Vestibular Neuritis and Labyrinthitis in Children:

Acute Vestibular Syndrome, Recovery, and Rehabilitation

Vestibular Medicine in Children

Topic 5 of 15

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How to Use This Review

This literature review is part of the Vestibular Medicine in Children series published by the Australian Dizziness Clinics Education Hub. It is written for vestibular physicians, paediatricians, and emergency physicians who assess and manage children presenting with vestibular disorders.

The review is designed to be read as a deep-reference resource or used as a clinical desktop companion. It is supported by a clinical 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.

Table of Contents

Feature	Vestibular neuritis	Labyrinthitis	Central mimic (stroke/tumour)
Hearing loss	ABSENT	PRESENT — cochlea involved	Variable; sudden SNHL = AICA stroke
Nystagmus	Horizontal-torsional; fixed direction	Same as neuritis	Direction-changing; vertical; gaze-evoked
Head impulse test	ABNORMAL — corrective saccade	ABNORMAL	NORMAL — VOR intact = alarm sign
Skew deviation	ABSENT	ABSENT	PRESENT — cover-uncover vertical deviation
Gait	Can walk; veers to lesion side	Can walk; severe nausea	Often cannot walk; truncal ataxia
Preceding illness	URTI 1–2 weeks; varicella common	URTI or febrile illness	None — or vascular/demyelinating history

Component	Peripheral (reassuring)	Central (alarm — MRI urgently)
H — Head Impulse Test	ABNORMAL: corrective saccade on rapid passive impulse	NORMAL: no corrective saccade — VOR intact
I — Nystagmus direction	Unidirectional horizontal-torsional; does not change with gaze	Direction-changing OR pure vertical OR gaze-evoked
T — Test of Skew	ABSENT: no vertical deviation on cover-uncover	PRESENT: vertical skew — brainstem/cerebellar sign
Overall	All three peripheral = neuritis until proven otherwise	ANY central sign = MRI-DWI urgently
Age feasibility	Cooperative from ~age 5; passive HIT from 18 months	Always attempt; partial result still informative

Investigation	Purpose	When to order
HINTS exam (bedside)	Peripheral vs central differentiation	ALL cases of acute sustained vertigo
Pure-tone audiogram	Neuritis vs labyrinthitis; SNHL detection	All cases within 24–48 hours
MRI brain + posterior fossa + DWI	Central pathology exclusion	Abnormal HINTS OR atypical features
FBC, CRP, viral serology (VZV, HSV)	Aetiology; guide antiviral treatment	Severe/bilateral; immunocompromised
vHIT + caloric (post-acute)	Quantify VOR deficit; guide VRT	Week 2–4 after acute phase

Audiogram at 6 weeks	Detect permanent SNHL post-labyrinthitis	All labyrinthitis cases — SNHL in 30–50%

V. Bedside Examination: HINTS and Adapted Paediatric Assessment

Intervention	Detail	Duration
Vestibular suppressants	Ondansetron 0.15 mg/kg (max 8 mg) for vomiting	72 hours MAXIMUM then STOP
IV fluids	If unable to maintain oral intake	Until oral intake re-established
Corticosteroids	Prednisolone 1 mg/kg/day — limited paediatric evidence	5 days; unlicensed — discuss
Antivirals (aciclovir)	If HSV labyrinthitis; immunocompromised; bilateral loss	Per indication
Early mobilisation	Day 2–3 walking; exercises as tolerated	Critical — promotes compensation

VI. Investigations and Differential Diagnosis

VII. Acute Management

VIII. Vestibular Compensation and Rehabilitation

Phase	Timing	Approach
Early mobilisation	Day 2–3	Supervised walking; head movement tolerance; prevent fear avoidance
Gaze stabilisation VOR x1	Week 1–2	Head movement while fixating stationary target; start slow
Balance retraining	Week 2–4	Tandem stance; foam surface; reduce visual reliance
Full VRT programme	Weeks 2–8	Vestibular physiotherapist; school-adapted exercises
Return to school	Most within 1–2 weeks	Reduced sessions; avoid PE until compensation progressing
BPPV check at 6 weeks	Dix-Hallpike	Post-neuritis BPPV in 10–15%; treat with Epley if positive

