Vestibular Neuritis (Acute Unilateral Vestibulopathy):

A Vestibular Physician's Deep Review of Pathophysiology, Diagnosis, and Management

Vestibular Medicine for Vestibular Physicians

Peripheral Vestibular Pathology — Module 2.2

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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 peripheral vestibular practice, where a working command of mechanism, criteria, 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

Vestibular neuritis — now more precisely termed acute unilateral vestibulopathy (AUVP) by the Barany Society [1] — is the second most common peripheral vestibular disorder after benign paroxysmal positional vertigo and the most frequent cause of acute prolonged vertigo presenting to emergency departments and vestibular clinics worldwide [2,3]. It is defined by the abrupt onset of sustained, severe rotatory vertigo arising from acute dysfunction of one vestibular nerve or its end-organ, in the absence of auditory symptoms or central nervous system signs [1,4]. Despite its clinical prevalence, vestibular neuritis remains under-recognised in primary care and is frequently misattributed to non-specific dizziness, labyrinthitis, or — critically — posterior circulation stroke [5,6].

Population-based epidemiological studies estimate the annual incidence between 3.5 and 15.5 cases per 100,000 persons, with variability reflecting differences in diagnostic ascertainment and case definition [7,8]. A Croatian prospective study recorded an incidence of 11.7–15.5 per 100,000 per year; Italian multi-centre data from 2023 reported incidences near 20 per 100,000 in districts with comprehensive diagnostic protocols; and a Japanese survey reported approximately 3.5 per 100,000, attributed in part to differences in diagnostic thresholds [8,9,10]. The condition accounts for 3–10% of all presentations to tertiary dizziness and otoneurologic clinics [3,11].

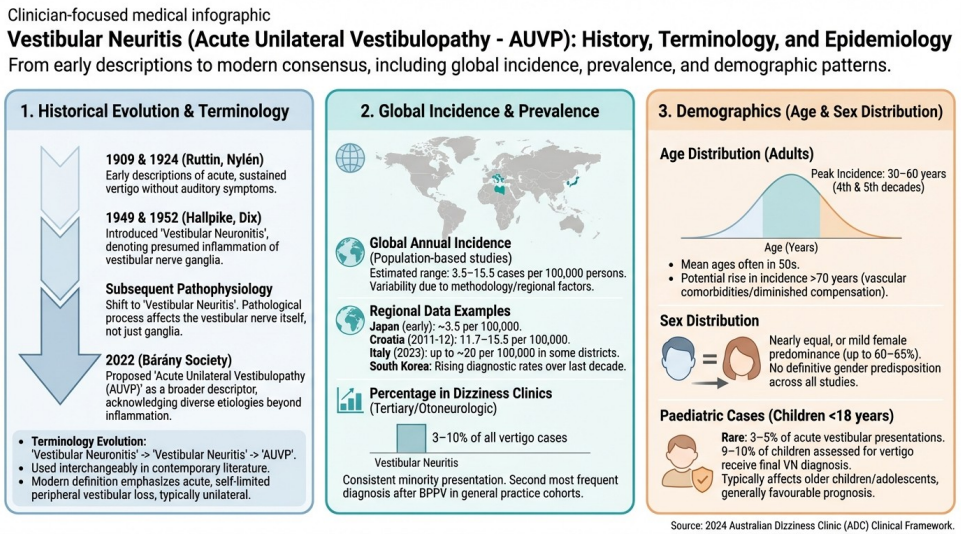


Figure 1. Clinical history-taking and examination in vestibular neuritis — the bedside assessment sequence.
Source: Australian Dizziness Clinics educational series.

Vestibular neuritis occurs predominantly between the 4th and 6th decades, with peak incidence in the 50s across most large cohorts [7,12]. Sex distribution is approximately equal, though some datasets report a mild female predominance of 55–60%, and a large Korean longitudinal analysis (2007–2022) demonstrated a higher male incidence in patients aged over 50 years [12,13]. Paediatric occurrence is markedly rare, representing only 3–5% of acute vestibular presentations in paediatric cohorts, often precipitated by a concurrent viral illness [14]. Recurrence rates of 2–15% have been reported, with contralateral involvement being more common than ipsilateral recurrence [15].

Table 1. Epidemiology of vestibular neuritis at a glance.

Parameter	Data
Annual incidence	3.5–20 per 100,000 (varies by region and ascertainment method) [7,8,10]
Peak age	4th–6th decade; mean age 50–55 years across large cohorts [7,12]
Sex ratio	Approximately 1:1 (mild female predominance in some series; male predominance >50 y in Korean data) [12,13]
Clinic prevalence	3–10% of all vestibular clinic presentations [3,11]
Recurrence rate	2–15% across studies; contralateral side more common than ipsilateral [15]
Paediatric cases	3–5% of acute vestibular presentations in children; markedly rare

	[14]

Key Point: Vestibular neuritis is the second most common peripheral vestibular disorder and the leading cause of acute sustained vertigo in emergency and vestibular clinic settings. Annual incidence ranges 3.5–20 per 100,000 depending on regional diagnostic infrastructure [2,3,7,8].

II. Pathophysiology — Viral Aetiology, HSV-1, and Inflammatory Mechanisms

Vestibular neuritis is pathophysiologically defined as acute inflammatory or ischaemic dysfunction of the peripheral vestibular apparatus — primarily the vestibular nerve — without concomitant cochlear involvement. Three mechanistic hypotheses have been advanced: viral inflammation (dominant), vascular ischaemia, and immune-mediated neuritis [16,17]. Each mechanism converges on the same clinical endpoint: acute unilateral peripheral vestibular deafferentation, generating an abrupt tonic asymmetry in the central vestibular network that drives spontaneous nystagmus, postural instability, and the subjective sensation of continuous vertigo [18].

Viral Infection and HSV-1 Reactivation

The leading hypothesis implicates reactivation of latent Herpes Simplex Virus Type 1 (HSV-1) within the vestibular ganglion of Scarpa. The foundational pathological evidence was established by Arbusow and colleagues, who detected HSV-1 DNA in human vestibular ganglia at autopsy in approximately two-thirds of individuals, accompanied by CD8+ T-lymphocytes and elevated interferon-gamma markers — consistent with an active or recently resolved immune response [16,19]. In contrast, HSV-1 DNA was rarely found in the cochlear ganglia of the same specimens, providing a histopathological correlate for the preserved cochlear function observed clinically in vestibular neuritis [19].

