Vestibular Neuritis and Labyrinthitis:

Recognising and Managing Acute Peripheral Vestibular Syndromes

Vestibular Medicine for General Clinicians

Topic 6 of 14

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How to Use This Review

This literature review is part of the Vestibular Medicine for General Clinicians series published by the Australian Dizziness Clinics Education Hub. It is written for general practitioners, hospital generalists, nursing, and allied health staff who assess and manage patients presenting with dizziness.

The review is designed to be read in a single 20–30 minute sitting, or used as a desktop reference. It is supported by an A4 one-page cheat sheet, short-form clinician videos, and audio episodes that cover the same material.

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, emergencies, and critical safety points requiring immediate action.

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I. The Clinical Problem

Vestibular neuritis and labyrinthitis together account for approximately 7 percent of presentations to outpatient dizziness services and are among the most common causes of acute vestibular syndrome (AVS) — continuous vertigo lasting days [1]. They are benign in the sense that the underlying pathology is self-limiting, but three aspects make them clinically important to the generalist: they must be reliably distinguished from posterior circulation stroke, which shares the presentation; early treatment with short-course corticosteroids and vestibular rehabilitation meaningfully improves outcomes; and prolonged use of vestibular suppressants — a common and understandable reflex — delays central compensation and prolongs disability [2,3].

The two conditions differ in a single feature: whether hearing is affected. Isolated vestibular nerve involvement produces vestibular neuritis (no hearing change). Simultaneous involvement of the cochlea produces labyrinthitis (vertigo plus sensorineural hearing loss and/or tinnitus). However — and this is the central message of this review — anterior inferior cerebellar artery (AICA) infarction can mimic labyrinthitis exactly, and must be considered every time vertigo presents with acute hearing loss, particularly in older patients with vascular risk factors [15,16].

□ **Key Point:** Acute continuous vertigo over 24 hours with unidirectional horizontal nystagmus and a clearly abnormal head impulse test is vestibular neuritis (no hearing change), labyrinthitis (with hearing change), or AICA infarction (with hearing change in older or vascular-risk patients). The critical task is to exclude stroke — use HINTS-plus, and image with MRI-DWI when hearing change occurs in any patient over 50 or with vascular risk.

The presenting syndrome is dramatic. Patients describe sudden-onset continuous rotational vertigo, severe nausea and vomiting, inability to walk without support, and marked head-motion intolerance. Symptoms peak within 24 hours and plateau for several days before gradually improving over weeks. Many patients initially present to emergency departments or out-of-hours services; primary care commonly sees them 48–72 hours later as symptoms begin to settle but daily function is still poor.