IX. Prognosis and Long-Term Outcomes

X. Summary and Key Clinical Takeaways

Indication	Urgency	Refer to
Normal head impulse test + acute sustained vertigo	Emergency	MRI-DWI + neurology — central cause
Severe SNHL in labyrinthitis	Urgent (within 72 hours)	ENT; intratympanic steroids
Failure to compensate at 4–6 weeks	Soon	Vestibular physician; formal vHIT + VRT review
Bilateral vestibular loss at presentation	Urgent	Vestibular physician + autoimmune/haematological workup
PPPD developing post-AUVL	Routine (within 4 weeks)	Vestibular physician +

		paediatric psychologist

References

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I. Introduction: Acute Vestibular Syndrome in the Paediatric Context

The acute vestibular syndrome (AVS) is defined as a sudden-onset, sustained, constant vertigo persisting for more than 24 hours, accompanied by nausea or vomiting, gait instability, spontaneous nystagmus, and intolerance of head movement. In the emergency and acute paediatric setting, AVS is an important and frequently challenging presentation — challenging because the peripheral causes (vestibular neuritis, labyrinthitis) are clinically indistinguishable from potentially catastrophic central causes (posterior fossa stroke, tumour, demyelination) on initial presentation without systematic bedside evaluation. [1,2]

Vestibular neuritis and labyrinthitis are the most common causes of peripheral AVS in children. Vestibular neuritis refers to isolated inflammation of the vestibular nerve (predominantly the superior division), producing acute unilateral vestibular hypofunction without cochlear involvement. Labyrinthitis extends this to include cochlear dysfunction, presenting with the additional features of sudden sensorineural hearing loss (SNHL) and tinnitus. Both conditions are thought to arise from viral or post-viral inflammation of the membranous labyrinth or vestibular ganglion, following the pattern established in adult studies by Arbusow and colleagues. [3,4]

While vestibular neuritis and labyrinthitis are generally benign and self-limiting, they carry significant acute morbidity in children — including inability to attend school, prolonged nausea, and anxiety about recurrence. The key challenge at the time of initial presentation is not diagnosis of the peripheral condition per se, but rather the safe exclusion of central pathology, which requires systematic application of the HINTS examination and a low threshold for neuroimaging in the paediatric context.

□ **Important:** AVS in a child is a posterior fossa lesion until proven otherwise. Always perform HINTS before discharge from the emergency department. The threshold for MRI neuroimaging is lower in children than in adults, given the higher relative prevalence of posterior fossa pathology.

