Brainstem Stroke and Transient Ischaemic Attack:

A Vestibular Physician's Deep Review of Vascular Vertigo, Localisation, and Acute Management

Vestibular Medicine for Vestibular Physicians

Central Vestibular Pathology — Module 3.4

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How to Use This Review

This literature review forms part of the Vestibular Medicine for Vestibular Physicians series published by the Australian Dizziness Clinics Education Hub. It is written for vestibular physicians, neuro-otologists, advanced ENT trainees, and vestibular physiotherapists working at the deep end of central vestibular practice, where a working command of mechanism, localisation, and atypical presentations is expected rather than optional.

The review is dense by design — intended as a 30–40 minute deep read or a desktop reference. It is supported by an A4 clinician cheat sheet, short-form clinician videos, audio episodes, and a patient information leaflet within the same Education Hub module.

Callout Box Guide

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

□ **Clinical Insight:** Clinically relevant observations for direct application in assessment and management.

□ **Clinical Pearl:** High-yield memorable clinical points — the take-home messages most likely to change practice.

□ **Important:** Red flags, atypical presentations, and critical safety points requiring escalation or imaging.

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I. Introduction and Epidemiology

Dizziness and vertigo account for roughly 4% of emergency-department presentations, and a clinically important minority of these are caused by posterior-circulation ischaemia rather than benign inner-ear disease [7,8]. Among patients presenting with an acute vestibular syndrome — continuous vertigo with nystagmus, nausea, head-motion intolerance and unsteady gait lasting days — approximately one quarter are due to brainstem or cerebellar stroke [1,8,10]. The stakes are high in both directions: missing a vertebrobasilar stroke exposes the patient to a preventable, potentially fatal posterior-fossa catastrophe, while over-investigating benign vestibular neuritis wastes imaging, beds and reassurance [2,18].

Posterior-circulation stroke is disproportionately misdiagnosed. Population data show that patients with cerebrovascular events presenting with dizziness are misdiagnosed at initial contact far more often than those presenting with classic hemispheric deficits, and that isolated dizziness is the single most common 'benign' label later overturned by stroke [7,18,34]. The reasons are structural: the symptom is non-specific, the examination that discriminates is unfamiliar to many generalists, and early imaging is falsely reassuring [2,26]. For the vestibular physician, the diagnostic responsibility is therefore twofold — to recognise the small central stroke hiding behind an apparently peripheral picture, and to avoid mislabelling true neuritis as stroke [1,34].

Vascular vertigo is also age- and risk-factor dependent. The probability that an acute vestibular syndrome is vascular rises steeply with age, vascular risk burden, and the presence of any subtle central sign [1,8,36]. In older patients with multiple risk factors, the pretest probability of stroke in continuous vertigo with nystagmus may approach that of vestibular neuritis, which is why a structured oculomotor examination — not symptom quality — must drive triage [2,8,10].

Table 1. Brainstem and cerebellar stroke in the dizzy patient — epidemiology at a glance [1,7,8,11,18].

Parameter	Approximate figure / observation
Dizziness/vertigo as proportion of ED visits	About 3–4% of all emergency presentations
Stroke among acute vestibular syndrome (AVS)	Roughly 25% of high-risk AVS presentations
Isolated cerebellar infarction mimicking neuritis	Around 11% of isolated-vertigo cerebellar strokes
Most common territory in vertigo-predominant stroke	PICA (lateral medulla / inferior cerebellum)
Initial misdiagnosis rate, posterior-circulation stroke	Substantially higher than anterior-circulation stroke

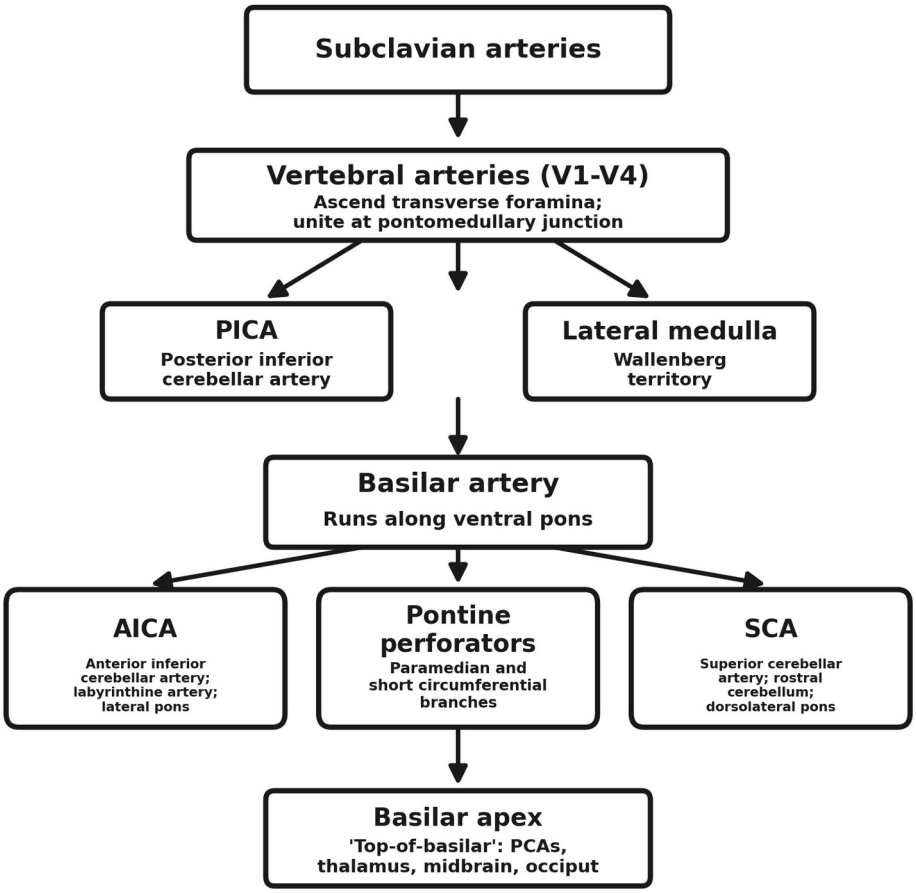
The clinical and economic stakes are considerable. Misdiagnosed posterior-circulation stroke is a leading source of serious, preventable harm from diagnostic error in emergency medicine, and the downstream costs — recurrent stroke, disability, and litigation — dwarf the cost of getting the assessment right at first contact [18,34]. At the same time, indiscriminate neuroimaging of every dizzy patient is neither affordable nor accurate, given the false-negative rate of early scanning [2,7]. A diagnostic strategy built on examination rather than imaging is therefore both safer and more efficient, which is the central argument for vestibular-physician involvement in acute dizziness pathways [3,34].

Diagnostic-error research frames the problem in two complementary ways. Posterior-circulation strokes that present atypically — as isolated vertigo, isolated hearing loss, or a syndrome indistinguishable from neuritis at first glance — are the 'chameleons' that evade recognition [2,34]. Conversely, benign vestibular disorders that prompt unnecessary alarm are the 'mimics' that drive over-investigation [3,34]. The vestibular physician is uniquely placed to reduce both, because the discriminating skill set — interpretation of the vestibulo-ocular reflex, nystagmus and ocular alignment — is core vestibular expertise rather than incidental neurology [1,30]. Embedding that expertise at the point of triage, whether in person or by tele-assessment, is among the highest-value contributions vestibular medicine can make to acute care [34,42].

□ **Key Point:** Around one in four high-risk acute vestibular syndromes is a stroke. Symptom quality does not separate vascular from peripheral causes — the discriminator is a structured oculomotor examination interpreted in the light of timing, triggers, and vascular risk.

II. Vascular Anatomy and Mechanisms of Posterior-Circulation Ischaemia

The vestibular structures are supplied almost entirely by the posterior (vertebrobasilar) circulation, which is why ischaemia here so readily produces vertigo [9,40]. The paired vertebral arteries ascend through the cervical transverse foramina, give off the posterior inferior cerebellar arteries (PICA), and unite at the pontomedullary junction to form the basilar artery [9,15]. The basilar gives rise to the anterior inferior cerebellar arteries (AICA), numerous pontine perforators, and the superior cerebellar arteries (SCA), before terminating at the basilar apex in the posterior cerebral arteries [9,13]. Each territory has a characteristic audiovestibular signature, summarised in Figure 1 and expanded in Section III [5,15].



