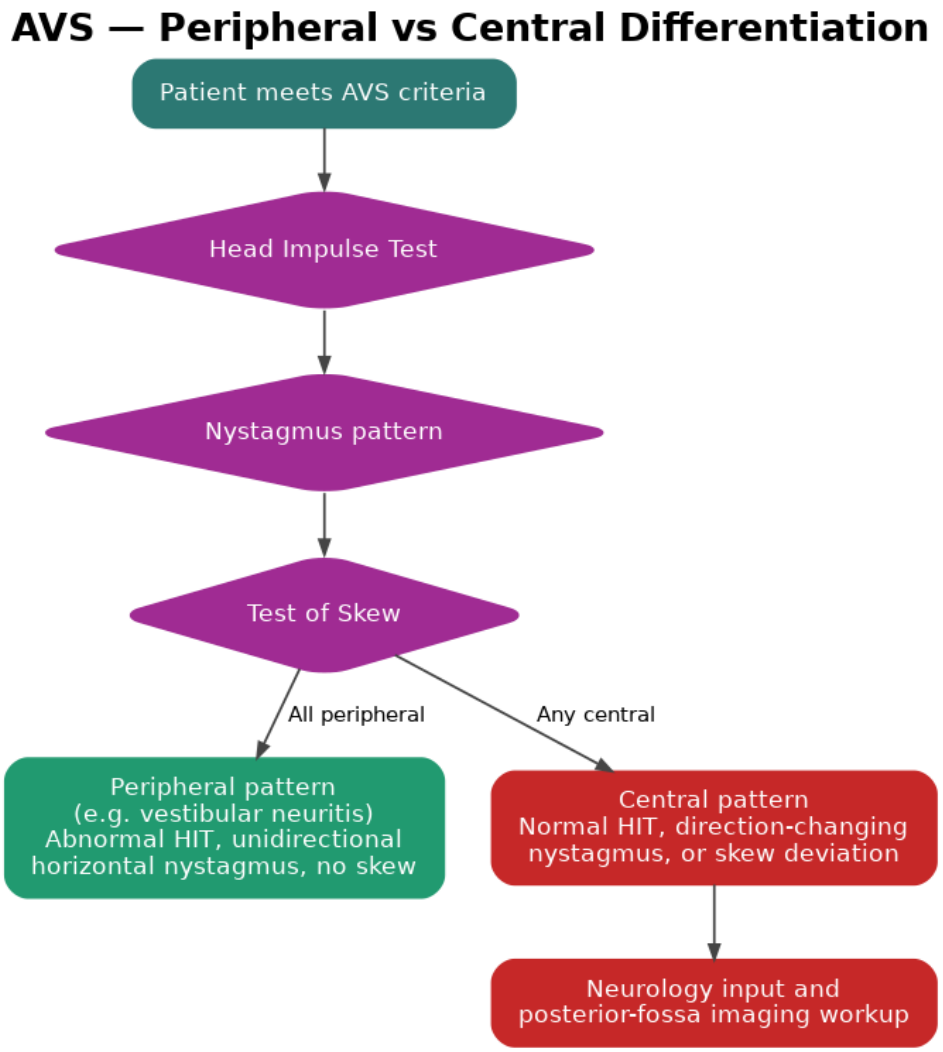

Figure 2. Bedside differentiation of peripheral vs central pattern in AVS.

Pathophysiologically, vestibular neuritis is thought to be a reactivation of latent herpes simplex virus type 1 within the vestibular ganglion, producing inflammation and axonal injury of the superior or inferior vestibular nerve [9]. The superior division is affected in roughly 90% of cases, sparing the posterior canal and saccule — a useful confirmatory finding on bedside testing.

Recovery is typically gradual over 4–8 weeks, driven by central vestibular compensation rather than peripheral reinnervation [9,15]. Persistent symptoms beyond 3 months should prompt reconsideration of the diagnosis or referral for vestibular rehabilitation, where supervised gaze-stabilisation and habituation exercises substantially reduce chronic dizziness handicap [19].

 Clinical Pearl: Vestibular neuritis: peripheral vestibular disease with excellent prognosis. Recovery occurs with vestibular compensation.