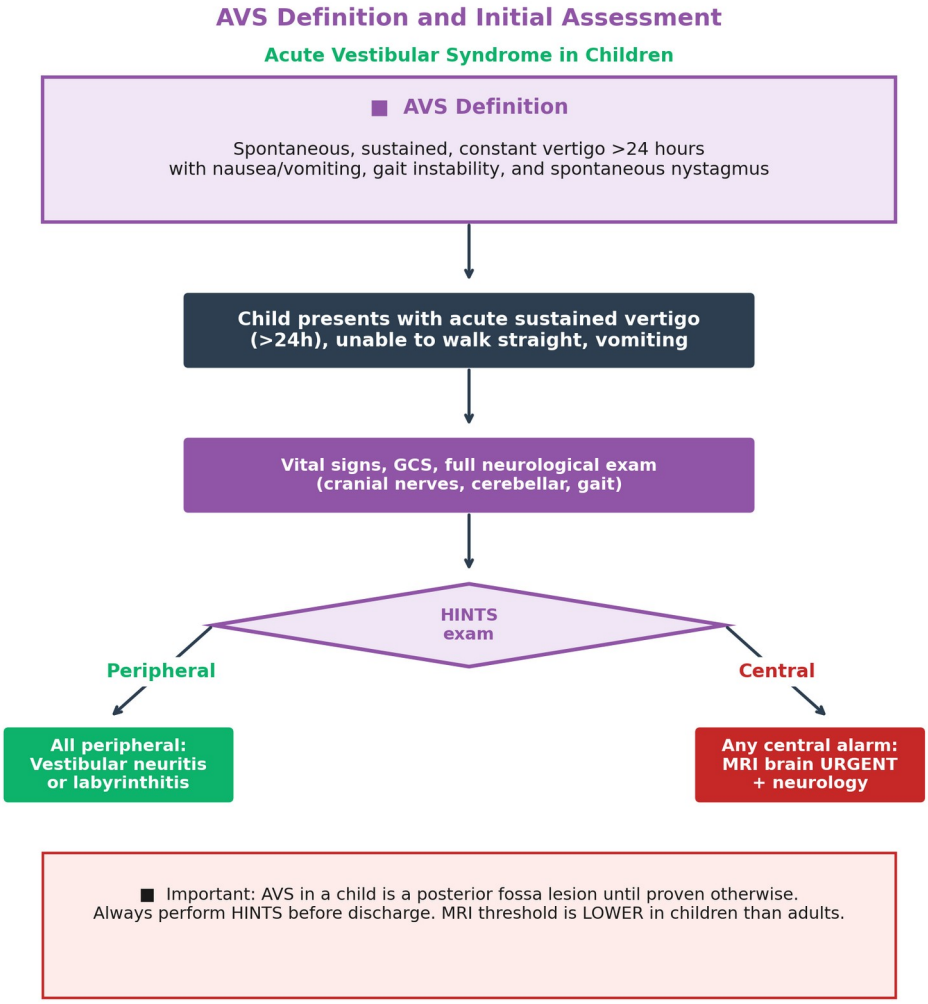

Figure 1. AVS Definition and Initial Assessment — diagnostic framework for paediatric acute vestibular syndrome including HINTS triage.

Source: Australian Dizziness Clinics — clinical flowchart.

II. Epidemiology and Aetiology

Vestibular neuritis is substantially less common in children than in adults, accounting for approximately 5–12% of paediatric vestibular diagnoses in tertiary referral series compared with 15–20% in adult series. Population-based incidence data for paediatric vestibular neuritis are limited, but estimates suggest an incidence of approximately 3–5 per 100,000 children per year. The condition can occur at any age in childhood but is most commonly reported in school-aged children and adolescents; it is uncommon but not unrecognised in children under 5 years. [5,6]

Viral Aetiology

The most widely accepted aetiological hypothesis is reactivation of latent herpes simplex virus type 1 (HSV-1) within the vestibular ganglion (Scarpa's ganglion), producing a ganglionitis that is analogous to the mechanism of Bell's palsy in the facial nerve. This hypothesis, advanced by Arbusow and colleagues, is supported by PCR evidence of HSV-1 DNA in the vestibular ganglia of post-mortem specimens and the observation that the superior vestibular nerve division is more commonly and more severely affected — which may relate to the anatomy of the bony canal through which the superior division passes (the superior vestibular nerve canal is longer and narrower, potentially predisposing to ischaemic damage during inflammation). [3,4]

Other viruses implicated in paediatric vestibular neuritis and labyrinthitis include cytomegalovirus (CMV), Epstein-Barr virus (EBV), influenza, adenovirus, and enterovirus. Seasonal variation is reported, with peaks in spring and autumn corresponding to respiratory viral seasons. Post-infectious labyrinthitis may

follow upper respiratory tract infection or otitis media, with the cochlear component potentially reflecting direct viral spread through the round window membrane or haematogenous seeding. [6,7]

Vascular and Autoimmune Causes

A critical differential diagnosis is anterior inferior cerebellar artery (AICA) territory infarction, which involves the labyrinthine artery as its terminal branch. AICA infarction produces a syndrome that mimics vestibular neuritis with acute vertigo, nausea, and ipsilateral canal hypofunction — but adds ipsilateral SNHL (from cochlear ischaemia) and ipsilateral facial numbness or weakness from brainstem/cerebellar involvement. While AICA stroke is rare in children, it is catastrophic if missed. Autoimmune causes, including Cogan syndrome (keratitis + vestibulo-auditory involvement), are rare in children but should be considered when recurrent episodes occur or systemic inflammatory features are present. [8]