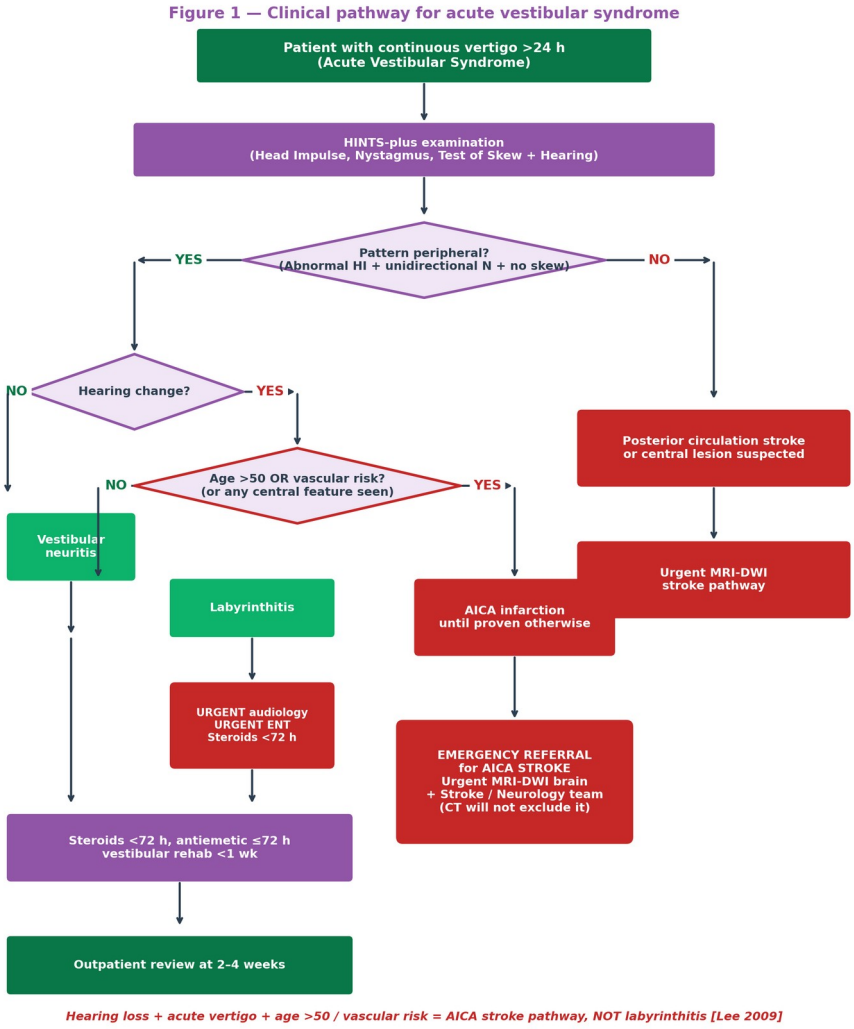

Figure 1. Clinical pathway for acute vestibular syndrome — HINTS-first triage, branching to peripheral treatment or stroke pathway.

Source: Australian Dizziness Clinics — clinical flowchart.

II. Pathophysiology and Terminology

Vestibular neuritis is an acute mononeuropathy of the vestibular nerve, most commonly the superior division (which innervates the horizontal and anterior semicircular canals and the utricle) [4]. Histopathological and imaging evidence supports a viral or post-viral inflammatory mechanism, with reactivation of latent herpes simplex virus type 1 in the vestibular ganglion being the leading hypothesis [5].

Labyrinthitis implies involvement of the entire membranous labyrinth, causing vertigo plus cochlear symptoms. It can be viral (most common, typically following an upper respiratory infection), bacterial (a middle-ear or meningitic complication, now uncommon but serious), autoimmune (Cogan syndrome, autoimmune inner ear disease), or vascular. Bacterial labyrinthitis is an otological and medical emergency.

Vascular Aetiology — AICA Infarction

The anterior inferior cerebellar artery (AICA) gives rise to the labyrinthine artery (also termed internal auditory artery) in approximately 80 percent of people, supplying both the cochlea and the vestibular labyrinth. Infarction in the AICA territory therefore commonly produces acute audiovestibular loss — vertigo and ipsilateral sensorineural hearing loss — and can mimic labyrinthitis exactly, even on bedside examination [15,16]. In the largest case series, audiovestibular symptoms occurred in around 95 percent

of patients with AICA territory infarction, and isolated audiovestibular presentation (without other cerebellar or brainstem signs) was the initial picture in approximately 8 percent [15]. Sudden hearing loss preceded the cerebellar or brainstem features by hours to days in another subgroup, providing a narrow window for prevention of completed stroke if the diagnosis is recognised early [17].

Approximate frequencies for the underlying mechanism in patients clinically diagnosed with acute peripheral vestibulopathy plus hearing loss are summarised in Table 1. Bacterial and autoimmune causes are uncommon, but vascular AICA territory infarction is sufficiently common in older patients with vascular risk that it should be actively excluded with MRI-DWI rather than presumed absent.

Table 1. Aetiology of acute peripheral vestibulopathy with hearing loss — approximate frequency in adult patients.

Mechanism	Approximate frequency	Clinical context
Viral / post-viral labyrinthitis	60–80%	Recent URTI; younger and middle-aged adults; isolated peripheral pattern.
Vascular — AICA infarction	5–10% (higher in age >50 with vascular risk)	Older patients; vascular risk factors; may have transient or evolving central signs; up to 95% of AICA strokes have audiovestibular involvement [15].
Bacterial labyrinthitis	<5%	Otogenic from chronic suppurative OM, or meningitic spread. Fever, otorrhoea, meningism. Otological + medical emergency.
Autoimmune inner ear disease	<5%	Cogan syndrome, AIED, systemic autoimmune disease. Often bilateral, fluctuating, steroid-responsive.
Idiopathic / unknown	remainder	Treated empirically as labyrinthitis if peripheral pattern is clear and AICA excluded.

Frequencies are approximations from cohort and review data [1,3,15,16]; not all patients undergo definitive aetiological workup. Adapted from Strupp & Magnusson 2015 [1] and Lee et al. 2009 [15].

Terminology — Why It Matters

- Vestibular neuritis: vertigo, no hearing loss. Most common.
- Labyrinthitis: vertigo plus acute sensorineural hearing loss or tinnitus — viral, bacterial, autoimmune, or vascular (AICA infarction).
- AICA infarction: stroke in the territory supplying the inner ear — clinically indistinguishable from labyrinthitis until imaged.
- "Acute unilateral vestibulopathy": the Bárány Society consensus term for the peripheral picture and avoids presuming the mechanism [6].
- Acute vestibular syndrome (AVS): the clinical syndrome — continuous vertigo over 24 h with nystagmus, nausea, and gait instability.