The labyrinthine (internal auditory) artery usually arises from AICA and is an end-artery to the cochlea and vestibular labyrinth - with no collateral supply, AICA occlusion infarcts the inner ear.

Figure 1. The posterior circulation and its vestibular supply.
Source: Adapted from Savitz and Caplan [9] and Kim and Lee [5].

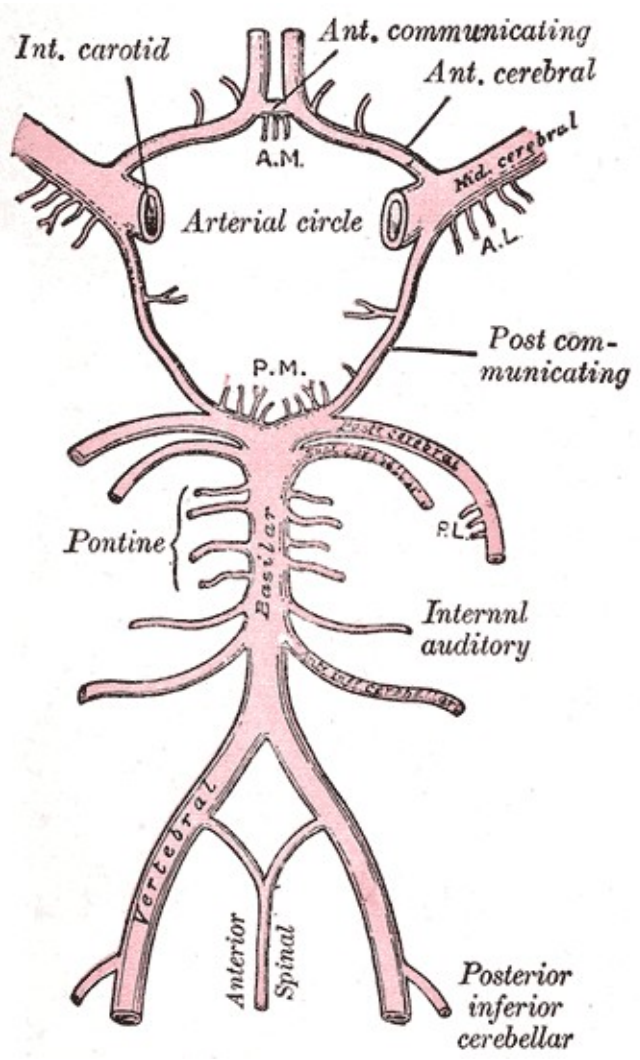


Figure 2. Classical anatomy of the arterial circle and vertebrobasilar system — the basilar, pontine and internal auditory (labyrinthine) branches and PICA.

Source: Gray's Anatomy of the Human Body (1918), public domain.

The clinically pivotal vessel for the inner ear is the labyrinthine (internal auditory) artery, which in most people arises from AICA [5,14,24]. It is a functional end-artery with negligible collateral supply, so AICA occlusion can infarct the cochlea and vestibular labyrinth as well as the lateral pons and cerebellum — producing the combined auditory and vestibular loss that distinguishes AICA stroke from purely central events [4,5,23]. This anatomy explains why acute hearing loss accompanying vertigo should raise, not lower, suspicion of stroke in a patient with vascular risk factors [4,24].

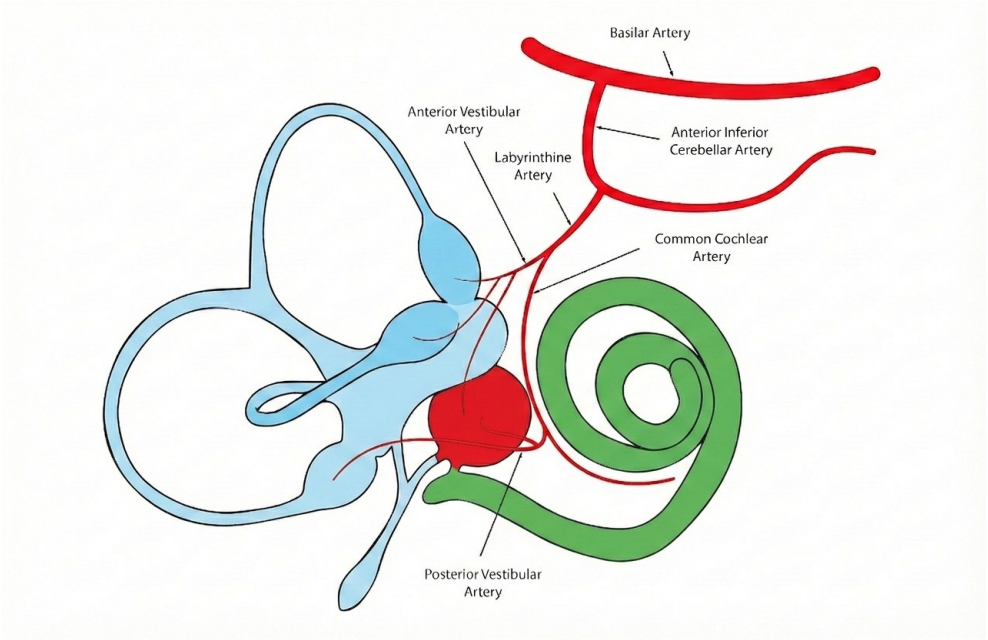


Figure 3. Arterial supply of the labyrinth — the labyrinthine artery arises from AICA and divides into cochlear and vestibular branches, an end-arterial supply with no collateral.
Source: Australian Dizziness Clinics anatomy collection.

Mechanisms of ischaemia

Posterior-circulation ischaemia arises through the same mechanisms as anterior-circulation stroke, but with a different distribution [9,40]. Large-artery atherosclerosis of the vertebral or basilar arteries is the dominant mechanism, frequently at the vertebral origin or the intracranial vertebral and basilar segments [12,40]. Artery-to-artery and cardiac embolism account for many cerebellar and basilar-apex infarcts [12,13]. Small-vessel (lacunar) disease produces discrete pontine and medullary syndromes [31,32]. Vertebral artery dissection — spontaneous or following neck trauma or manipulation — is a disproportionately common cause in younger patients and must not be overlooked [2,40].

Mechanism matters because it determines secondary prevention and recurrence risk. Nonlacunar mechanisms, including vertebral occlusion and dissection, are over-represented even among very small vestibular infarcts, so a small lesion does not imply a benign cause or low recurrence risk [2,40]. Defining the mechanism — through vessel imaging and a cardioembolic work-up — is therefore integral to management, not an academic afterthought [40,28].

Variant anatomy and collateral supply

Posterior-circulation anatomy is unusually variable, and the variants are clinically relevant [9,12]. One vertebral artery is hypoplastic or terminates in PICA in a substantial minority of people, so a single small embolus or dissection can have a disproportionate territorial effect [9,40]. The labyrinthine artery occasionally arises directly from the basilar artery rather than AICA, and the relative contributions of PICA and AICA to the inferior cerebellum vary reciprocally between individuals [5,15]. These variations explain why territory-to-syndrome maps are guides rather than rules, and why a given clinical picture may not respect the textbook boundaries [12,15].

Collateral supply also shapes outcome. The posterior communicating arteries and leptomeningeal collaterals can partly protect the basilar-apex and cerebellar territories, so the same arterial occlusion may produce a devastating infarct in one patient and a minor deficit in another [9,40]. For the vestibular nucleus complex, which sits in a watershed served by perforators from several parent vessels, small lesions can selectively damage central vestibular projections while sparing adjacent structures — the substrate for isolated central vertigo [31,47].

□ **Clinical Insight:** Acute hearing loss with vertigo in a patient with vascular risk factors points toward AICA territory infarction, because the labyrinthine artery is an end-branch of AICA. Treat sudden audiovestibular loss as a possible stroke prodrome, not a reassuring 'inner-ear' sign.

III. Clinical Syndromes by Vascular Territory

Posterior-circulation strokes produce recognisable syndromes that map onto the vascular territories of Figure 1 [9,15]. The vestibular physician should hold these patterns in mind because the accompanying signs — when sought — convert an apparently isolated vertigo into a localisable stroke [11,32]. Figure 6 and Table 2 consolidate the territory-to-syndrome relationships [5,15].