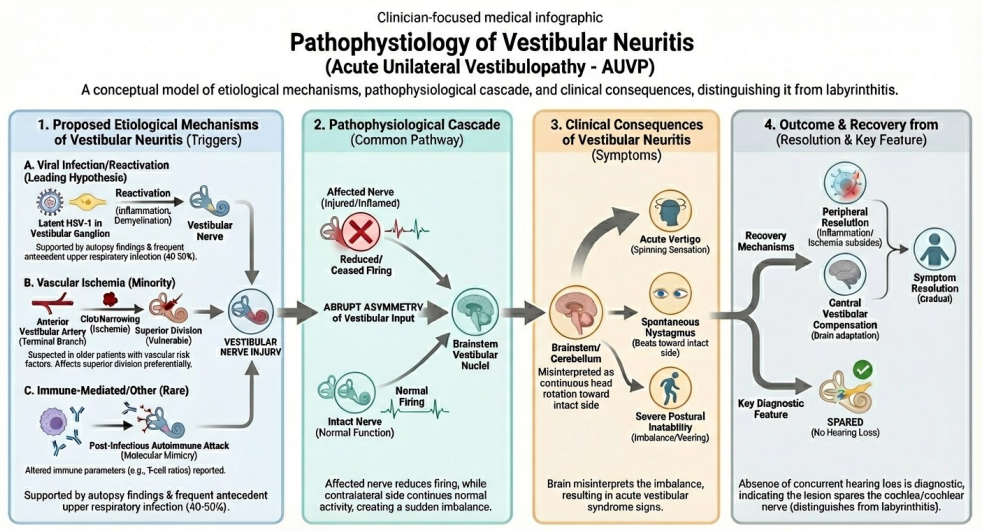


Figure 2. Pathophysiology of vestibular neuritis — viral reactivation in the vestibular ganglion of Scarpa, the resulting inflammatory cascade, and unilateral nerve deafferentation.
Source: Australian Dizziness Clinics educational series.

Strupp and colleagues demonstrated that methylprednisolone significantly improved peripheral vestibular function recovery — measured by caloric testing — at three months compared to placebo, validating the inflammatory hypothesis at a clinical level [20]. In contrast, the same study found no significant benefit of acyclovir over placebo, creating a persistent evidential tension: if the mechanism is HSV-1 reactivation, why does antiviral treatment not clearly accelerate recovery? [20]. This paradox underpins much of the ongoing controversy discussed in Section X and likely reflects that the dominant injury mechanism at clinical presentation is immune-mediated host response rather than ongoing active viral replication.

Other viral candidates include varicella zoster virus (VZV), Epstein-Barr virus (EBV), cytomegalovirus (CMV), influenza A, and adenovirus, proposed particularly in the context of prodromal or concurrent upper respiratory tract infection [17,21]. A prodromal upper respiratory illness is reported in 40–50% of adult cases and is even more frequent in paediatric series [14,22]. Direct viral culture from vestibular nerve tissue has not been achieved in living humans; the evidence base remains inferential from PCR, serology, and autopsy histopathology [16,19].

Vascular Ischaemia Hypothesis

A minority of cases may reflect ischaemic injury within the territory of the anterior vestibular artery, a terminal branch of the internal acoustic (labyrinthine) artery originating from the anterior inferior cerebellar artery [23]. This vessel has limited or no collateral supply, rendering the anterior vestibular nerve and the lateral semicircular canal particularly vulnerable to ischaemia. The vascular hypothesis is supported by the preponderance of superior vestibular division involvement, the increased incidence in vascular risk factor populations, and rare MRI-based demonstrations of restricted diffusion in the internal auditory canal [24]. However, diffusion-weighted MRI has insufficient resolution to resolve individual nerve fascicles, and confirmation remains difficult in most cases [24,25].

Immune-Mediated and Autoimmune Mechanisms

A third, increasingly studied mechanism involves immune dysregulation — either primary autoimmune neuritis or a post-infectious immune-mediated attack on vestibular nerve antigens [26]. Elevated serum anti-ganglioside antibodies and anti-HSP70 antibodies have been identified in subgroups of vestibular neuritis patients [26,27]. The latency between upper respiratory infection and symptom onset (typically 1–2 weeks in some cases) is consistent with a post-infectious molecular mimicry mechanism rather than direct viral cytopathic effect [26]. The relative contributions of direct viral injury, immune-mediated damage, and ischaemia vary across individual patients — which likely explains the heterogeneity in clinical severity, recovery trajectory, and treatment response observed in clinical practice [17,26].

Clinical Pearl: HSV-1 DNA is found in approximately two-thirds of human vestibular ganglia at autopsy — but antiviral therapy does not clearly accelerate recovery. This paradox likely reflects that the dominant injury mechanism is immune-mediated host response rather than active viral replication at the time of clinical presentation [16,20].

III. Clinical Features and AUVP Subtypes

Vestibular neuritis classically presents with acute onset of spontaneous, sustained, severe rotatory vertigo typically developing over minutes to several hours, reaching maximum intensity within 24 hours, and persisting for days to weeks [1,4]. The attack is continuous — not episodic — and is accompanied by nausea, vomiting, postural instability, and intense vegetative symptoms [28]. Hearing is preserved: audiometry and speech discrimination are normal, distinguishing vestibular neuritis from labyrinthitis, which involves concurrent sensorineural hearing loss [29].

The characteristic bedside finding is spontaneous unidirectional horizontal-torsional nystagmus of peripheral type, with the fast phase directed toward the intact ear and the slow phase toward the affected (paretic) ear [4,28]. Nystagmus follows Alexander's law: amplitude increases with gaze directed toward the fast phase and decreases with gaze toward the slow phase. Fixation suppression is intact — nystagmus diminishes under visual fixation, a critical feature distinguishing peripheral from central nystagmus [30]. The video head impulse test (vHIT) is positive on the affected side — an abnormal VOR gain produces visible or quantifiable catch-up saccades on head impulse toward the paretic ear [31].