□ **Important:** "Labyrinthitis" is used loosely in the community as a catch-all for any vertigo. Used accurately, it specifically denotes vertigo with new sensorineural hearing loss — and because it carries a time-critical treatment window for sudden hearing loss (under 72 hours for steroids) AND because AICA infarction can present identically, the distinction matters clinically. Have a low threshold for MRI-DWI in any patient with AVS plus hearing change — particularly age over 50 or vascular risk [7,15].

Distinguishing peripheral from central causes is the dominant clinical task at first presentation. Approximately 10–20 percent of patients presenting with an AVS have a posterior circulation stroke rather than a peripheral vestibulopathy, and the clinical appearance can be indistinguishable on superficial examination [2,8]. The HINTS battery was developed precisely for this scenario and outperforms early MRI [2] — detailed in LR 03. AICA infarction, however, is the one stroke pattern that can produce a HINTS pattern indistinguishable from peripheral vestibulopathy, because the lesion is effectively in the labyrinth itself. Hearing change is the key clinical pointer.

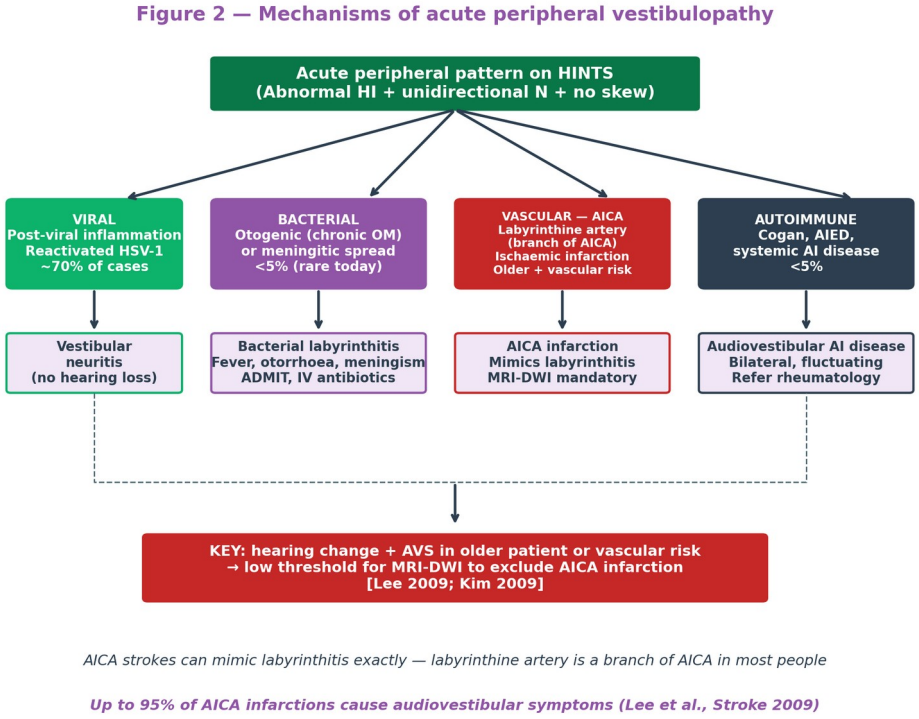

Figure 2. Mechanisms of acute peripheral vestibulopathy — viral, bacterial, vascular (AICA), autoimmune.
Source: Australian Dizziness Clinics — clinical flowchart. Frequency data adapted from Lee et al. 2009 [15].

III. Clinical Features and Diagnostic Criteria

The Bárány Society published consensus diagnostic criteria for acute unilateral vestibulopathy (AUV) / vestibular neuritis in 2022 [6]. These criteria are intended to be used at the bedside and should be applied explicitly.

Table 2. Bárány Society diagnostic criteria for acute unilateral vestibulopathy / vestibular neuritis.

Criterion	Requirement
A	Acute or subacute onset of sustained spinning or non-spinning vertigo lasting at least 24 hours.
B	Spontaneous peripheral vestibular nystagmus with trajectory appropriate for the semicircular canal afferents involved (usually horizontal-torsional, beating away from the affected side).
C	Unambiguous evidence of reduced VOR function on the affected side (abnormal head impulse, caloric paresis, or reduced video head impulse gain).
D	No evidence of acute central neurological or otological symptoms (other than cochlear symptoms, in which case labyrinthitis applies).
E	Not better accounted for by another disease or disorder.

All criteria must be met. The critical positive finding is an abnormal head impulse test on the affected side combined with peripheral-pattern nystagmus.

Symptom Profile

Symptoms develop over hours and peak within 24 hours. Continuous rotational vertigo is the dominant feature, accompanied by severe nausea and vomiting, oscillopsia (visual instability with head movement), gait unsteadiness with a tendency to fall towards the affected side, and head-motion intolerance. Patients commonly adopt a position of stillness with eyes closed, opening them only when necessary. Symptoms at rest improve over 48–72 hours; motion-provoked dizziness persists for weeks.