Lateral medullary (Wallenberg) syndrome — PICA / vertebral artery

Lateral medullary infarction, usually from vertebral-artery or PICA disease, is the classic vascular vertigo syndrome [9,15]. Vertigo and nystagmus accompany ipsilateral Horner syndrome, ipsilateral facial and contralateral body pain-and-temperature loss, dysphagia and hoarseness, limb ataxia, and a striking tendency for the body and eyes to be pulled toward the lesion (ipsipulsion and ocular lateropulsion) [15,32]. Many of these signs are subtle and missed unless specifically tested; a deliberate search for Horner syndrome, crossed sensory loss and lateropulsion is the highest-yield manoeuvre when a medullary stroke is suspected [2,32].

AICA syndrome — lateral pons and labyrinth

AICA infarction is the syndrome the vestibular physician is most likely to mistake for peripheral disease [4,5]. Because the labyrinthine artery arises from AICA, the picture combines acute vertigo with ipsilateral hearing loss and, often, facial weakness, facial sensory loss, and ataxia [4,5,23]. The audiovestibular loss may even precede the brainstem signs by hours to days, offering a window in which the stroke is mislabelled as labyrinthitis or sudden sensorineural hearing loss [4,24]. In a patient with vascular risk factors, sudden combined hearing and vestibular loss should prompt urgent imaging and vessel assessment rather than reassurance [5,14].

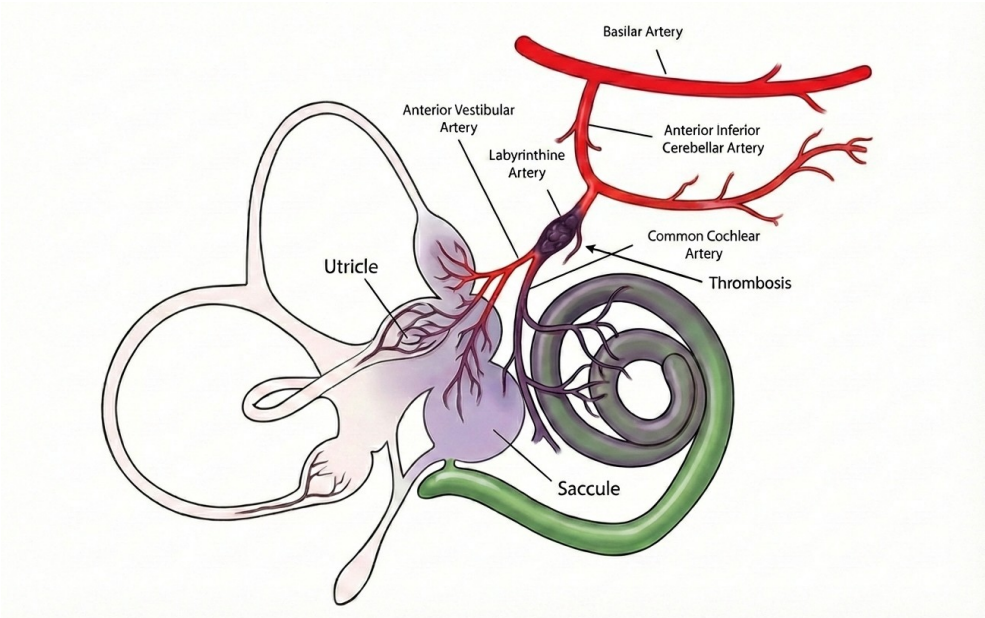


Figure 4. Thrombosis of the labyrinthine/common cochlear artery — the vascular basis of acute audiovestibular loss in AICA-territory ischaemia.

Source: Australian Dizziness Clinics anatomy collection.

Cerebellar infarction — PICA and SCA

Cerebellar infarction is the most dangerous mimic of vestibular neuritis [11,41]. Around 11% of isolated cerebellar strokes — typically in the medial branch of PICA — present with vertigo, nystagmus and gait ataxia and no other localising sign, closely imitating neuritis [11]. The discriminators are a normal head-impulse test, direction-changing gaze-evoked nystagmus, and gait ataxia out of proportion to the vertigo [1,11,35]. Superior cerebellar artery infarcts more often produce dysarthria and prominent gait ataxia,

with vertigo reported in up to half of cases [6,15]. The clinical urgency is that large PICA or SCA infarcts can swell over 24–72 hours and compress the fourth ventricle and brainstem [41].

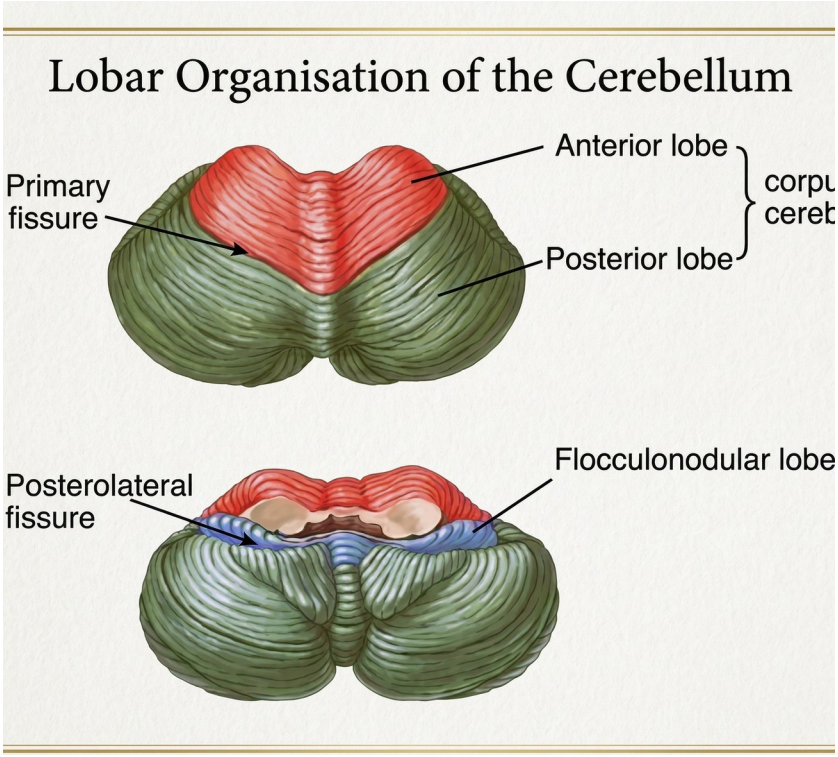


Figure 5. External (lobar) organisation of the cerebellum — the structure infarcted in PICA and SCA territory strokes.

Source: OpenStax, Anatomy & Physiology (Rice University). Licensed under CC BY 4.0.

Basilar and 'top-of-the-basilar' syndromes

Basilar-artery occlusion is a neurological emergency that may open with vertigo before progressing to depressed consciousness, quadriparesis, cranial-nerve palsies and locked-in syndrome [13,40]. 'Top-of-the-basilar' embolic syndrome combines vertigo with vertical gaze palsy, pupillary abnormalities, somnolence, and visual-field or behavioural disturbance from thalamic, midbrain and occipital ischaemia [13]. Fluctuating or stuttering brainstem symptoms in this setting are ominous and demand emergent vessel imaging and reperfusion assessment [13,40].

Isolated vascular vertigo and central vestibular-nucleus infarction

A subset of posterior-circulation strokes presents as truly isolated vertigo, without any accompanying deficit [11,31,33]. Small infarcts of the vestibular nucleus, inferior cerebellar peduncle or nodulus can reproduce a peripheral-appearing acute vestibular syndrome, including an apparently abnormal head-impulse test, because they damage central vestibular pathways [31,47]. These are precisely the lesions that early diffusion-weighted imaging misses, and they reinforce the rule that bedside oculomotor testing — not symptom isolation — governs safety [2,31].