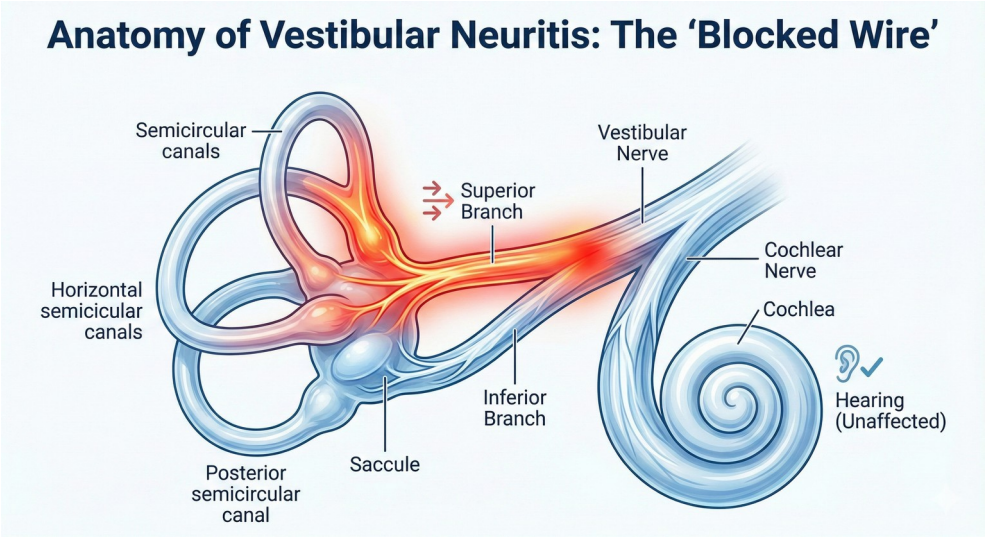


Figure 3. Superior vestibular neuritis — territory of the superior vestibular nerve branch, involving the anterior and lateral semicircular canals and the utricle.

Source: Australian Dizziness Clinics anatomical series.

AUVP Subtypes by Nerve Division Involvement

The Barany Society's 2022 AUVP framework recognises three anatomical subtypes based on the division of the vestibular nerve affected [1]. The clinical and physiological distinctions between subtypes have direct implications for bedside examination interpretation, as different subtypes produce different patterns of vHIT gain loss and nystagmus characteristics.

Superior Vestibular Neuritis

The superior division of the vestibular nerve supplies the lateral and anterior semicircular canals and the utricle. Superior VN is by far the most common subtype, accounting for approximately 85–90% of all cases [1,32]. Clinically it produces the classic presentation: prominent rotatory-horizontal nystagmus with the fast phase beating away from the affected side, positive horizontal vHIT (reduced gain and corrective saccades on head impulse toward the affected ear), intact posterior canal function on vHIT, and variable otolith dysfunction — utricular involvement may produce skew deviation or subjective visual vertical tilt [28,32]. The preponderance of superior division involvement reflects the anatomy of the anterior vestibular artery: this end-artery supplies the superior nerve fascicles with little collateral flow, making them disproportionately vulnerable to both ischaemic and inflammatory injury [23].

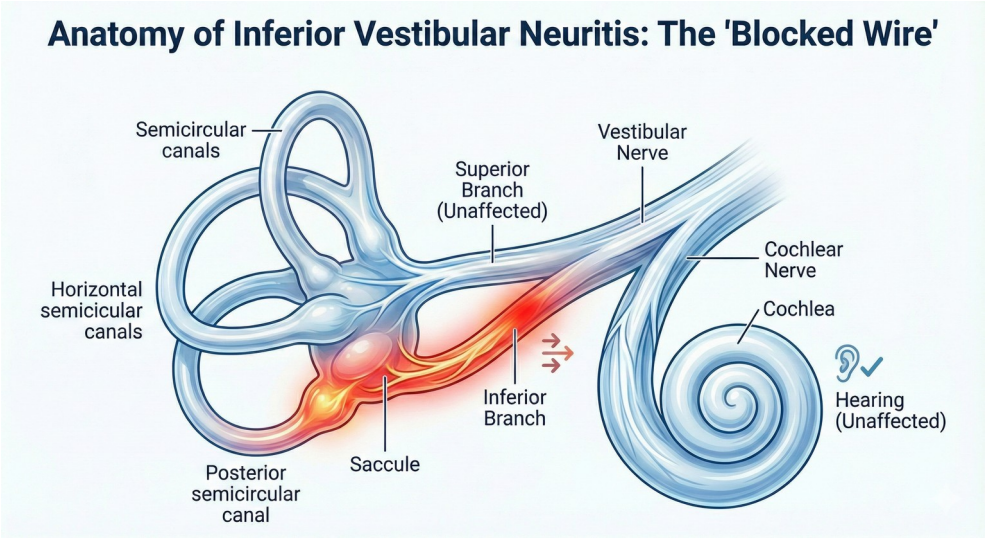


Figure 4. Inferior vestibular neuritis — territory of the inferior vestibular nerve branch, involving the posterior semicircular canal and the saccule.

Source: Australian Dizziness Clinics anatomical series.

Inferior Vestibular Neuritis

The inferior division supplies the posterior semicircular canal and the saccule. Inferior VN was historically considered either rare or clinically silent, but more recent systematic analysis suggests a prevalence of 5–10% of AUVP cases [33,34]. The diagnostic challenge is considerable: inferior VN produces a predominantly vertical nystagmus pattern with intact horizontal canal function on vHIT, positive posterior canal vHIT gain loss, and saccular dysfunction identifiable on cervical vestibular evoked myogenic potentials (cVEMP) [33,34]. Misattribution to BPPV (specifically posterior canal cupulolithiasis) or to central pathology is a genuine risk if bedside examination does not include vertical canal vHIT and cVEMP [34].

Pan-Vestibular (Total) Neuritis

Involvement of both superior and inferior vestibular nerve divisions — pan-vestibular or total neuritis — accounts for approximately 5–10% of AUVP cases [35]. All three semicircular canals and both otolith organs (utricle and saccule) are affected. Bedside findings include gain loss on horizontal, anterior, and posterior canal vHIT, profoundly abnormal caloric response, and bilateral VEMP abnormality [35]. Functionally, pan-VN is the most severe subtype and is associated with the longest recovery trajectories and greatest risk of persistent oscillopsia [35]. Some cases represent incomplete cochlear nerve sparing rather than pure vestibular restriction, warranting audiometric monitoring during follow-up [29].