V. Posterior Fossa Stroke Mimicking Neuritis

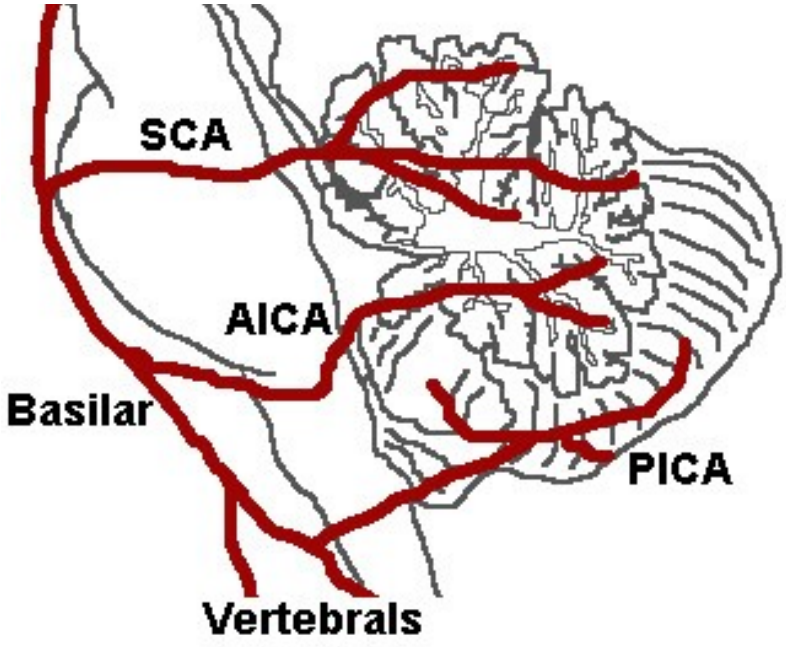

Figure B. Posterior circulation arterial supply — vertebral arteries, basilar artery, and the three cerebellar branches (PICA, AICA, SCA) supplying brainstem and cerebellum.
Source: Wikimedia Commons

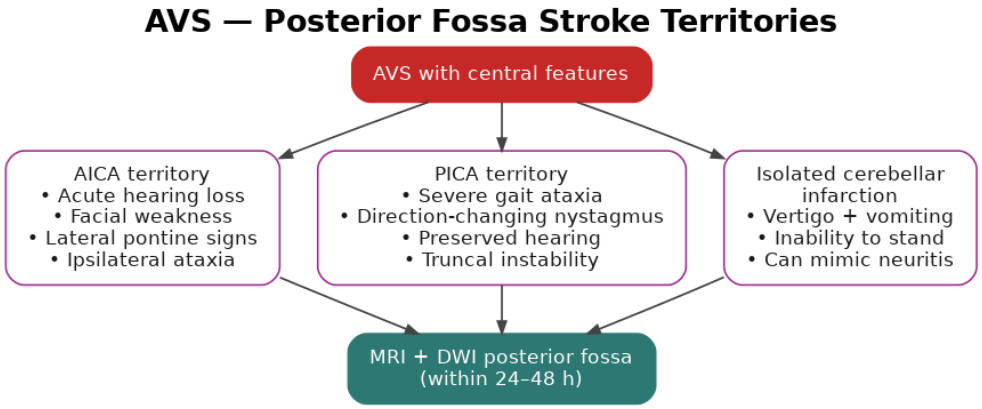

Figure 3. Posterior-fossa stroke territories relevant to AVS: AICA, PICA, and isolated cerebellar infarction.

AICA Territory

AICA-territory infarction classically combines acute vertigo with ipsilateral sensorineural hearing loss owing to shared blood supply from the labyrinthine artery [3,7]. Additional features may include facial weakness, Horner syndrome, and lateral pontine signs such as contralateral spinothalamic sensory loss [19].

Because hearing loss is a pathognomonic flag for AICA stroke in the context of AVS, a bedside whispered-voice or finger-rub assessment should be incorporated into every acute vestibular examination [7]. Sudden sensorineural hearing loss accompanying AVS should prompt urgent MRI with diffusion-weighted imaging [19].

⚠ **Important:** Acute hearing loss + acute vertigo in the same ear = presumptive AICA stroke; obtain urgent MRI.

PICA Territory

PICA-territory infarction frequently produces the lateral medullary (Wallenberg) syndrome with ipsilateral facial sensory loss, Horner syndrome, contralateral body analgesia, dysphagia, dysarthria, and vertical or torsional nystagmus [3,13]. Pure vertigo with severe truncal ataxia and inability to stand can also occur with isolated PICA cerebellar infarction [8].

Direction-changing nystagmus, skew deviation, and a normal head impulse test are hallmark central findings in this territory and should not be attributed to peripheral disease [1,2].