Pontine and less common syndromes

Pontine infarction adds further patterns the vestibular physician should recognise [12,32]. Paramedian and tegmental pontine lesions can cause vertigo with internuclear ophthalmoplegia, one-and-a-half syndrome, abducens palsy, or facial weakness, reflecting damage to the medial longitudinal fasciculus and adjacent oculomotor structures [15,32]. Isolated infarction of the vestibular nuclei or the root entry zone of the eighth nerve can reproduce a peripheral-appearing syndrome, including an abnormal head-impulse test, which is precisely why no single bedside sign is infallible and the whole pattern must be weighed [31,47]. Medial medullary and purely cerebellar-peduncle lesions occasionally present with vertigo and gait disturbance as the dominant complaint, again with few localising signs [11,32].

The eight subgroups of AICA infarction

AICA territory infarction has been subclassified by the pattern of audiovestibular involvement, with at least eight recognised subgroups depending on whether the cochlea, the vestibular labyrinth, the eighth nerve, or central structures bear the brunt [5]. The commonest pattern is combined auditory and vestibular loss, but isolated cochlear, isolated vestibular, and various combinations with brainstem and cerebellar signs all occur [5,23]. The practical message is not the taxonomy itself but its implication: any combination of acute auditory and vestibular symptoms in a vascular-risk patient can be AICA ischaemia, and the absence of a 'complete' syndrome does not exclude it [4,5,24].

Nystagmus and ocular signs by localisation

The oculomotor signature varies with the lesion site and helps localisation at the bedside [15,32]. Lateral medullary lesions produce mixed horizontal-torsional nystagmus, ocular lateropulsion and a skew, with the eyes and saccades pulled toward the side of the lesion [15]. Cerebellar nodulus and uvula lesions cause direction-changing gaze-evoked nystagmus and may generate positional nystagmus that mimics BPPV but lacks its latency and fatigability [15,32]. Pontine and floccular lesions impair smooth pursuit and gaze-holding, producing gaze-evoked nystagmus and saccadic pursuit [16,32]. An ocular tilt reaction — skew, head tilt and ocular counter-roll — localises to the graviceptive pathway from the vestibular nuclei to the midbrain [15,31].

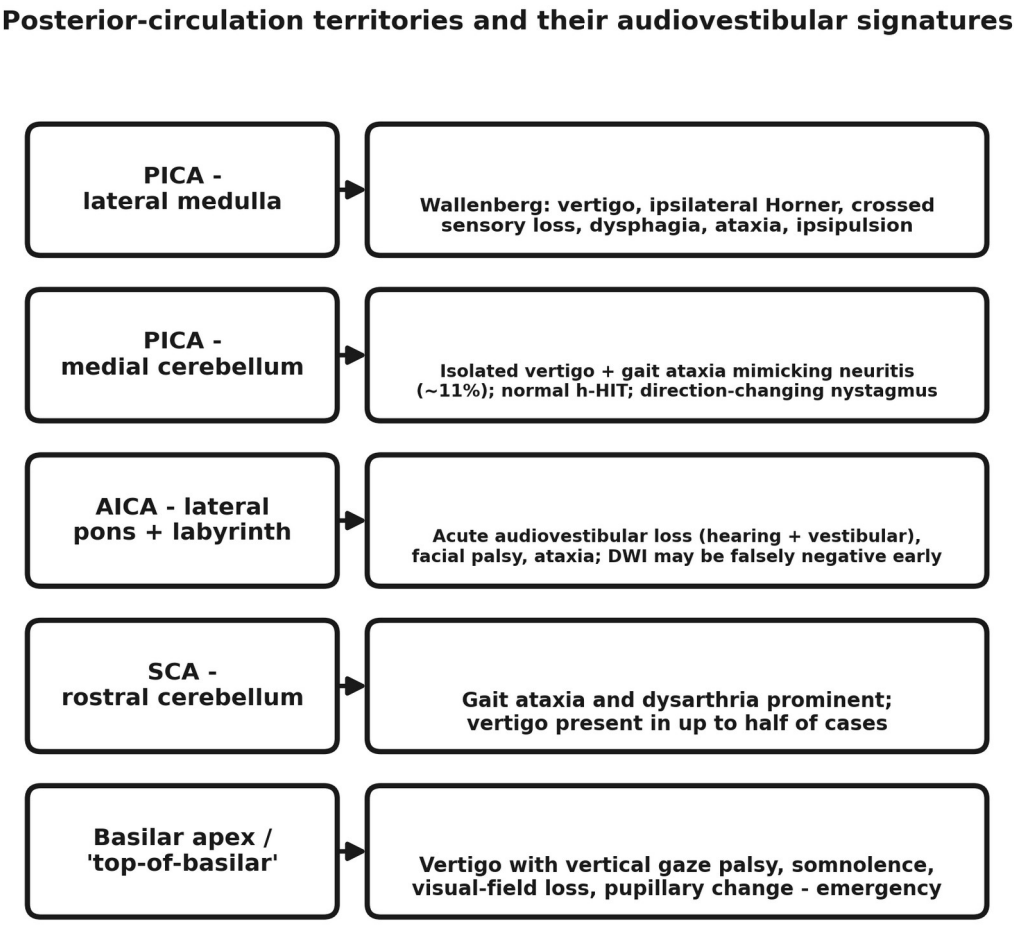


Figure 6. Posterior-circulation territories and their audiovestibular signatures.
Source: Adapted from Kim and Lee [5] and Choi, Lee and Kim [15].

Table 2. Vascular territory, stroke syndrome, and audiovestibular features [4,5,11,13,15,32].

Territory	Syndrome	Audiovestibular and associated features
PICA / vertebral (lateral	Wallenberg	Vertigo, Horner, crossed sensory loss,

medulla)		dysphagia, ataxia, ipsipulsion
PICA (medial cerebellum)	Isolated cerebellar infarct	Vertigo + gait ataxia mimicking neuritis; normal h-HIT; direction-changing nystagmus
AICA (lateral pons + labyrinth)	AICA syndrome	Vertigo + hearing loss, facial palsy/sensory loss, ataxia; DWI often negative early
SCA (rostral cerebellum)	SCA infarct	Gait ataxia, dysarthria; vertigo in up to half of cases
Basilar apex / distal basilar	Top-of-the-basilar	Vertigo with vertical gaze palsy, pupillary change, somnolence, visual loss

IV. The Acute Vestibular Syndrome and the HINTS Examination

The acute vestibular syndrome (AVS) is the clinical battleground on which vascular and peripheral vertigo are separated [1,10]. AVS denotes rapid-onset, continuous vertigo with nystagmus, nausea or vomiting, head-motion intolerance and gait unsteadiness, persisting for days [1,10]. Its two dominant causes are vestibular neuritis and posterior-circulation stroke, and the central task is to tell them apart at the bedside before imaging [1,2]. The HINTS battery — Head-Impulse, Nystagmus, Test-of-Skew — was designed precisely for this discrimination and is summarised in Figure 7 [1,30].