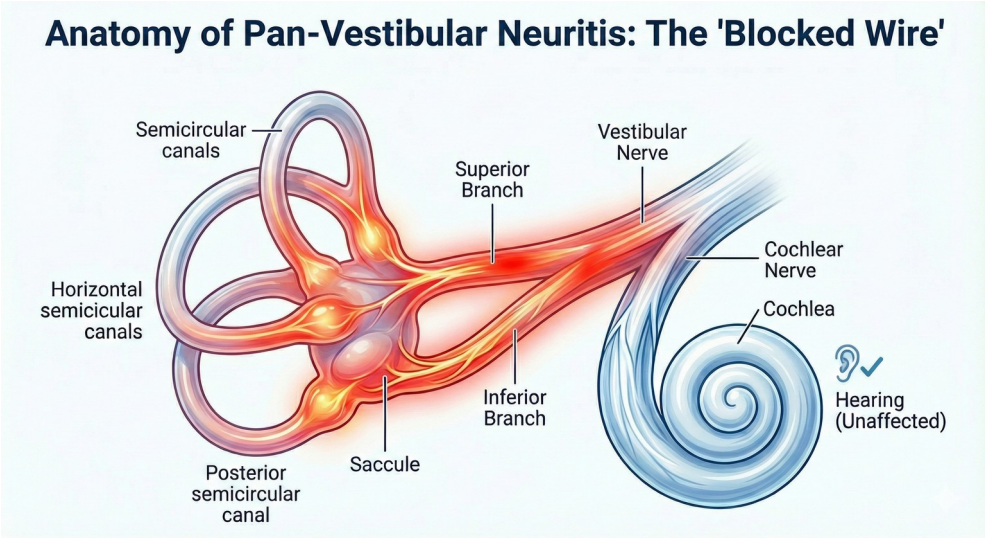


Figure 5. Pan-vestibular neuritis — involvement of the complete vestibular nerve, affecting all three semicircular canals and both otolith organs.

Source: Australian Dizziness Clinics anatomical series.

Table 2. AUVP subtypes — nerve division, canal involvement, and bedside findings.

Subtype	Prevalence	vHIT Pattern	VEMP Pattern
Superior VN	85–90% [1,32]	Horizontal + anterior canal gain loss; posterior canal intact; overt/covert corrective saccades	Abnormal oVEMP (utricular); cVEMP typically normal
Inferior VN	5–10% [33,34]	Posterior canal gain loss only; horizontal canal intact — easily mistaken for BPPV or central	Abnormal cVEMP (saccular); oVEMP normal
Pan VN	5–10% [35]	All 3 canal gains reduced; profound caloric canal paresis; complete peripheral deafferentation	Both cVEMP and oVEMP abnormal; worst prognosis subtype

Clinical Insight: Inferior vestibular neuritis is a clinical trap. Absent horizontal canal vHIT gain loss, the diagnosis is missed unless posterior canal vHIT and cVEMP are performed. Central pathology — specifically nodular infarction — can mimic inferior VN precisely. Always perform HINTS-Plus in any

suspected AUVP before diagnosing inferior vestibular neuritis [33,34].

IV. Diagnostic Criteria and the Barany Society AUVP Framework

The Barany Society's 2022 classification of acute unilateral vestibulopathy (AUVP) represents a paradigm shift from the older term 'vestibular neuritis' — which carries aetiological implications — toward a syndrome-based nosology [1]. The rationale is that the aetiology (viral, vascular, immune) cannot be confirmed in most patients, and a phenotypic label is more clinically reproducible and less mechanistically presumptuous [1,36]. In practice, vestibular neuritis and AUVP are used interchangeably, but the Barany framework provides a formal diagnostic scaffold that supports research comparability and international database harmonisation.

Diagnostic Criteria for Definite AUVP

The Barany Society diagnostic criteria for definite AUVP require all four of the following [1]:

A. Acute onset of sustained rotatory vertigo lasting days — not minutes or seconds — distinguishing from BPPV, TIA, or vestibular paroxysmia.

B. At least one objective sign of acute unilateral peripheral vestibular dysfunction: spontaneous nystagmus of peripheral type, abnormal vHIT (gain below 0.8 with corrective saccades), or unilateral caloric canal paresis (asymmetry exceeding 20% by Jongkees formula).

C. Absence of acute sensorineural hearing loss — cochlear function preserved on audiometry (pre-existing stable hearing loss excepted).

D. No signs of acute central nervous system dysfunction — normal HINTS examination, no cerebellar ataxia, no diplopia, no dysphagia, no facial numbness, no limb ataxia.

Probable AUVP is diagnosed when the typical symptom profile is present but objective vestibular testing is unavailable or technically limited — for example in the acute ED setting without access to vHIT [1,36]. In this context, clinical history, spontaneous nystagmus characteristics, and a negative HINTS examination are the diagnostic anchors.

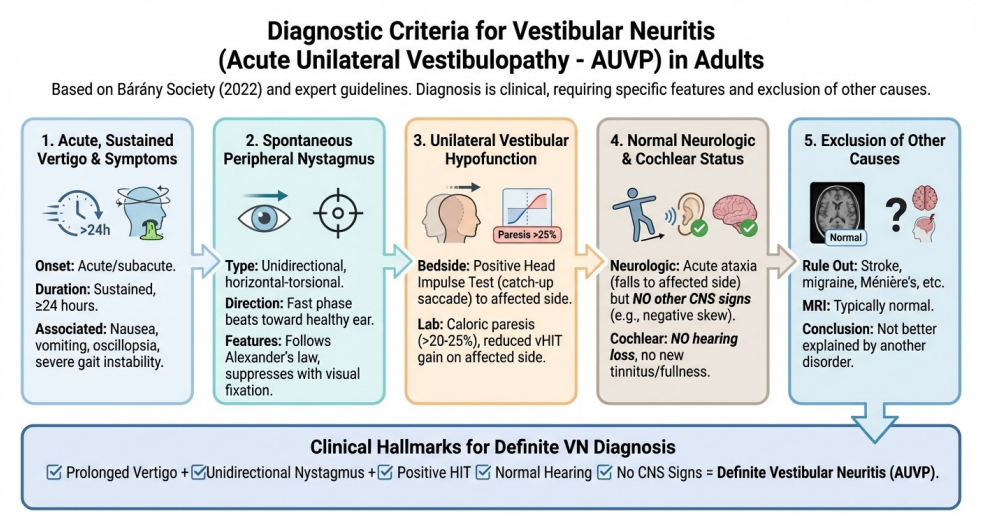


Figure 6. AUVP diagnostic assessment algorithm — integrating clinical history, HINTS, vHIT, and caloric testing to establish definite versus probable diagnosis.

Source: Australian Dizziness Clinics educational series.

HINTS and HINTS-Plus in the Emergency Setting

The HINTS battery — Head Impulse, Nystagmus direction, Test of Skew — is the single most powerful bedside discriminator between peripheral (vestibular neuritis) and central (posterior fossa stroke) acute vestibular syndrome [5,6,37]. A peripheral HINTS pattern — abnormal HIT, unidirectional nystagmus, absent skew — has sensitivity exceeding 100% and specificity of 96% for stroke when performed by trained clinicians [5,37]. In contrast, DWI-MRI within the first 24–48 hours of PICA or AICA territory infarction has a false-negative rate of 12–20% [38,39]. HINTS therefore out-performs acute MRI for early stroke detection in the acute vestibular syndrome.