Cerebellar Infarction

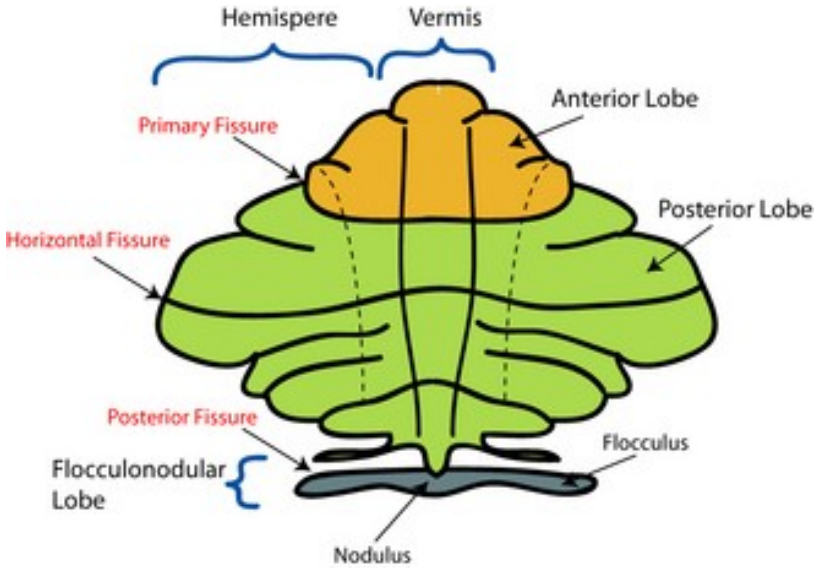

Figure C. Cerebellar anatomy (unfolded) — hemispheres, vermis, and the flocculonodular lobe (flocculus and nodulus) that constitute the vestibulocerebellum.
Source: Wikimedia Commons

Isolated cerebellar infarction can present with vertigo and vomiting in the apparent absence of focal neurology, closely mimicking vestibular neuritis [3,7]. Inability to sit or stand unaided - so-called astasia - carries a strong positive predictive value for central cause and has been shown in prospective data to predict stroke in AVS better than most individual HINTS components [8].

Additional bedside red flags in this group include direction-changing nystagmus, skew deviation, severe occipital or neck pain, and persistent truncal ataxia disproportionate to the limb examination [1,7].

VI. Red Flags for Stroke

Bedside red flags that raise the probability of central cause include new focal neurological deficit, acute hearing loss, severe truncal ataxia with inability to stand unaided, sudden severe headache or neck pain, direction-changing nystagmus, vertical or skew deviation, and a normal head impulse test in a patient with AVS [1,8,19].

Age over 60, vascular risk factors, and a history of prior stroke or TIA further raise the pre-test probability and should lower the threshold for posterior-fossa imaging [11,12]. Any red flag in an AVS patient warrants neurology input, MRI with diffusion-weighted imaging, and disposition to inpatient assessment [1,7].