Associated Features

- A viral prodrome (URTI or gastrointestinal illness) in the preceding 1–3 weeks in around 50% of cases.
- Hearing loss or tinnitus distinguishes labyrinthitis from neuritis. Always ask. Where positive, arrange audiometry — and consider AICA infarction in older or vascular-risk patients.
- Headache is typically absent. New severe headache with vertigo should raise suspicion for central pathology.
- Symptoms are constant, not episodic. A history of multiple discrete episodes is against this diagnosis and suggests vestibular migraine or Ménière disease.

□ **Clinical Insight:** If the vertigo is episodic rather than continuous, this is not vestibular neuritis. The diagnostic pattern requires sustained, continuous symptoms lasting at least 24 hours. Patients with multiple previous attacks almost certainly have a different diagnosis.

Figure 3 — Distinguishing neuritis, labyrinthitis, and AICA infarction

Figure 3. Distinguishing neuritis from labyrinthitis from AICA infarction — gated by hearing change and vascular risk.

Source: Australian Dizziness Clinics — clinical algorithm.

IV. Examination — HINTS Plus Hearing

Examination of the patient with an acute vestibular syndrome has one overriding goal: to determine whether findings are peripheral (neuritis / labyrinthitis / AICA mimic) or central (other posterior circulation stroke, cerebellitis, demyelination). The HINTS battery — detailed in LR 03 — has sensitivity above 95 percent for stroke in this setting, outperforming early MRI, and should be performed in every patient with an AVS [2].

Table 3. Expected HINTS findings in vestibular neuritis / labyrinthitis versus posterior circulation stroke.

Component	Peripheral (neuritis / labyrinthitis)	Central (stroke)
Head Impulse (HI)	Abnormal (corrective saccade) on affected side	Normal bilaterally

Component	Peripheral (neuritis / labyrinthitis)	Central (stroke)
Nystagmus (N)	Unidirectional horizontal + torsional, suppressed by fixation	Direction-changing, vertical, or torsional; not suppressed by fixation
Test of Skew (TS)	Absent	Present — vertical skew deviation
Hearing	Normal (neuritis) or reduced (labyrinthitis)	Usually normal — but new hearing loss should prompt AICA stroke consideration
Gait	Unsteady, leans towards affected ear	Frankly ataxic, may be unable to stand
Other neurology	Absent	May have dysarthria, diplopia, limb weakness, Horner syndrome

The "INFARCT" mnemonic summarises central findings: Impulse Normal, Fast-phase Alternating, Refixation on Cover Test. Always add hearing — "HINTS-plus" — because AICA stroke can cause acute hearing loss [15,16].

□ **Important:** A normal head impulse test in a patient with acute vestibular syndrome is a red flag for stroke, not a reassurance. Do not be falsely reassured by the absence of focal limb weakness — posterior circulation strokes commonly present with isolated vertigo and ataxia only [8]. AICA infarction is the one exception where the head impulse can be abnormal — hearing change in a vascular-risk patient must therefore drive imaging.

Hearing Assessment — Bedside Methods

Bedside hearing testing should be performed on every patient with an AVS. The four tests below take less than two minutes combined and provide useful information at the point of decision. Formal pure tone audiometry is the definitive bedside-equivalent investigation and should be arranged urgently where any acute hearing change is suspected.

Table 4. Bedside hearing assessment in acute vestibular syndrome.

Test	Method	Interpretation
Whispered voice	Whisper a 3-digit number 60 cm from the test ear, masking the other.	Repetition correct → no significant loss; failure → acute SNHL or conductive loss possible.
Finger rub	Rub fingertips together 10–20 cm from each ear with the other masked.	Asymmetry suggests acute high-frequency loss on the affected side.
Weber (512 Hz)	Tuning fork on midline (forehead or vertex). Ask which ear it is heard in.	Lateralises to opposite ear in SNHL on affected side; lateralises to same ear in conductive loss.
Rinne (512 Hz)	Compare bone vs air conduction in each ear.	AC > BC = normal or SNHL; BC > AC = conductive loss.
Audiometry	Pure tone threshold testing across 250–8000 Hz.	Definitive — quantifies loss, identifies sudden SNHL ≥30 dB / 3 frequencies.

Bedside testing is screening. If any test suggests acute change, arrange urgent formal audiometry within 72 hours.

Additional Examination

A full cranial nerve and cerebellar examination (finger-to-nose, heel-shin, dysdiadochokinesia, tandem gait) should be documented. Any abnormality of speech, eye movements beyond peripheral nystagmus, limb coordination, or gait disproportionate to the vertigo mandates imaging.