Important: A normal head impulse test in a patient presenting with acute sustained vertigo and nystagmus is a central sign until proven otherwise. This is the single most critical HINTS finding. Do not diagnose vestibular neuritis if the HIT is normal — arrange urgent posterior fossa neuroimaging [5,6,37,38].

HINTS-Plus adds acute hearing loss assessment to the battery, acknowledging that AICA territory infarction can produce combined vestibulo-cochlear syndrome [40]. Sudden sensorineural hearing loss in the context of acute vestibular syndrome should trigger urgent MRI with DWI and posterior fossa sequences regardless of HINTS result [40,41].

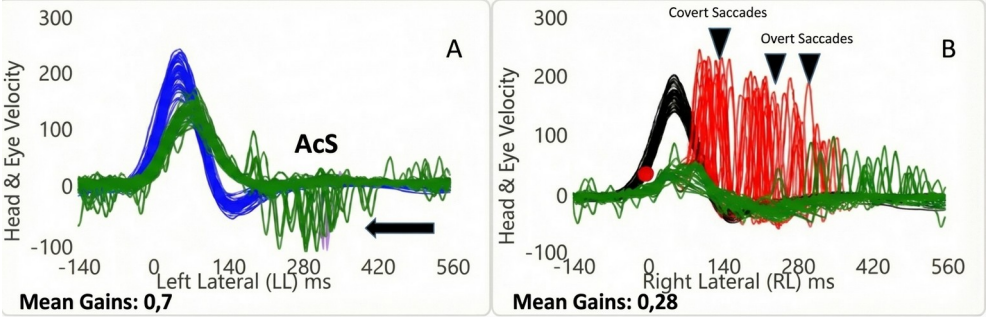
Table 3. Barany Society 2022 AUVP diagnostic criteria summary.

Criterion	Definite AUVP	Probable AUVP
A. Symptom duration	Sustained vertigo lasting days (not seconds/minutes)	Sustained vertigo; objective testing unavailable
B. Objective vestibular sign	vHIT gain below 0.8 OR caloric paresis over 20% OR peripheral nystagmus	Clinical nystagmus consistent with peripheral type without formal testing
C. Hearing	Normal audiometry (or stable pre-existing loss only)	No acute hearing loss reported by history
D. Central exclusion	No ASNHL; normal HINTS; no brainstem or cerebellar signs	Clinical assessment suggests absence of central signs

V. Investigations — vHIT, Caloric Testing, and Neuroimaging

Video Head Impulse Test (vHIT)

The vHIT is the gold-standard quantitative bedside test for vestibular neuritis [31,42]. By measuring the ratio of compensatory eye velocity to head velocity during rapid, unpredictable, small-amplitude head impulses (10–15 degrees), vHIT directly quantifies VOR gain in each of the six semicircular canal planes [31]. In acute vestibular neuritis, the gain on the affected side falls below 0.8 (normal threshold), and overt or covert corrective saccades are present. The vHIT's principal advantage over the traditional Halmagyi head impulse test is objective measurement, canal-plane specificity, and insensitivity to visual fixation interference [31,42].



Video head impulse test showing- Right lateral semi-circular canal shows reduced gain of 0.28 shows persistent **Overt** and **Covert** saccades showing dysfunction of right lateral semi-circular canal

Figure 7. vHIT demonstrating right horizontal semicircular canal dysfunction — reduced VOR gain with corrective catch-up saccades on head impulse toward the right paretic ear.
Source: Australian Dizziness Clinics clinical imaging series.

Canal-specific vHIT is critical for subtype identification: in superior VN, horizontal and anterior canal gains are reduced with a preserved posterior canal gain; in inferior VN, only the posterior canal gain is reduced with intact horizontal and anterior canal gains; in pan-VN, all three canal gains are reduced [32,33,35]. This pattern analysis directly informs the differential diagnosis and guides vestibular rehabilitation targeting [42,43].

Caloric Testing (Bithermal Irrigation)

Caloric testing remains an important complement to vHIT because it selectively probes the lateral (horizontal) semicircular canal at very low stimulus frequencies (0.003 Hz) — inaccessible to vHIT or rotatory chair [44]. In vestibular neuritis, caloric testing reveals unilateral canal paresis with asymmetry greater than 20% by the Jongkees formula [44]. The residual caloric response in vestibular neuritis is of significant prognostic importance: patients with caloric canal paresis exceeding 80% demonstrate slower recovery trajectories and more persistent symptoms [45]. Caloric testing cannot assess vertical canal function and will be normal in isolated inferior VN — a critical limitation when evaluating atypical presentations [44].

Vestibular Evoked Myogenic Potentials (VEMP)

VEMPs provide the most direct clinical assessment of otolith organ function and are essential for comprehensive AUVP subtyping. Cervical VEMPs (cVEMP) assess saccular and inferior vestibular nerve function; ocular VEMPs (oVEMP) assess utricular and superior vestibular nerve function [46]. In superior VN, oVEMP is typically absent or attenuated on the affected side while cVEMP is normal. In inferior VN, cVEMP is abnormal while oVEMP is preserved. In pan-VN, both are abnormal [33,35]. VEMP testing is particularly valuable in differentiating inferior VN from BPPV (VEMPs normal in BPPV) and from central mimics such as nodular infarction [34,46].

Neuroimaging — MRI and DWI

Neuroimaging is not required for typical vestibular neuritis but is indicated when central pathology is suspected. High-resolution T1-weighted MRI with intravenous gadolinium may demonstrate enhancement of the affected vestibular nerve, confirming inflammatory aetiology — however, this finding is present in fewer than 50% of cases and is not required for diagnosis [25,47]. Diffusion-weighted imaging (DWI) is the sequence of choice for acute posterior fossa stroke, but false-negative rates of 12–20% in the first 24–48 hours are well documented [38,39]. A negative DWI does not exclude stroke when HINTS examination suggests central pathology.

Important: A negative DWI-MRI within 48 hours does not exclude posterior circulation stroke. If HINTS suggests central pathology — particularly normal head impulse test, direction-changing nystagmus, or positive skew — the patient requires neurology consultation and repeat MRI at 72–96 hours, regardless of the initial DWI result [38,39].

VI. Differential Diagnosis of the Acute Vestibular Syndrome

The acute vestibular syndrome (AVS) — acute-onset, sustained vertigo with nystagmus and imbalance lasting hours to days — encompasses both benign peripheral and potentially catastrophic central aetiologies [5,6]. The differential diagnosis is clinically urgent because posterior circulation stroke requires immediate intervention. The following conditions constitute the primary differential diagnoses for AUVP and must be systematically considered at each presentation.