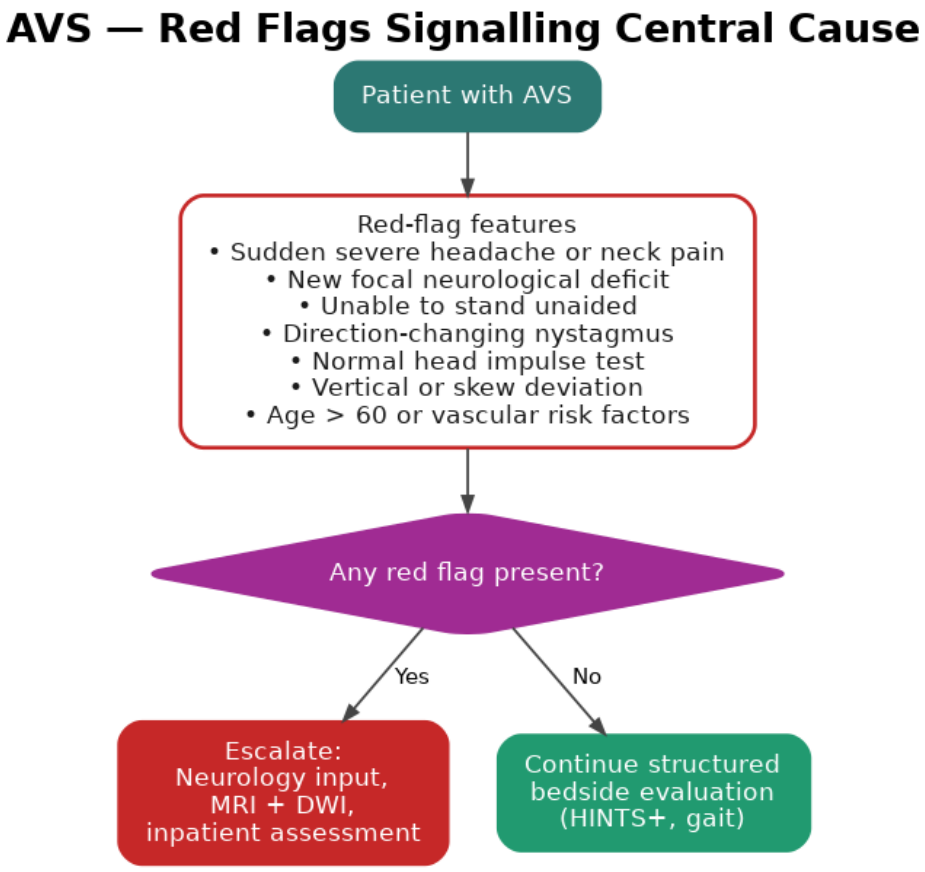

Figure 4. Bedside red-flag checklist signalling central cause in AVS.

Practical red flags in AVS include: age over 60, vascular risk factors (hypertension, diabetes, smoking, atrial fibrillation), inability to stand or walk unsupported, abnormal HINTS (any one component central), focal neurological signs (diplopia, dysarthria, dysphagia, hemisensory loss), and severe headache [3,4,11,12].

⚠ **Important:** Absence of red flags does NOT exclude stroke. Use HINTS and imaging liberally.

VII. Bedside Differentiation

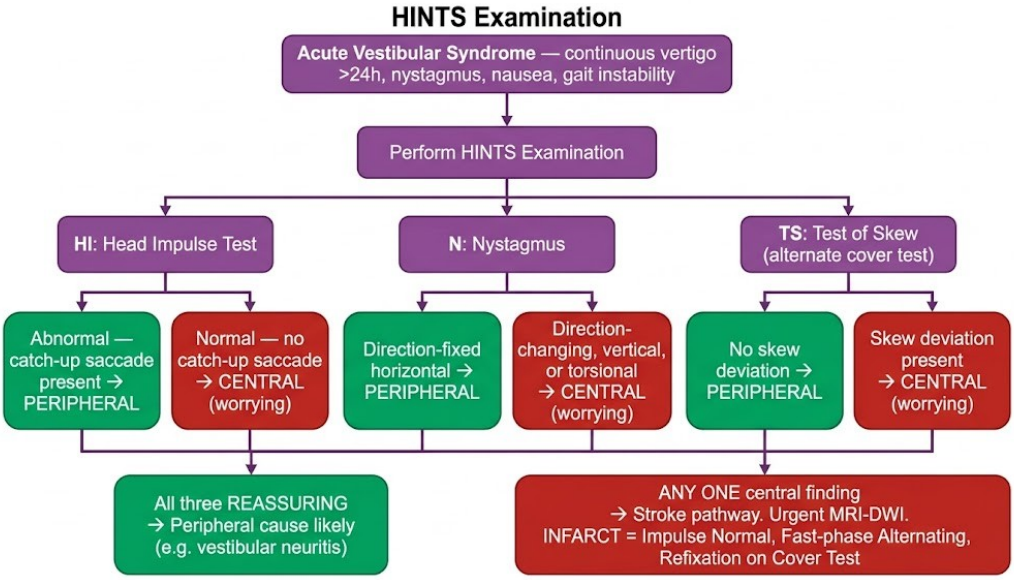

Figure 5. HINTS+ and gait examination sequence for AVS.