□ **Clinical Pearl:** In older patients and those with vascular risk factors, the threshold for imaging should be low even when the clinical picture looks peripheral. MRI with diffusion-weighted sequences is the investigation of choice; CT is insensitive for posterior fossa stroke and should not be used to exclude it. Hearing change in this group is a particularly strong driver because of AICA mimicry.

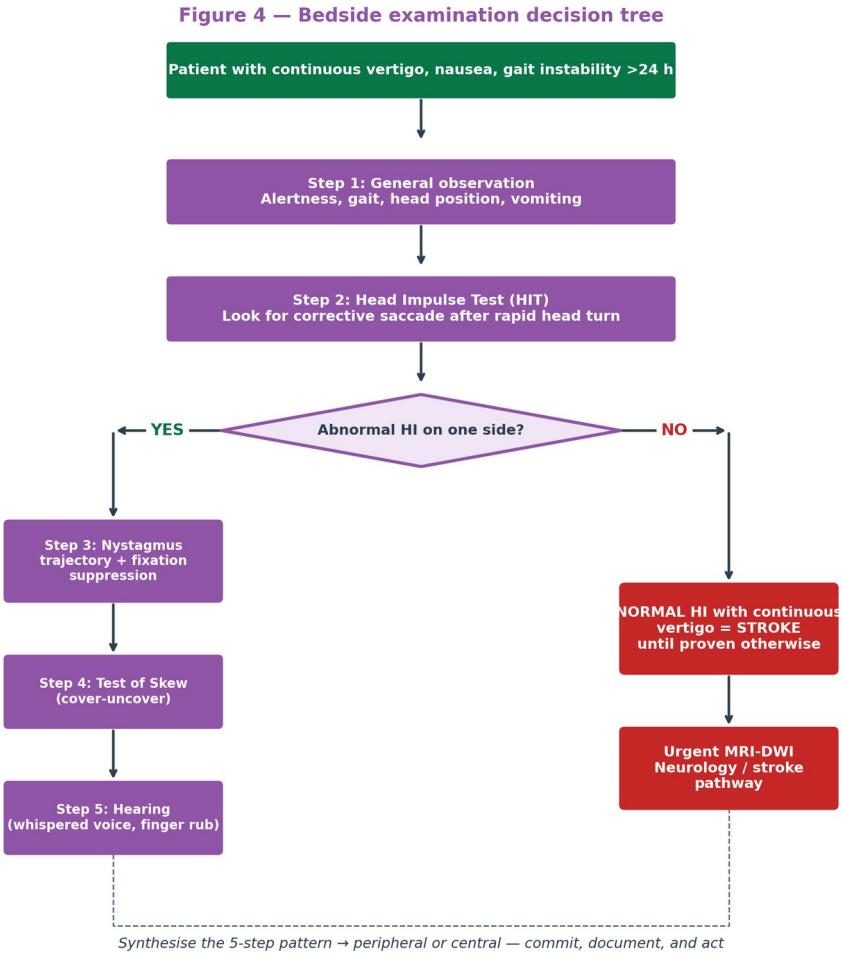

Figure 4. Bedside examination decision tree — five-step structured assessment in acute vestibular syndrome.
Source: Australian Dizziness Clinics — clinical flowchart.

V. Investigations

Vestibular neuritis is a clinical diagnosis. Investigations are used to exclude stroke, characterise any cochlear involvement, and identify alternative or additive diagnoses. The key principle is that investigations confirm and exclude — they do not gate treatment. Steroids and rehabilitation should be commenced on clinical grounds when the pattern is clear.

Table 5. Investigation indications and interpretation in acute vestibular syndrome.

Investigation	Indication	Notes / interpretation
MRI brain with DWI	Any central feature on HINTS-plus; hearing change with vascular risk; age >50 with first AVS; persistent symptoms; trauma onset.	Investigation of choice for posterior fossa. False-negative up to 20% in first 48 h for small infarcts — HINTS-plus is more sensitive in that window [2,9].
CT brain	Only when MRI contraindicated and acute haemorrhage suspected.	No role for excluding posterior fossa ischaemia. Do not use to "rule out stroke".
Pure tone audiometry	Any suspected acute hearing change. Mandatory before and after steroids in suspected sudden SNHL.	Sudden SNHL = ≥30 dB loss across 3 consecutive frequencies — otological emergency, steroids <72 h [7].
vHIT / VEMP / caloric	Incomplete recovery beyond 12 weeks; diagnostic uncertainty; residual deficit mapping for rehabilitation.	NOT required acutely. Reserved for the post-acute phase or expert review.
Bloods (targeted)	Older patients; vascular risk profiling;	Glucose, HbA1c, lipids, ESR/CRP, ANA

Investigation	Indication	Notes / interpretation
	suspected AI disease.	if AI disease suspected. No single test confirms diagnosis.
LP / borreliosis serology	Atypical features only.	Reserved for clinically indicated cases — not routine.