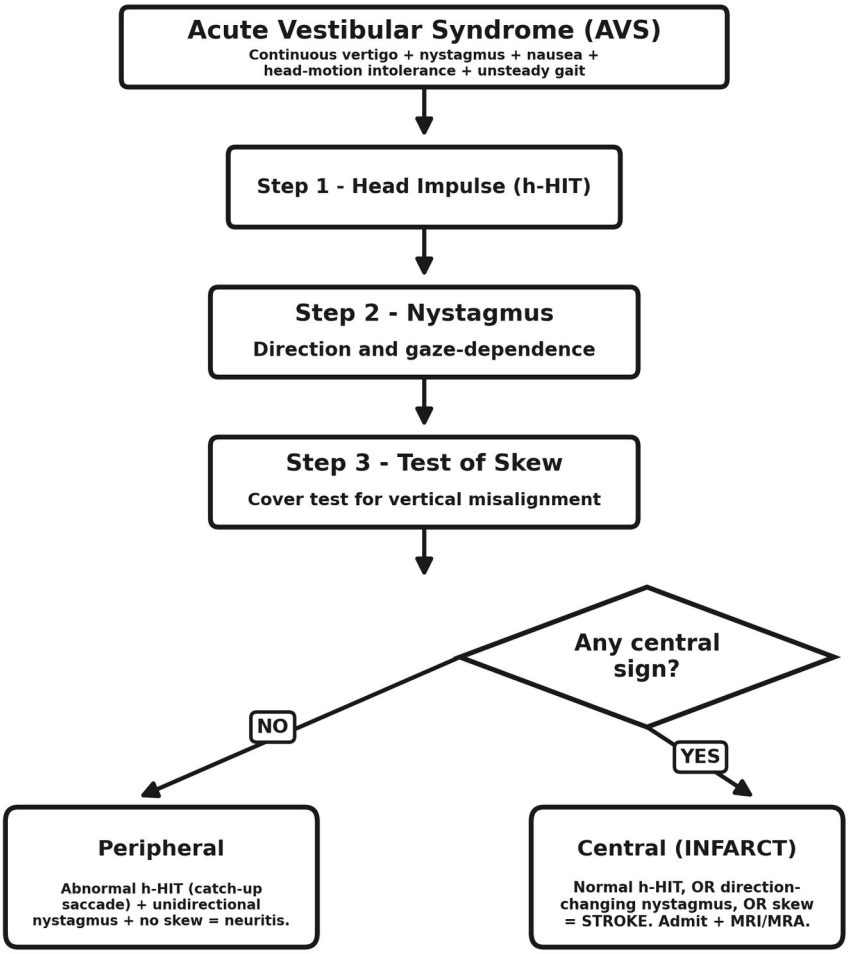
The three steps

The horizontal head-impulse test assesses the vestibulo-ocular reflex: a corrective catch-up saccade (an abnormal, 'positive' test) indicates a peripheral lesion of the labyrinth or vestibular nerve, whereas a normal test in a patient with florid spontaneous nystagmus is paradoxically a central sign [1,16]. Nystagmus that changes direction with gaze (gaze-evoked, direction-changing) is central, in contrast to the fixed, unidirectional, horizontal-torsional nystagmus of neuritis [1,30]. Skew deviation — a vertical ocular misalignment revealed by the alternate cover test — is a brainstem sign present in roughly 17% of central AVS and is especially useful when an abnormal head-impulse test falsely suggests a peripheral lesion [1].

The combination is powerful. In the seminal prospective study, the presence of any one central feature — a normal head-impulse test, direction-changing nystagmus, or skew — was 100% sensitive and 96% specific for stroke, outperforming early diffusion-weighted MRI, which was falsely negative in 12% within 48 hours [1]. Adding a bedside test of hearing (HINTS 'plus') further increases sensitivity, capturing AICA-pattern strokes that produce audiovestibular loss [2,5]. The mnemonic INFARCT — Impulse Normal, Fast-phase Alternating, Refixation on Cover Test — captures the central pattern [30].

Table 3. HINTS — peripheral versus central pattern in the acute vestibular syndrome [1,16,30].

Test	Peripheral (e.g. neuritis)	Central (stroke)
Head-Impulse (h-HIT)	Abnormal — corrective saccade present	Normal — no corrective saccade
Nystagmus	Unidirectional, horizontal-torsional	Direction-changing / gaze-evoked, or vertical
Test of Skew	Absent	Skew (vertical misalignment) may be present



HINTS (HINTS+ with hearing) is 100% sensitive and 96% specific for stroke in AVS, outperforming early MRI-DWI [Kattah 2009]. Apply only to continuous AVS with spontaneous nystagmus - never to a normal examination.

Figure 7. The HINTS three-step bedside oculomotor examination.
Source: Adapted from Kattah et al. [1] and Newman-Toker et al. [30].

Pitfalls and prerequisites

HINTS is only valid in a patient who actually has continuous AVS with spontaneous nystagmus; applying it to an episodic, triggered, or examination-normal patient generates dangerous false reassurance [2,34]. It must be performed and interpreted by a clinician competent in the head-impulse test, since an incorrectly performed test is the commonest source of error [29,49]. Truncal ataxia severe enough to prevent unsupported sitting or standing is itself a central red flag, regardless of the oculomotor findings, and should override an apparently 'peripheral' HINTS result [35]. Video-oculography is increasingly used to quantify the vestibulo-ocular reflex and remove inter-observer variability [29,49].

Performing the head-impulse test correctly

The diagnostic value of HINTS collapses if the head-impulse test is performed poorly [29,49]. The examiner delivers a small-amplitude (10–20 degree), high-acceleration, unpredictable horizontal head rotation while the patient fixates a target, watching for a corrective catch-up saccade that betrays a deficient vestibulo-ocular reflex [16]. Common errors include excessive amplitude, predictable timing, inadequate acceleration, and failure to ensure fixation — each of which can mask a peripheral deficit and thereby mislabel neuritis as stroke [29,49]. Covert corrective saccades occurring during the head movement are invisible to the naked eye and are a major reason quantitative video head-impulse testing outperforms the bedside version in equivocal cases [29,49].

Additional bedside signs

Several signs complement the core triad [15,32]. Gaze-evoked, direction-changing nystagmus and impaired smooth pursuit or gaze-holding point centrally [16,32]. Severe truncal ataxia — graded by the inability to sit unsupported — is an independent central sign that performed as well as the oculomotor triad in prospective study and should always be assessed [35]. The head-shaking and positional tests add information but must be interpreted cautiously, since perverted (cross-coupled) head-shaking nystagmus and atypical positional nystagmus are themselves central signs [15,32]. A bedside finger-rub or whispered hearing test completes the HINTS-plus battery and captures the AICA pattern [2,5].

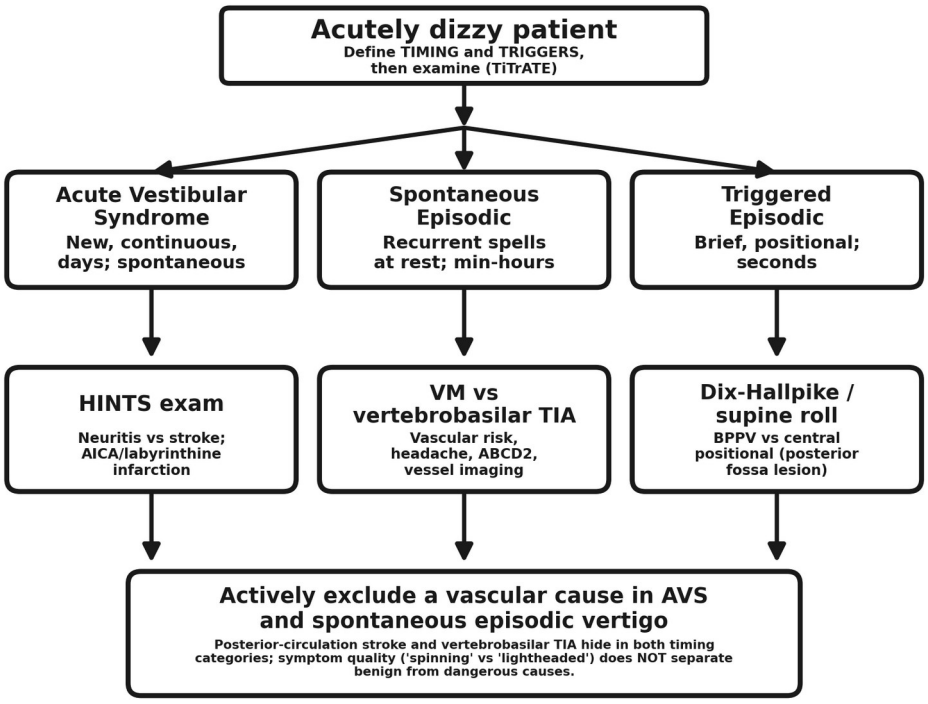
□ **Important:** A normal head-impulse test in a patient with acute, continuous spontaneous vertigo and nystagmus is a stroke until proven otherwise. The inability to sit or stand unaided (truncal ataxia) is a central sign in its own right and trumps a reassuring HINTS pattern.

V. Investigations and the Role of Imaging

Imaging in vascular vertigo is genuinely counter-intuitive: the more reassuring the early scan, the more important the examination [2,26]. Non-contrast CT is insensitive for acute posterior-fossa ischaemia and is useful chiefly to exclude haemorrhage; it should never be used to 'rule out' brainstem or cerebellar stroke [27,40]. Diffusion-weighted MRI is the imaging standard, but it is falsely negative in a clinically important proportion of posterior-fossa strokes when performed early [1,26].