HINTS Examination

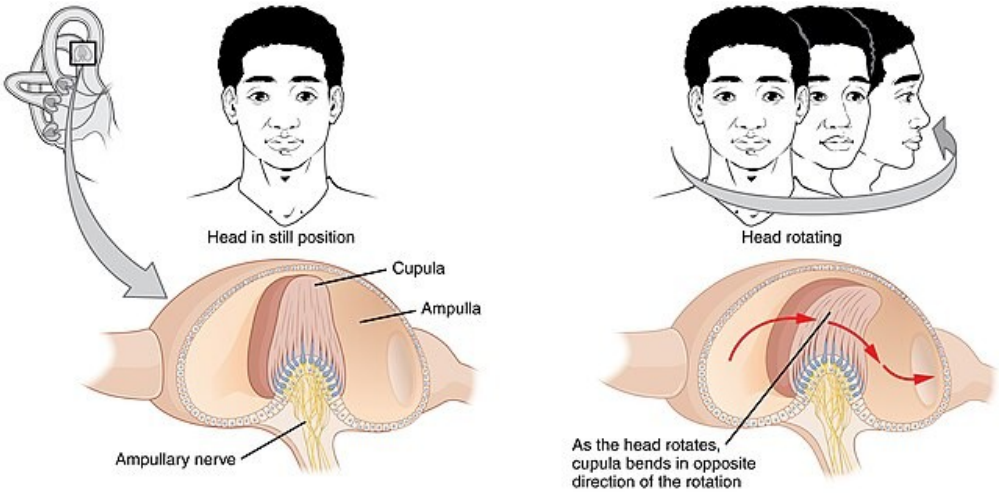

Figure D. Peripheral vestibular physiology — with head rotation, endolymph inertia deflects the cupula in the opposite direction, activating ampullary nerve afferents. This drives the vestibulo-ocular reflex tested by HINTS.
Source: Wikimedia Commons

The HINTS battery combines the Head Impulse Test, assessment of Nystagmus pattern, and Test of Skew and has been validated with pooled sensitivity and specificity exceeding 95% for central cause when performed within the AVS window by trained clinicians [1,2]. A peripheral pattern requires all three to be peripheral - an abnormal HIT toward the lesion, unidirectional horizontal nystagmus, and absence of skew deviation [1,16].

Any central sign on any component of HINTS - normal HIT, direction-changing or vertical nystagmus, or skew deviation - should be treated as a positive central screen and escalated for imaging and neurology review [1,2,17]. Adding a bedside hearing test ('HINTS+') improves sensitivity for AICA-territory stroke [7].

Gait Assessment

Gait examination complements the HINTS battery and should be part of every AVS evaluation [8]. Peripheral lesions typically cause lateropulsion toward the affected side with preserved ability to stand

unaided, whereas central lesions cause midline truncal ataxia, veering in multiple directions, or complete inability to sit or stand [1,8].

Severe truncal ataxia with astasia is one of the strongest single bedside predictors of stroke in AVS and should prompt urgent imaging regardless of other findings [8].

When bedside differentiation is equivocal — for example a corrective saccade that is small or asymmetric, or nystagmus that does not fit a clear pattern — the clinician should default to imaging and admit for observation [2,7]. False-negative bedside testing in the hands of non-experts is well documented and carries a >10% missed-stroke rate [4,11].

 Key Point: *Veering to one side = peripheral. Midline ataxia and inability to sit upright = cerebellar disease.*

VIII. Imaging Considerations

Non-contrast CT of the brain is insensitive for acute posterior-fossa ischaemia, with reported sensitivities of only 10-30% in the first 24 hours and a high false-negative rate for cerebellar and brainstem infarction [7,19]. A normal CT therefore does not exclude stroke in AVS and should not be used as the sole imaging strategy [19].

MRI with diffusion-weighted imaging is the gold standard, with sensitivity of approximately 90-95% for acute infarction, although false-negative DWI within the first 24-48 hours is well described in small posterior-fossa lesions [7,19]. Repeat MRI at 72 hours should be considered if the initial scan is negative and clinical concern persists [19].