All investigations are confirmatory or exclusionary; none gate empirical treatment when the peripheral pattern is clinically clear.

□ **Clinical Insight:** Do not delay treatment to await investigations. In a typical presentation with clearly peripheral HINTS findings and no hearing change in a low-vascular-risk patient, commence oral corticosteroids and early vestibular rehabilitation on clinical grounds. In patients with hearing change, age over 50, or vascular risk, image with MRI-DWI in parallel — do not let the imaging delay the steroid decision.

Figure 5 — Investigation algorithm in acute vestibular syndrome

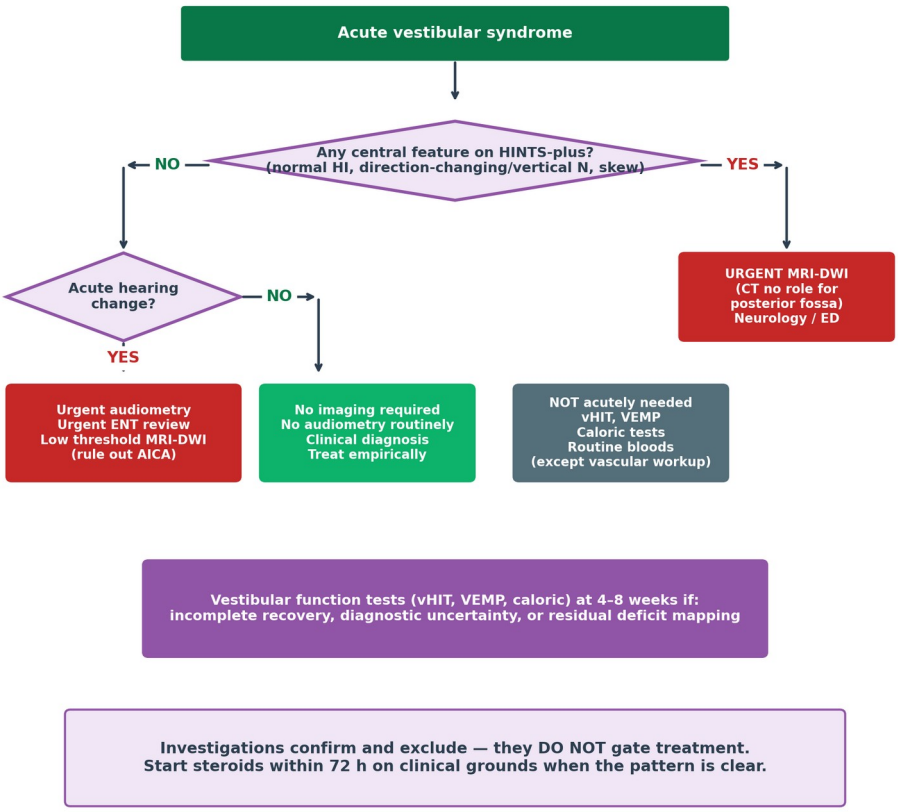

Figure 5. Investigation algorithm in acute vestibular syndrome — when to image, when to audiogram, when to defer.

Source: Australian Dizziness Clinics — clinical algorithm.

VI. Acute Management — Steroids and Symptom Control

Corticosteroids

Short-course oral corticosteroids, started within 3 days of symptom onset, accelerate recovery of vestibular function and improve rates of full caloric recovery at 12 months in vestibular neuritis. The landmark Strupp trial (2004) showed methylprednisolone 100 mg daily tapered over 22 days produced significant improvement in caloric recovery compared with placebo, with no added benefit from concurrent

valaciclovir [10]. A 2011 Cochrane review rated evidence as moderate — benefits on objective vestibular recovery are supported, effects on subjective symptom outcomes are less certain [11].

Table 6. Acute management summary for vestibular neuritis / labyrinthitis.

Intervention	Regimen / principle
Oral corticosteroids	Prednisolone 1 mg/kg (max 60 mg) daily for 5 days, then taper over 10–15 days. Start within 72 hours. Exclude diabetes decompensation, active infection, and TB contraindications.
Sudden SNHL (labyrinthitis)	Urgent ENT referral for intratympanic or oral high-dose steroids within 72 hours; audiogram before and after treatment. AICA infarction must also be excluded with MRI-DWI in older or vascular-risk patients.
Antiemetics / vestibular suppressants	Prochlorperazine or promethazine for 24–72 hours ONLY, to enable oral intake and sleep. Stop as soon as tolerated.
Hydration	Oral or IV as needed; short admission may be required for intractable vomiting.
Early mobilisation	Out of bed and walking with support as soon as tolerated — often day 2–3. Supervised movement accelerates compensation.
Vestibular rehabilitation	Begin within the first week. Formal program via vestibular physiotherapist ideal; structured home program acceptable.
Antivirals	No benefit demonstrated for herpes-directed antivirals in vestibular neuritis — not routinely recommended.