The magnitude of this pitfall is striking. Early diffusion-weighted MRI missed 12% of strokes in the original HINTS cohort, all within 48 hours of onset [1]. For small infarcts — those most likely to present as isolated vertigo — false-negative rates reach around half within the first 24 to 48 hours, and the bedside HINTS-plus examination identifies them with substantially greater sensitivity than the scan [2]. The practical corollary is that a normal early MRI in a patient with a central oculomotor pattern does not exclude stroke; admission, observation and repeat imaging at 48 to 72 hours are warranted [2,26].

Vessel imaging is essential to define mechanism and guide prevention [40]. CT or MR angiography of the vertebrobasilar system identifies large-artery stenosis, basilar occlusion and arterial dissection, the last being a critical and treatable cause in younger patients [40]. A cardioembolic source should be sought with electrocardiography, prolonged rhythm monitoring and echocardiography where appropriate, because the mechanism dictates antithrombotic choice [28,40]. The timing-and-triggers (TiTrATE) framework in Figure 8 places this work-up within a structured diagnostic approach to the dizzy patient [3].



Timing + triggers + a targeted oculomotor exam replace the outdated 'type of dizziness' paradigm [Edlow 2018].

Figure 8. The timing-and-triggers (TiTrATE) approach to the acutely dizzy patient.
Source: Adapted from Edlow, Gurley and Newman-Toker [3].

Table 4. Red flags in the dizzy patient that mandate central work-up and MRI [2,3,32,35].

Red flag	Why it matters
Normal head-impulse test in continuous AVS	Suggests central rather than peripheral lesion
Direction-changing or vertical/pure-torsional nystagmus	Central oculomotor sign
Skew deviation	Brainstem localisation
Inability to sit or stand unaided (truncal ataxia)	Cerebellar/brainstem involvement
New headache or neck pain, especially in the young	Possible vertebral artery dissection
Any focal deficit (dysarthria, diplopia, weakness, sensory loss)	Localises to brainstem/cerebellum

Choosing and timing vessel imaging

Vessel imaging should be obtained early because it changes management [40]. CT angiography is fast, widely available and excellent for large-vessel stenosis and basilar occlusion, at the cost of contrast and radiation [27,40]. MR angiography, including non-contrast time-of-flight techniques, avoids iodinated contrast and pairs naturally with diffusion-weighted imaging, but is more susceptible to motion and flow artefact [26,40]. Where dissection is suspected, fat-suppressed axial T1 sequences through the neck demonstrate the intramural haematoma that angiography alone may miss [40]. Digital subtraction angiography is reserved for equivocal cases or endovascular planning [28,40]. The guiding principle is that mechanism cannot be defined without imaging the vessels, and mechanism drives both acute treatment and the intensity of secondary prevention [28,40].

Laboratory vestibular testing

Beyond neuroimaging, quantitative vestibular testing has a defined role once the acute phase is navigated [29,30]. The video head-impulse test quantifies semicircular-canal function and can demonstrate the preserved vestibulo-ocular reflex gain that, in the context of acute spontaneous nystagmus, points centrally [29]. Audiometry documents the cochlear component of an AICA infarct and may show the low-to-mid frequency loss of labyrinthine ischaemia [4,5]. Vestibular-evoked myogenic potentials and caloric testing help characterise the pattern of peripheral involvement when the diagnosis remains uncertain, although they do not substitute for the acute discrimination of stroke from neuritis [23,30]. These tools are most useful for confirming and characterising a vascular inner-ear lesion rather than for acute triage [29,30].

□ **Key Point:** A normal early MRI does not exclude posterior-fossa stroke — diffusion-weighted imaging misses up to half of small infarcts in the first 48 hours. When the bedside examination is central, admit and re-image; do not discharge on the strength of a reassuring scan.

VI. Transient Ischaemic Attack and Episodic Vascular Vertigo

Not all vascular vertigo is continuous. Vertebrobasilar transient ischaemic attack (TIA) produces brief, recurrent spells that fall into the spontaneous episodic vestibular syndrome and must be separated from vestibular migraine and Ménière's disease [3,51]. The contemporary definition of TIA is tissue-based — a transient episode of neurological dysfunction without infarction on imaging — rather than purely time-based [51]. Historically, isolated vertigo was thought never to represent TIA, but population-based data have overturned this: transient isolated brainstem symptoms, including isolated vertigo, frequently precede posterior-circulation stroke and carry a high short-term stroke risk [19].

The clinical features that should raise suspicion of a vascular mechanism are recurrent, stereotyped, abrupt spells in an older patient with vascular risk factors, particularly when accompanied — even fleetingly — by diplopia, dysarthria, perioral numbness, ataxia, drop attacks or visual disturbance [19,40]. Recurrent isolated vertigo lasting minutes, with vascular risk factors and no migrainous history, should be investigated as possible vertebrobasilar ischaemia rather than dismissed [19,48].

Risk-stratification tools designed for carotid-territory TIA perform poorly here. The ABCD2 score has limited discriminatory value for cerebrovascular causes of dizziness and should not be used to exclude stroke risk in the dizzy patient; a structured examination outperforms it [17,22]. The early risk of stroke after TIA is front-loaded — highest in the first days to weeks — so urgent vessel imaging and secondary prevention are warranted rather than deferred outpatient work-up [21,37]. Posterior-circulation TIA with vertebrobasilar stenosis carries a particularly high recurrence risk [20].

Table 5. Distinguishing vertebrobasilar TIA from vestibular migraine in spontaneous episodic vertigo [3,17,19,48].

Feature	Vertebrobasilar TIA	Vestibular migraine
Typical age / risk profile	Older; vascular risk factors	Younger; migraine history
Associated transient symptoms	Diplopia, dysarthria, ataxia, visual loss	Headache, photophobia, aura
Duration of spells	Seconds to minutes	Minutes to hours (sometimes longer)
Investigation priority	Urgent vessel imaging, secondary prevention	Clinical diagnosis; image if atypical

Positional and rotational vascular vertigo

Two uncommon but instructive vascular mechanisms deserve mention [9,40]. Rotational vertebral-artery (bow-hunter) syndrome produces vertigo and nystagmus provoked by sustained head rotation, when a dominant vertebral artery is mechanically compressed at the atlanto-axial level and the contralateral vessel is hypoplastic or occluded [9,40]. Subclavian steal can cause exertional posterior-circulation symptoms when proximal subclavian stenosis reverses vertebral flow during arm exercise [9]. Both are

rare, but each illustrates that positional or exertional vertigo is not invariably benign and that the vascular history and examination — including inter-arm blood-pressure comparison — retain their place [9,40].

□ **Clinical Pearl:** Isolated, recurrent, stereotyped vertigo in an older patient with vascular risk factors can be vertebrobasilar TIA. Do not use the ABCD2 score to reassure — it underperforms a structured examination and the early stroke risk is front-loaded.

VII. Acute Management and Secondary Prevention

Once posterior-circulation stroke is suspected, management is time-critical and follows the same reperfusion principles as anterior-circulation stroke, with territory-specific caveats [28,40]. The pathway in Figure 9 begins with confirming onset time, stabilising the airway and circulation, and determining eligibility for reperfusion [28].