Table 4. Differential diagnosis of the acute vestibular syndrome.

Diagnosis	Key Distinguishing Features	HINTS Pattern	Differentiating Test

Posterior circulation stroke (PICA/AICA)	Normal HIT, direction-changing or vertical nystagmus, skew deviation, cerebellar or brainstem signs	Central (INFARCT)	DWI-MRI (may be false-negative at under 48 h); neurology consult
Labyrinthitis	Acute SNHL + vertigo; cochlear involvement; often post-viral	Peripheral	Audiometry (SNHL confirms); MRI to exclude retrocochlear cause
Ramsay Hunt syndrome	Vesicular rash (pinna or EAC), facial palsy (CN VII), SNHL + vertigo	Peripheral	Clinical diagnosis; VZV PCR from vesicular fluid
Meniere's disease (acute attack)	Episodic (20 min–12 h), aural fullness, fluctuating SNHL, tinnitus	Peripheral	Audiometry; electrocochleography; glycerol test
Vestibular paroxysmia	Brief attacks (seconds to minutes), not sustained AVS pattern	Variable	MRI neurovascular compression; carbamazepine response
Superior canal dehiscence	Positional or sound or pressure-induced; Tullio's phenomenon; no sustained AVS	Non-specific	High-res CT temporal bone; oVEMP enhanced amplitude
Inferior VN vs. nodular infarct	Posterior canal vHIT loss only; no horizontal loss — mimics nodular infarction precisely	May appear peripheral	DWI-MRI posterior fossa; HINTS-Plus full battery [33,34]

Clinical Pearl: AICA territory infarction is the most dangerous mimic of vestibular neuritis. It can produce an identical acute vestibular syndrome — sustained vertigo, peripheral nystagmus, and even an abnormal head impulse test — because the AICA supplies both the labyrinth and the lateral brainstem [40,41]. Any hearing change, ipsilateral facial numbness, or limb ataxia mandates immediate imaging even in the presence of an apparently peripheral HINTS pattern [40].

VII. Medical Management — Acute Phase and Pharmacotherapy

General Supportive Care

Vestibular neuritis is self-limited; acute management focuses on symptom control while central vestibular compensation is established. Short-term bed rest (1–3 days maximum) may provide comfort during severe acute vertigo, but prolonged immobility delays central compensation and must be avoided [48,49]. Maintenance of adequate hydration and nutrition, early controlled mobilisation once acute nausea permits, and fall prevention with environmental modification are the foundational supportive measures during the acute vestibular deficit period.

Vestibular Suppressants — Principles and Limits

Vestibular suppressants reduce the intensity of acute vestibular symptoms by dampening the sensory mismatch signal within the vestibular network, but simultaneously retard the central compensation process [48,49]. This creates a management tension: adequate symptom control is necessary for patient comfort, but prolonged suppression extends the recovery timeline. The evidence-based approach limits vestibular suppressant use to 72 hours maximum, with active weaning commenced as soon as tolerated [49].

Diazepam 2–5 mg orally or IV: effective acute vertigo intensity reduction; GABAergic action at central vestibular nuclei; avoid beyond 48–72 hours.

Promethazine 12.5–25 mg IM or oral: dopamine D2 antagonist with vestibular suppressive and antiemetic properties; sedating — use with caution in elderly patients.

Prochlorperazine 5–10 mg orally or buccal: first-line antiemetic in the acute setting; minimal vestibular suppressive effect relative to benzodiazepines.

Ondansetron 4–8 mg orally: 5-HT3 antagonist; useful for nausea control without vestibular suppressive properties — can be used beyond 72 hours for nausea management.

Corticosteroids — Evidence and Recommendations

The landmark RCT by Strupp et al. (2004) demonstrated that methylprednisolone (100 mg daily for 22 days with tapering) significantly improved recovery of peripheral vestibular function at three months — 83% recovery by caloric testing in the steroid group versus 58% in the placebo group [20]. Acyclovir provided no additional benefit [20]. However, subsequent meta-analyses have produced more ambiguous conclusions: the VISA-D study and later systematic reviews found that while caloric function was modestly improved by steroids, patient-centred outcomes — symptom resolution, functional recovery, and dizziness handicap — did not differ significantly between steroid and placebo groups [50,51].

Current practical recommendations: corticosteroids are most likely to improve vestibular physiological recovery when commenced within 72 hours, in patients with severe initial deficit (profound caloric canal paresis), and in the absence of contraindications [20,49]. Oral prednisolone 1 mg/kg/day (maximum 60 mg) for 7–10 days followed by a 10-day taper is a reasonable pragmatic regimen. The evidence does not support universal steroid use, but shared decision-making — acknowledging modest physiological benefit against the risk-benefit calculation — is appropriate for individual patients.

Antivirals — Limited Evidence

Antiviral agents (acyclovir, valacyclovir, famciclovir) are theoretically attractive given the HSV-1 reactivation hypothesis, but clinical trial evidence is consistently negative [20,51]. The Strupp 2004 RCT found no benefit of acyclovir over placebo for caloric or symptom recovery [20]. A retrospective series by Goudakos et al. reported modestly better outcomes with combined prednisolone plus valacyclovir, but methodological limitations preclude firm conclusions [51]. Antivirals are not recommended in routine clinical practice for vestibular neuritis; their use may be considered in Ramsay Hunt syndrome (VZV confirmed) or in immunocompromised patients [49,51].

Table 5. Pharmacological agents in acute vestibular neuritis management.

Agent	Dose	Role and Evidence	Limitations
Methylprednisolone	100 mg/day orally x 3 days then taper over 21 days	Improves caloric recovery at 3 months (RCT, Strupp 2004) [20]	No clear patient-centred outcome benefit in meta-analysis; GI and metabolic side effects
Prednisolone	1 mg/kg/day (max 60 mg) x 7–10 days then taper	Practical outpatient regimen; extrapolated from methylprednisolone data [49]	Variable absorption; cushingoid effects with prolonged use
Diazepam	2–5 mg oral or IV up to 72 hours	Effective acute vertigo suppression; GABAergic mechanism [48,49]	Retards central compensation; dependence risk; avoid beyond 72 h
Prochlorperazine	5–10 mg oral or buccal	First-line antiemetic; minimal vestibular suppression [49]	Extrapyramidal side effects; avoid in Parkinson's

			disease
Acyclovir or Valacyclovir	Standard antiviral dosing	No demonstrated benefit in RCTs [20,51]	Not recommended routinely; consider in Ramsay Hunt or immunocompromised

Clinical Insight: Corticosteroids improve physiological recovery of vestibular function (caloric testing) but may not translate to faster symptom resolution or reduced disability. Reserve for patients with severe acute deficit (caloric canal paresis over 80%), onset within 72 hours, and no contraindications. Antivirals are not supported by current evidence for routine vestibular neuritis [20,49,51].