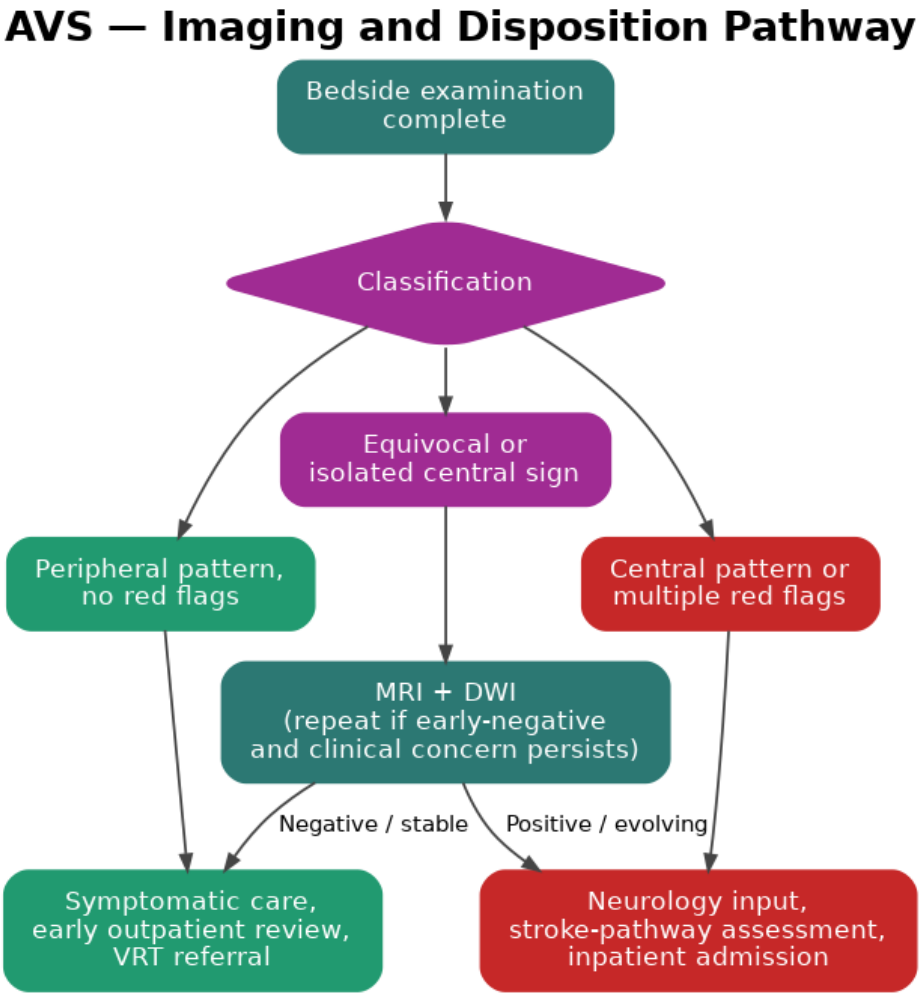

Figure 6. Imaging and disposition pathway for AVS.

MRI with diffusion-weighted imaging is the gold standard for confirming or excluding posterior-circulation infarction in AVS, with sensitivity approaching 95% beyond 24 hours but as low as 50–70% within the first 6–12 hours [13,15]. Repeat MRI at 48–72 hours is justified when initial imaging is negative but clinical suspicion remains high.

Computed tomography brain has limited utility — sensitivity for acute posterior-fossa ischaemia is below 30% — and should not be used to exclude stroke in AVS [14]. CT angiography is, however, a useful adjunct to identify large-vessel occlusion or dissection while awaiting MRI [11].

⚠ **Important:** Normal CT brain does NOT exclude posterior circulation stroke. MRI DWI is required for definitive exclusion.

IX. Management Pathways

A fully peripheral HINTS+ pattern in an AVS patient with preserved gait, no red flags, and reassuring vascular profile supports a working diagnosis of vestibular neuritis [1,10]. These patients can be managed with symptomatic care, early vestibular rehabilitation referral, and outpatient follow-up [10] [19].

Any central sign, inability to stand unaided, or combination of red flags should trigger urgent MRI with diffusion-weighted imaging, neurology input, and inpatient pathway assessment [1,7,19]. Uncertainty - an equivocal

examination or an isolated red flag - should be resolved with imaging and specialist review rather than empirical discharge [11,20].

Disposition planning should integrate symptom trajectory, examination findings, imaging results and patient social context [4,18]. Patients with HINTS-peripheral signs and normal MRI may be safely discharged with vestibular suppressant for 48–72 hours, vestibular rehabilitation referral, and clear return precautions for new neurological symptoms or worsening gait [9,18] [19].

Patients with HINTS-central, evolving deficits or imaging-confirmed stroke require admission to a stroke or high-dependency unit, with neurology and neurosurgery input as appropriate [3,4,11]. Early access to thrombolysis or thrombectomy improves functional outcomes substantially in posterior-circulation stroke [18,20].

 Clinical Insight: The "golden hours" of acute stroke are real. Early recognition enables thrombolysis and thrombectomy.

X. Conclusions

AVS presentations sit on a spectrum from benign peripheral vestibular disease to posterior-circulation stroke, and at the bedside these causes can appear almost identical [1,4]. A systematic approach combining focused history, the HINTS+ battery, careful gait assessment, and a low threshold for MRI in equivocal cases allows emergency clinicians to stratify risk reliably before imaging [1,2,8,19].

Applied consistently, this framework improves detection of central causes, reduces unnecessary investigation of peripheral disease, and supports appropriate disposition - discharge with vestibular rehabilitation for peripheral cases and inpatient stroke-pathway assessment for central cases [10,11,19] [19].

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