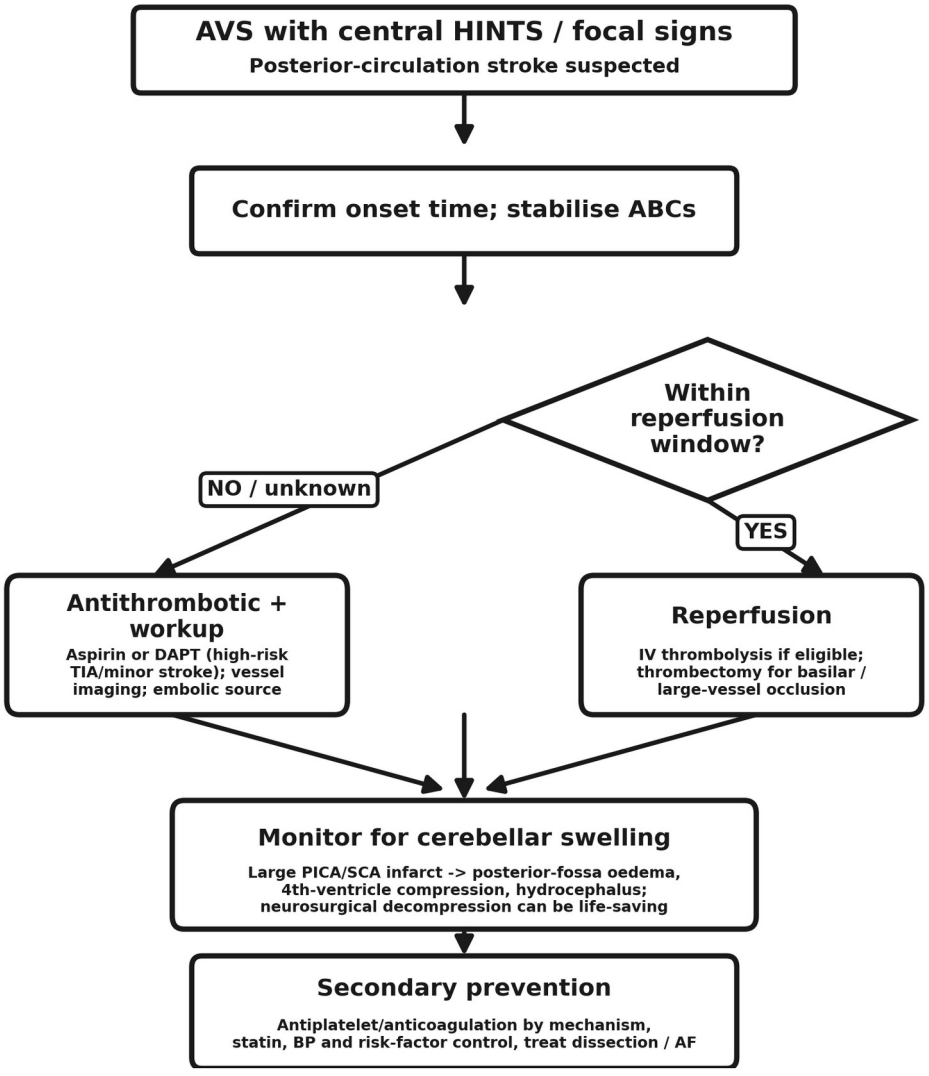


Figure 9. Acute management pathway for suspected posterior-circulation stroke.
Source: Adapted from Powers et al. [28] and Markus, van der Worp and Rothwell [40].

Reperfusion

Intravenous thrombolysis is indicated for eligible patients within the licensed time window, and is effective in posterior-circulation as in anterior-circulation stroke [28]. Endovascular thrombectomy is the critical intervention for basilar-artery occlusion and other large-vessel posterior occlusions, where the natural history is otherwise catastrophic; recanalisation can be life-saving and is increasingly offered in extended

windows at comprehensive stroke centres [28,40]. Because basilar occlusion may begin with vertigo and fluctuating brainstem symptoms, early recognition and rapid vessel imaging are decisive [13,40].

Antithrombotic therapy and prevention

For minor stroke or high-risk TIA not undergoing reperfusion, short-term dual antiplatelet therapy (aspirin plus clopidogrel) started early and continued for about three weeks reduces recurrent stroke compared with aspirin alone, after which single antiplatelet therapy continues [38,39]. Anticoagulation is indicated where a cardioembolic source such as atrial fibrillation is identified [28,40]. Arterial dissection is managed with antithrombotic therapy and, in selected cases, endovascular intervention [40]. Long-term prevention combines high-intensity statin therapy, blood-pressure control, and aggressive risk-factor modification, individualised to the confirmed mechanism [28,40,52].

Cerebellar swelling and neurosurgical rescue

A management priority unique to the posterior fossa is space-occupying cerebellar oedema [41]. Large PICA or SCA infarcts may swell over 24 to 72 hours, compressing the fourth ventricle to cause obstructive hydrocephalus and brainstem compression [41]. Deterioration in consciousness in this window is an emergency; suboccipital decompressive craniectomy, with or without external ventricular drainage, can be life-saving and is associated with good functional outcomes when performed before irreversible brainstem injury [41]. Any patient with a sizeable cerebellar infarct therefore requires close neurological observation in a setting with rapid access to neurosurgery [41].

Supportive care and rehabilitation

Beyond reperfusion and prevention, several supportive measures shape outcome after posterior-circulation stroke [28,52]. Blood pressure is managed according to reperfusion status — permissive in the acute ischaemic phase, then progressively lowered for secondary prevention [28]. Dysphagia is common with medullary and large cerebellar infarcts and must be screened before oral intake to prevent aspiration [28,52]. Vestibular suppressant drugs, useful for the first day or two of severe vertigo and vomiting, should be withdrawn early because they impair central compensation [32]. Vestibular and balance rehabilitation, begun once the patient is stable, accelerates recovery of gaze stability and gait in central as well as peripheral vestibular injury, and is a natural extension of the vestibular physician's role into the recovery phase [32,52].

□ **Important:** A large cerebellar infarct can kill through delayed swelling, not the initial ischaemia. Admit for monitoring, watch for declining consciousness over 24–72 hours, and involve neurosurgery early — decompression before brainstem injury saves lives.

VIII. Differential Diagnosis

The differential for acute vascular vertigo is dominated by its benign mimics, and the cost of confusion runs in both directions [2,10]. Vestibular neuritis is the principal peripheral mimic of central AVS; it produces unidirectional nystagmus, an abnormal head-impulse test and no other neurological sign, and is separated from stroke by the HINTS pattern and the absence of central features [1,10,16]. Labyrinthitis adds hearing loss, which — crucially — overlaps with AICA infarction and therefore cannot by itself be taken as reassurance of a peripheral cause [4,5].

Among episodic disorders, vestibular migraine is the commonest mimic of vertebrobasilar TIA and is distinguished by a migraine history, associated headache or photophobia, younger age and a benign longitudinal course [3,48]. Ménière's disease produces episodic vertigo with fluctuating hearing loss and aural fullness over a longer time-course [48]. Benign paroxysmal positional vertigo causes brief, positionally triggered vertigo and is separated by the Dix–Hallpike and supine-roll tests; persistent or atypical positional nystagmus, however, can reflect a posterior-fossa lesion and warrants central work-up [3,32]. Posterior-fossa tumours and demyelination round out the central differential [32].

Two principles reduce error. First, symptom quality (the patient's description of 'spinning' versus 'lightheadedness') does not separate dangerous from benign causes and should be abandoned as a

triage tool in favour of timing, triggers and examination [3,10]. Second, isolated vertigo in an older patient with vascular risk factors carries a measurable stroke risk and an elevated risk of stroke in the following months, so a confident peripheral diagnosis requires a confidently peripheral examination [43,44].

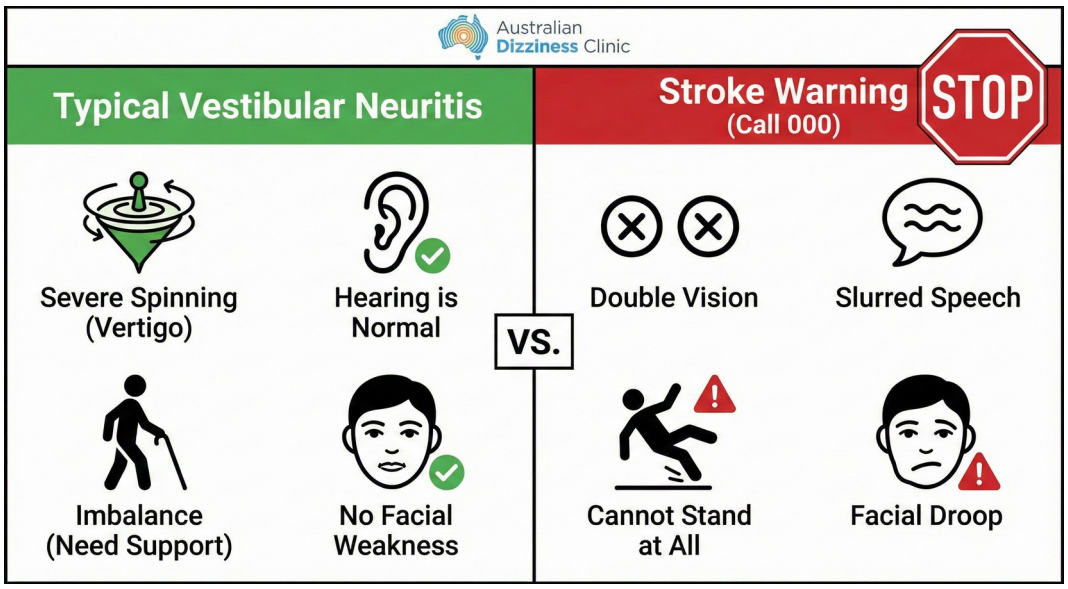


Figure 10. Bedside red flags separating typical vestibular neuritis from posterior-circulation stroke.
Source: Australian Dizziness Clinics.