VIII. Vestibular Rehabilitation Therapy

Vestibular rehabilitation therapy (VRT) is the most evidence-supported and clinically impactful intervention for vestibular neuritis recovery, and early initiation is the single most modifiable determinant of recovery speed and completeness [52,53]. VRT operates through three central nervous system mechanisms: adaptation (modification of VOR gain through repeated error-based learning); substitution (recruitment of alternative sensory strategies — visual, somatosensory — to replace lost vestibular input); and habituation (reduction of maladaptive responses through graded exposure) [52]. Each mechanism corresponds to a different exercise domain in the rehabilitation programme and is driven by the cerebellar and brainstem plasticity pathways engaged during active movement [18,52].

Non-Pharmacological Management of Vestibular Neuritis (VN): Summary Table (Clinician Guide)

Acute support and compensation.

INTERVENTION	PURPOSE & MECHANISM	INDICATIONS / TIMING (When to Order)	KEY CLINICAL PEARLS
General Supportive Care	Symptom management; prevent dehydration. Safe environment.	**Acute Phase** (1–3 days); bedridden/vomiting.	Ensure hydration/rest; IVs if needed. Reassurance. Mobilize after rest.
Vestibular Rehabilitation Therapy (VRT)	Accelerates compensation & recovery. Enhances adaptation.	**Start Early** (Day 2–3); as symptoms settle.	Early exercises improve outcomes. Mild provocation necessary.
Balance and Gait Training	Improves postural stability & gait.	Integrated with VRT; start early. Follow-up ~2–3 weeks.	Emphasize "use it to improve it". Sedentary slows recovery. Teach safety.
Positioning Manoeuvres (for BPPV)	Treat secondary BPPV. E.g., Epley.	For brief positional spells after VN. Test with Dix-Hallpike.	Secondary BPPV is common; treating it improves dizziness.
Psychological Support & Counselling	Manage anxiety; prevent chronic dizziness (PPPD). Addresses fear.	Acute reassurance. Persistent anxiety warrants support/CBT.	Reassurance vital. Vestibular CBT helps. Education gives control.
Return to Activity	Gradual resumption of activities. Promotes recovery.	Light activity within a week. Driving/heavy work when secure.	Encourage gradual return. Driving clearance crucial. Fall precautions essential.
Diet and Hydration	Supportive care. Maintain hydration. Avoid alcohol, caffeine.	Ongoing. No specific diet.	Maintain hydration. Low-salt not evidence-based. Avoid excess alcohol/caffeine.

Source: Adapted from clinical guidelines and expert consensus.

Figure 8. Non-pharmacological rehabilitation interventions in vestibular neuritis — gaze stabilisation, balance retraining, and habituation exercise sequences.
Source: Australian Dizziness Clinics clinical series.

VRT Programme Components

A comprehensive VRT programme for vestibular neuritis incorporates four principal exercise domains [52,53,54]:

Gaze stabilisation exercises: X1 manoeuvres (head movement while maintaining visual focus on a stationary target) and X2 manoeuvres (head and target moving in opposite directions). These directly drive VOR gain adaptation through the error signal generated by retinal slip during impaired VOR compensation.

Balance retraining: Progressive challenges to static and dynamic postural stability — standing on foam, tandem stance, single-leg stance, eyes-closed balance — systematically reducing reliance on visual dominance and promoting central vestibular re-weighting.

Habituation exercises: Repeated exposure to vertigo-provoking head positions and movements to drive neural habituation of the abnormal motion sensitivity signal. Head movement sequences, optic flow exposure, and VOR challenge paradigms.

Functional and activity-based training: Integration of vestibular challenge into activities of daily living — walking, stair negotiation, reaching tasks, and community mobility — to promote generalisation of central compensation to real-world demands.

Timing and Evidence

The Cochrane systematic review by Hillier and McDonnell (2023) confirmed that VRT significantly reduces dizziness, improves balance, and accelerates functional recovery in unilateral peripheral vestibular dysfunction [53]. Early initiation — within the first 1–2 weeks — consistently produces superior outcomes compared to delayed or no rehabilitation [52,53]. Recovery times are reduced by 30–50% with structured VRT compared to watchful waiting alone. VRT should commence as soon as acute nausea and vomiting permit active head movement — typically within 3–7 days of onset [52,53].

Home exercise compliance is a critical success variable: patient-directed home exercises, when adherence is confirmed, produce outcomes equivalent to supervised clinic-based programmes [52]. Digital therapeutic platforms with structured VRT exercise libraries and adherence tracking are emerging as an important delivery modality for patients with geographic or scheduling barriers to clinic access. Vestibular physiotherapists with specific AUVP expertise should coordinate the rehabilitation programme and adjust intensity as central compensation progresses.

Clinical Pearl: Early VRT is the most evidence-supported intervention for improving recovery speed and completeness in vestibular neuritis — more impactful than corticosteroids or antivirals in patient-centred outcomes. Begin within 1 week of onset. Do not wait for complete nausea resolution before referral to vestibular physiotherapy [52,53].

IX. Prognosis, Recurrence, and Special Populations

Natural History and Recovery Trajectory

Vestibular neuritis is a self-limited condition with a predictable natural history over weeks to months, though individual trajectories vary based on initial deficit severity, age, and rehabilitation access [45,55]. Central vestibular compensation begins immediately at onset but proceeds at different rates across individuals. In the acute phase (days 1–3), the primary mechanism is static compensation — reduction of the resting tone asymmetry between intact and paretic vestibular nuclei, driven by cerebellar-mediated inhibition of the contralateral intact nucleus [18,55]. Dynamic compensation — the restoration of VOR gain through adaptation pathways in the cerebellum and flocculus — proceeds over weeks to months and is the direct target of VRT [18,52].

Pooled data from prospective cohort studies provide the following recovery benchmarks [45,55,56]:

Days 1–3: Maximum symptoms — severe vertigo, nausea, inability to ambulate independently; spontaneous nystagmus prominent; vHIT gain at nadir.

Weeks 1–2: Rapid decline in vertigo severity; transition to assisted standing and gait; nystagmus amplitude decreasing under fixation suppression.

Weeks 3–8: Acute vertigo resolved in 70–80%; residual symptoms are dysequilibrium, postural imbalance, and oscillopsia with rapid head movements.