Steroids within 72 hours and early rehabilitation are the evidence-backed core. Prolonged vestibular suppressant use is the commonest management error.

□ **Important:** Do not prescribe prochlorperazine or promethazine for more than 48–72 hours. Prolonged vestibular suppressant use delays central compensation, prolongs symptoms by weeks to months, and is associated with chronic dizziness. This is the single most important management error in acute vestibulopathy [12].

Symptom Control

Vestibular suppressants have a narrow therapeutic window. In the first 24–48 hours, they reduce the intensity of acute vertigo and enable oral intake, hydration, and sleep. Beyond that, they impair the CNS's ability to recalibrate to the asymmetric vestibular input, prolonging symptoms [12]. The clinical message to patients and prescribers is identical: use briefly for acute relief, stop as soon as tolerated, and move early.

Admission Criteria

Most patients can be managed at home. Indications for hospital admission include intractable vomiting with dehydration, inability to mobilise safely, uncertainty about central versus peripheral cause requiring urgent imaging, and significant comorbidity.

VII. Vestibular Rehabilitation

Vestibular rehabilitation is the single most important disease-modifying intervention after the acute phase, with strong evidence from randomised trials and a 2015 Cochrane review demonstrating faster, more complete symptomatic recovery in acute peripheral vestibulopathy [13]. The principle is deliberate exposure to head and eye movements that initially provoke symptoms, driving central compensation — the CNS's remapping of the asymmetric vestibular input from the affected side.

Timing and Structure

Rehabilitation should begin within the first week, ideally within the first few days as tolerated. Formal assessment by a vestibular physiotherapist produces the best outcomes; where not accessible, a structured home program with written instructions and clear progression is a reasonable alternative. A typical early program includes:

- Gaze stabilisation: VOR x1 — fixate on a target while moving the head horizontally and vertically, 1–2 minutes, 3–5 times daily, progressing to VOR x2 (target moving in opposite direction to head).

- Habituation: repeated exposure to provoking movements (head turns, bending, rolling) at an intensity that elicits mild symptoms but settles within a minute.
- Balance: progressive standing exercises — feet together, tandem, one-foot, eyes open and closed, firm and soft surfaces.
- Gait: walking with head turns, then walking with vertical head movements, then busy visual environments.

□ **Clinical Pearl:** Patients often avoid movement because it provokes symptoms. This is the opposite of what they need. Reframe this explicitly: "Dizziness during exercise is expected and necessary — it is how your brain learns to recalibrate. A dizzy session is a productive session."

Progression and Duration

Most patients achieve substantial improvement in 4–8 weeks with consistent daily practice. Formal rehabilitation programs typically run for 6–12 weeks with weekly or fortnightly review. Persistent symptoms beyond 12 weeks warrant review — either for incomplete compensation (requiring program intensification) or development of secondary conditions such as PPPD or BPPV.

□ **Clinical Insight:** BPPV develops in approximately 10–15% of patients recovering from vestibular neuritis, due to utricular otoconial disruption by the underlying pathology. New positional vertigo during recovery is not treatment failure — it is a different, eminently treatable condition. Re-examine with Dix-Hallpike.

Figure 6 — Vestibular rehabilitation progression

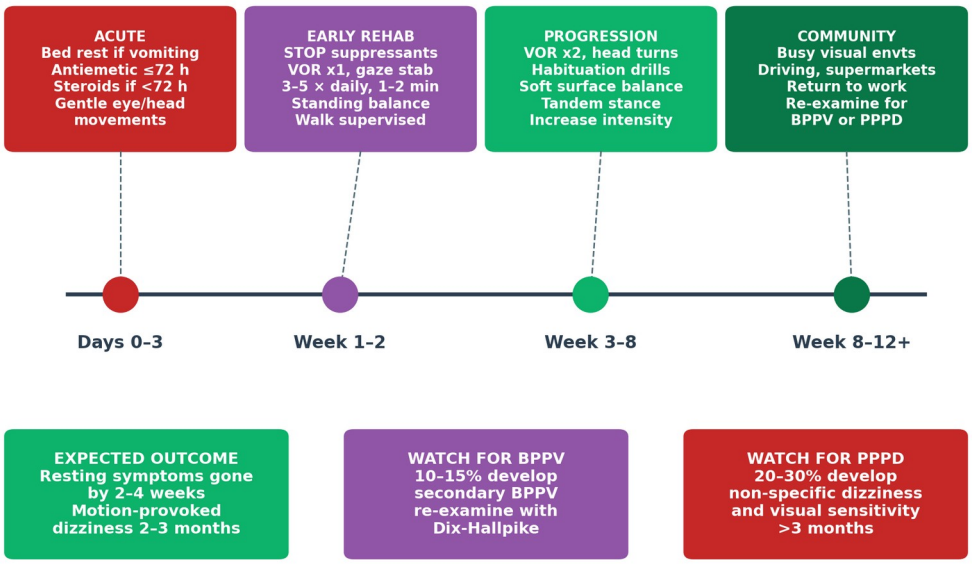

Figure 6. Vestibular rehabilitation progression — the four phases of recovery, with long-term complications to watch for.