Table 6. Differentiating central (vascular) AVS from peripheral causes [1,2,10,16,35].

Feature	Peripheral (neuritis/labyrinthitis)	Central (vascular)
Head-impulse test	Abnormal (corrective saccade)	Often normal
Nystagmus	Unidirectional, suppresses with fixation	Direction-changing, vertical, or fixation-resistant
Skew deviation	Absent	May be present
Gait / truncal stability	Unsteady but can stand	Often cannot sit or stand unaided
Other neurological signs	None	Dysarthria, diplopia, sensory loss, Horner, dysphagia

□ **Clinical Insight:** Hearing loss does not equal a peripheral diagnosis. Combined acute hearing and vestibular loss is the signature of AICA infarction — examine for facial and cerebellar signs and image the vessels rather than settling on labyrinthitis.

IX. Prognosis, Complications, and Special Populations

Prognosis in posterior-circulation stroke spans the full range from complete recovery to death, and is driven by mechanism, territory and the timeliness of recognition [12,40]. Small medullary and pontine infarcts presenting as vestibular syndromes often recover well functionally, although disabling chronic imbalance, oscillopsia and dysphagia can persist [15,32]. Audiovestibular loss from cerebellar and AICA infarction has a better long-term outcome than was once assumed, with partial recovery of vestibular function over months [6].

Complications

The dominant early complication is malignant cerebellar oedema, discussed in Section VII, which converts a survivable infarct into a brainstem-compression emergency over 24 to 72 hours [41]. Basilar occlusion carries a high mortality without recanalisation [13,40]. Recurrent stroke risk is substantial after posterior-circulation events, particularly with vertebrobasilar stenosis, which is why early mechanism definition and secondary prevention are integral rather than optional [20,37].

Stroke risk after apparently isolated vertigo

Patients hospitalised or discharged with isolated vertigo carry a higher subsequent stroke risk than matched controls, concentrated in the months after presentation [25,43,44]. This longitudinal risk reframes 'benign' isolated vertigo in the older, vascular-risk patient as a presentation that warrants risk-factor assessment and, where the examination is not reassuring, vascular work-up [43,44]. Historical electronystagmographic series likewise documented vascular vertigo as a discrete clinical entity long before modern imaging [45,46].

Chronic vestibular sequelae

Even after a small posterior-circulation infarct, central vestibular sequelae can be durable [15,32]. Central positional vertigo, downbeat nystagmus from floccular or nodular damage, persistent gaze-evoked nystagmus, and chronic imbalance with oscillopsia may all follow brainstem or cerebellar ischaemia [15,32]. Unlike peripheral lesions, central injuries compensate less reliably, and some patients require ongoing rehabilitation and, occasionally, pharmacological treatment of nystagmus [32]. Recognising these sequelae as the legacy of a vascular event — rather than a new peripheral disorder — prevents misdirected investigation and frames realistic expectations for recovery [15,32].

Younger patients and dissection

In younger patients, vertebral-artery dissection is a leading mechanism and may follow trivial neck trauma, sustained positions, or cervical manipulation [40]. The clue is neck pain or occipital headache preceding or accompanying the vertigo, and the diagnosis rests on vessel imaging [40]. Because the consequences of a missed dissection — progressive or embolic posterior-circulation stroke — are severe, a low threshold for angiographic imaging in the young dizzy patient with pain is appropriate [40].

Older adults

In older adults the pretest probability of a vascular cause is high, comorbidity complicates examination, and the consequences of both missed stroke and unnecessary admission are magnified [8,36]. A pragmatic stance combines a careful structured examination, liberal use of admission and delayed imaging when central features are present, and explicit attention to falls risk and rehabilitation during recovery [36,40].

X. Guidelines, Controversies and Future Directions

Contemporary practice is anchored by acute-stroke guidelines and by the maturing evidence base for bedside vestibular diagnosis [28,52]. National and international stroke guidelines define reperfusion eligibility, antithrombotic strategy and secondary prevention, and the modern tissue-based definitions of stroke and TIA frame the diagnostic categories used throughout this review [28,50,51,52]. What these documents historically under-emphasised — and what the vestibular literature has supplied — is the bedside method for catching the posterior-circulation stroke that hides as isolated vertigo [1,3,30].

Controversies

- **Symptom-quality triage —** the traditional paradigm that classifies dizziness by 'type' (vertigo versus presyncope versus disequilibrium) is unreliable and has been replaced by timing-and-triggers [3,10].
- **ABCD2 for dizziness —** the score underperforms a structured examination for identifying stroke in acute vertigo and should not be used to exclude risk [17,22].
- **Early MRI as a rule-out —** diffusion-weighted imaging is falsely negative in a substantial share of small early posterior-fossa strokes; the examination, not the scan, governs early safety [2,26,27].
- **Who should perform HINTS —** the battery is highly accurate in expert hands but error-prone when performed by the untrained; this drives interest in quantitative and automated approaches [29,49].

Future directions

Several developments are reshaping the field. Quantitative video-oculography — the emerging 'eye ECG' — measures vestibulo-ocular reflex gain and nystagmus objectively, reducing the dependence on

examiner skill and enabling portable, device-assisted triage in emergency settings [29,30,49]. Prospective imaging-based risk-stratification studies are refining who can be safely discharged and who needs admission [33,36]. The longer-term goal is an integrated pathway in which bedside or device-based oculomotor testing, selective vessel imaging, and rapid reperfusion are combined to cut the misdiagnosis of posterior-circulation stroke — still one of the most consequential diagnostic errors in acute care [34,42].

Implementation in acute pathways

Translating this evidence into practice is a systems problem as much as a clinical one [34,42]. Structured dizziness pathways that route continuous-AVS patients to a trained examiner, reserve imaging for those with central or equivocal findings, and provide rapid access to MRI and vessel imaging, reduce both missed strokes and unnecessary admissions [33,36]. Device-assisted oculography and tele-vestibular assessment extend specialist-level discrimination to centres without on-site expertise [29,30]. Education is the rate-limiting step: HINTS is only as good as the clinician performing it, so sustained training and audit are essential to any pathway that relies on it [29,42].

□ **Key Point:** The central message of this review is one rule: in acute continuous vertigo, the bedside oculomotor examination — not the symptom description and not the early scan — decides who has had a stroke. Master HINTS, respect its prerequisites, and image the vessels.

Summary

Brainstem and cerebellar stroke account for roughly a quarter of high-risk acute vestibular syndromes and are dangerously easy to miss [1,8]. The posterior circulation supplies the entire vestibular apparatus, so its ischaemia produces vertigo across a spectrum from isolated dizziness to locked-in syndrome, with AICA infarction uniquely combining central and labyrinthine loss [5,13]. The vestibular physician's value lies in a disciplined bedside method — timing and triggers, then a correctly performed HINTS-plus examination — interpreted against vascular risk, with truncal ataxia and any focal sign treated as central [1,3,35]. Imaging supports but does not replace this method, since early MRI misses small posterior-fossa strokes [2,26]. Recognising vascular vertigo opens the door to time-critical reperfusion, mechanism-based prevention, and vigilance for the delayed cerebellar swelling that can kill a patient who survived the initial infarct [40,41].

Five practice points consolidate this review:

- **Use timing and triggers, not symptom type —** classify the syndrome before examining, and abandon 'vertigo versus lightheadedness' as a triage tool [3].
- **Master HINTS and its limits —** a normal head-impulse test in continuous AVS is central; apply the battery only to spontaneous nystagmus and only when competent [1,30].
- **Treat audiovestibular loss as a possible stroke —** combined hearing and vestibular loss is the AICA signature, not reassurance [4,5].
- **Do not be reassured by an early normal MRI —** admit and re-image when the examination is central [2,26].
- **Watch the cerebellum swell —** monitor large infarcts for 24–72 hours and involve neurosurgery early [41].

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