Months 2–6: 85–95% functional recovery (return to work and normal activities); vHIT gain partially or fully normalised; residual oscillopsia with vigorous head movement may persist in pan-VN.

Beyond 6 months: Over 95% report near-baseline or baseline function; persistent oscillopsia is the most common residual complaint; secondary BPPV in up to 30% of cases [57].

Table 6. Prognostic factors in vestibular neuritis recovery.

Factor	Effect on Recovery	Evidence Level
Young age (under 50 years)	Faster central compensation; shorter time to functional recovery [45,55]	Consistent across multiple cohorts
Early VRT initiation (within 2 weeks)	30–50% faster recovery; reduced long-term disability [52,53]	Level I evidence (Cochrane systematic review)
Incomplete initial vestibular deficit	Partial residual function predicts faster and more complete compensation [45]	Multiple prospective cohorts
Caloric canal paresis below 80%	Better prognosis than profound canal paresis [45]	Prospective studies; caloric testing at 1 month
Advanced age (over 60 years)	Slower compensation; may require 8–12 months; higher residual disability risk [55]	Consistent across cohorts
Psychosocial factors (anxiety, fear-avoidance)	Significantly impair recovery; drive somatisation and PPPD transition risk [58]	Observational and qualitative studies

Secondary BPPV as a Sequela

The development of BPPV following vestibular neuritis — secondary or post-neuritis BPPV — is a clinically important and often missed complication occurring in 10–30% of patients [57]. Most cases involve the ipsilateral posterior semicircular canal. The proposed mechanism involves utricular macula damage during the initial neuritis event, with subsequent inappropriate shedding of otoconia into the semicircular canals [57]. Secondary BPPV must be distinguished from incomplete central compensation — both may present with persistent positional dizziness — through formal Dix-Hallpike and supine roll testing at follow-up visits.

Persistent Postural-Perceptual Dizziness (PPPD) Transition

Approximately 15–25% of PPPD cases are preceded by a vestibular neuritis event [58,60]. The transition from acute vestibular neuritis to PPPD is driven by persistent maladaptive central sensitisation and behavioural fear-avoidance patterns that develop during the acute phase [58]. Early identification of psychological risk factors — health anxiety, catastrophising, avoidance behaviours — at the acute presentation may allow targeted psychoeducation and early VRT referral to reduce PPPD transition risk. Integrated vestibular-psychological co-intervention is the emerging standard for high-risk presentations [58,60].

Special Populations

Paediatric vestibular neuritis is rare and frequently misattributed to non-organic dizziness, migraine, or anxiety in the absence of formal vestibular testing [14]. When it does occur, the clinical presentation mirrors the adult pattern, but central compensation may be faster due to greater neuroplasticity [14]. Audiometric testing is mandatory in all children with acute vestibular syndrome to exclude concurrent hearing loss. The aetiology in children is more frequently identified — acute viral illness is the most common precipitant — and outcomes are generally excellent [14].

In older adults (over 70 years), the combination of reduced neuroplasticity, comorbid systemic conditions (diabetes, hypertension, cardiovascular disease), polypharmacy, and concurrent central lesions significantly complicates management and extends recovery [55]. Fall risk during the acute and subacute phases is substantial; inpatient or closely supervised outpatient VRT is preferred over home exercise alone in this population. Vestibular suppressant medications must be used with particular caution in the elderly due to sedation, fall risk, and drug interaction profiles [49].

X. Guidelines, Controversies and Future Directions

Current Guideline Landscape

The Barany Society's 2022 AUVP diagnostic framework is the most current international consensus position [1]. However, management guidelines remain fragmented across neurology, otolaryngology, and emergency medicine bodies. The AAO-HNS, the European Academy of Neurology, and the Cochrane database each address different components of care without a unified disease-specific clinical practice guideline for AUVP [49,52,53]. The 2023 Cochrane review provides the highest-level evidence for VRT; corticosteroid guidance remains based primarily on extrapolation from the Strupp 2004 RCT [20,53].

The Corticosteroid Controversy

Despite the Strupp 2004 RCT demonstrating improved caloric recovery with methylprednisolone [20], subsequent meta-analyses consistently fail to demonstrate patient-reported benefit in functional recovery, dizziness handicap, or return to activity [50,51]. The physiological versus clinical outcome dissociation is conceptually important: caloric function improvement does not necessarily translate to subjective improvement, likely because central compensation — rather than peripheral recovery — is the dominant driver of clinical wellbeing in the medium term [18,50]. This creates genuine clinical equipoise for corticosteroid use in vestibular neuritis.

The Antiviral Debate

No adequately powered RCT has demonstrated clinical benefit of antivirals in vestibular neuritis, yet the histopathological evidence for HSV-1 reactivation as the primary pathological event is compelling [16,19,20,51]. A future multicentre RCT with adequate power, early treatment initiation (within 24 hours of symptom onset), and patient-centred primary endpoints is needed to definitively answer the antiviral question. Until then, antivirals are not recommended as standard care.

Nosological Debate — Vestibular Neuritis vs. AUVP

Whether to retain 'vestibular neuritis' (implying a specific inflammatory mechanism) or adopt 'AUVP' (syndrome-based, aetiologically neutral) has practical implications for patient communication, insurance coding, and research database harmonisation [1,36]. The emerging consensus favours AUVP as the research and diagnostic term while acknowledging 'vestibular neuritis' as the familiar clinical shorthand. Vestibular physicians should be familiar with both terms and their distinction.

Future Directions

Biomarker development for AUVP is an active research frontier. Serum and CSF inflammatory markers, anti-ganglioside antibody profiles, and viral PCR panels may eventually allow aetiological stratification of AUVP patients — identifying viral, vascular, and immune-mediated subgroups for personalised treatment selection [26,27]. Machine learning approaches applied to vHIT trajectories and recovery kinetics are showing promise for predictive modelling of individual compensation trajectories [42].

The therapeutic landscape may expand with novel targets: intratympanic corticosteroids as an adjunct to systemic therapy; valacyclovir in early-onset suspected HSV-1 cases; and neuroplasticity augmentation strategies such as transcranial direct current stimulation targeting the intact flocculus are under investigation [59]. The interface between vestibular neuritis and PPPD — a significant proportion of PPPD cases appear to originate with an acute vestibular event [58,60] — represents an important area where early psychological and vestibular rehabilitation co-intervention may reduce the transition from acute to chronic dizziness.

Key Point: Approximately 15–25% of PPPD cases are preceded by vestibular neuritis. Early identification of psychological risk factors at the acute presentation may allow targeted intervention to reduce PPPD transition risk — an area where vestibular physicians can have significant population-level impact [58,60].

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