Source: Australian Dizziness Clinics — clinical flowchart.

VIII. Recovery, Prognosis, and Long-Term Complications

The natural history of vestibular neuritis and labyrinthitis is gradual improvement over weeks to months. Caloric function recovers partially in around 40–60 percent of patients; the remainder retain a measurable permanent vestibular loss on the affected side [14]. This is usually clinically silent because central compensation adapts the CNS to the asymmetric input — most patients are asymptomatic at rest within 2–4 weeks and asymptomatic during normal activity within 2–3 months.

Factors Associated with Slower Recovery

- Older age — central compensation is slower beyond 70.
- Prolonged use of vestibular suppressants — the dominant modifiable factor.
- Delayed initiation of rehabilitation.
- Anxiety and depression, particularly pre-existing anxiety disorders.
- Comorbid migraine — predisposes to PPPD and to persistent visual dependence.
- Sedentary lifestyle or reluctance to mobilise during recovery.

Long-Term Complications

- Persistent postural-perceptual dizziness (PPPD): the commonest long-term complication, developing in 20–30% of patients. Characterised by chronic non-specific dizziness, unsteadiness, and hypersensitivity to visual motion lasting more than 3 months. Requires specific recognition and treatment (see LR on PPPD).
- Secondary BPPV: 10–15% — treated with canalith repositioning.
- Incomplete compensation: persistent head-motion intolerance and oscillopsia responsive to intensified rehabilitation.
- Bilateral vestibulopathy: rare progression or contralateral involvement — seek expert input.

□ **Key Point:** Early rehabilitation, short courses of vestibular suppressants, and active mobilisation are the three modifiable factors that most influence long-term outcome. The commonest reason patients are still symptomatic at 3 months is that they were told to rest and given ongoing prochlorperazine.

IX. Clinical Decision Pathway

The figures embedded throughout this review summarise the generalist approach. The overall AVS pathway (Figure 1) is the entry algorithm. Mechanisms of acute peripheral vestibulopathy (Figure 2) and the neuritis–labyrinthitis–AICA distinction (Figure 3) reinforce the differential at the moment of diagnosis. Bedside examination (Figure 4) and investigations (Figure 5) cover the workup. The vestibular rehabilitation progression (Figure 6) covers the recovery phase.

X. Red Flags and Referral Indications

Red Flags Requiring Urgent Imaging or Emergency Review

- Normal head impulse test with acute continuous vertigo — high probability of stroke.
- Direction-changing horizontal nystagmus, vertical nystagmus, or skew deviation.
- Any focal neurological sign — diplopia, dysarthria, limb weakness, Horner syndrome, sensory loss.
- Gait or limb ataxia disproportionate to the vertigo.
- New severe headache, particularly occipital.
- Acute hearing loss with vertigo in a patient over 50, or with vascular risk — consider AICA infarction; urgent ENT and MRI-DWI.
- Any patient with vascular risk factors plus acute vertigo, irrespective of HINTS pattern, warrants a low threshold for MRI-DWI.
- Trauma preceding onset — consider vertebral artery dissection.

Indications for Vestibular Physician or Dedicated Service Referral

- Diagnostic uncertainty or atypical features.
- Persistent symptoms beyond 12 weeks despite adequate rehabilitation.
- Recurrence of acute vestibular syndrome — suggests an alternative diagnosis.

- Development of chronic non-specific dizziness (PPPD) requiring structured treatment.
- Failure to tolerate rehabilitation or progression.
- Young patient with bilateral vestibular loss or progressive features.
- Coexisting audiological pathology requiring combined otological and vestibular input.

□ **Clinical Insight:** Recurrent "labyrinthitis" is almost always misdiagnosed vestibular migraine or Ménière disease. True vestibular neuritis recurs rarely and never in the same ear. A patient with "my third episode of labyrinthitis" should be reviewed with fresh diagnostic eyes.

□ **Important:** Two messages to emphasise at discharge and review: (1) stop vestibular suppressants as soon as tolerated — within 72 hours — and (2) move early and consistently. These two behavioural messages, delivered clearly, meaningfully change outcomes over the following months.

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