III. Pathophysiology: Viral, Inflammatory, and Vascular Mechanisms

The pathophysiology of vestibular neuritis is best understood through the ganglionitis hypothesis. In this model, reactivation of HSV-1 within the vestibular ganglion triggers an inflammatory response that damages afferent vestibular neurons — predominantly type I hair cell innervation — producing acute unilateral deafferentation of the affected labyrinth. The consequence is a sudden asymmetry in resting discharge rate between the two vestibular nuclei, which the brainstem and cerebellum interpret as continuous head rotation toward the unaffected side. [3,4]

Superior vs Inferior Vestibular Nerve Involvement

The superior division of the vestibular nerve innervates the posterior SCC ampulla, the horizontal SCC ampulla, and the anterior SCC ampulla, as well as the utricular macula. The inferior division innervates the saccular macula and the posterior SCC ampulla. Superior division neuritis is significantly more common, producing horizontal SCC hypofunction (abnormal head impulse on ipsilesional side), anterior SCC hypofunction, and utricular dysfunction (abnormal cervical VEMP). Inferior division neuritis is less common, producing saccular dysfunction (abnormal cervical VEMP) with preserved horizontal SCC function (normal vHIT). This distinction is clinically relevant as isolated inferior division neuritis produces an atypical clinical picture that may be mistaken for central pathology. [9]

Compensation Mechanisms

Following acute unilateral deafferentation, the central nervous system initiates vestibular compensation — a multi-stage process of neuroplastic reorganisation. In the acute phase (days 1–3), spontaneous nystagmus reflects the resting discharge asymmetry. Over the subsequent days to weeks, the contralateral vestibular nucleus upregulates its spontaneous activity (static compensation), abolishing the nystagmus at rest. Dynamic compensation — recovery of the vestibulo-ocular reflex (VOR) gain — occurs over weeks to months and depends on gaze stabilisation exercises, active head movement, and visual and somatosensory inputs. The cerebellar flocculus plays a critical role in adaptive VOR gain recovery. [10]

Labyrinthine Artery and AICA Anatomy

The labyrinthine (internal auditory) artery is typically a branch of the AICA (and occasionally the basilar artery). It is an end artery with no collateral circulation, supplying the entire membranous labyrinth. AICA territory ischaemia may involve the labyrinthine artery, producing cochlear and vestibular infarction, or may preferentially affect the AICA territory proper (lateral pons, cerebellum), producing central vestibular signs in addition to labyrinthine hypofunction. The clinical similarity between AICA infarction and peripheral labyrinthitis underscores the importance of systematic bedside examination in all AVS presentations. [8,11]

□ **Clinical Insight:** Isolated inferior division vestibular neuritis produces a normal vHIT (horizontal SCC unaffected) with abnormal cervical VEMP (saccular hypofunction). This pattern can be mistaken for central pathology. Always combine vHIT with VEMP assessment when clinical features are atypical.

IV. Clinical Features: Neuritis vs Labyrinthitis

The clinical distinction between vestibular neuritis and labyrinthitis rests on the presence or absence of cochlear involvement. Accurate distinction is clinically important, as labyrinthitis demands audiological

follow-up and has a broader differential diagnosis (including AICA territory vascular disease, labyrinthine fistula, autoimmune inner ear disease, and Ménière's disease in adolescents). [12]

Vestibular Neuritis

Vestibular neuritis presents with sudden-onset sustained vertigo, nausea, vomiting, and gait instability — typically following or concurrent with a viral upper respiratory illness. Spontaneous nystagmus is horizontal-torsional, beating away from the affected (hypoactive) side, consistent with Alexander's first law. The nystagmus is direction-fixed and increases in amplitude when gaze is directed toward the fast phase (contralateral side). Cochlear function is normal — no hearing loss, no tinnitus. The head impulse test (HIT) is positive on the ipsilesional side, demonstrating a corrective saccade that confirms peripheral canal hypofunction. Neurological examination is otherwise normal. [2,12]

Labyrinthitis

Labyrinthitis shares all the vestibular features of neuritis but adds cochlear involvement. The hearing loss is typically sudden-onset unilateral SNHL on the affected side, which may be accompanied by tinnitus and aural fullness. The audiogram demonstrates a high-frequency or pan-frequency SNHL on the affected side. In children, even mild hearing loss is clinically significant and requires audiological monitoring for recovery. The vestibular nystagmus pattern is identical to neuritis. [12]

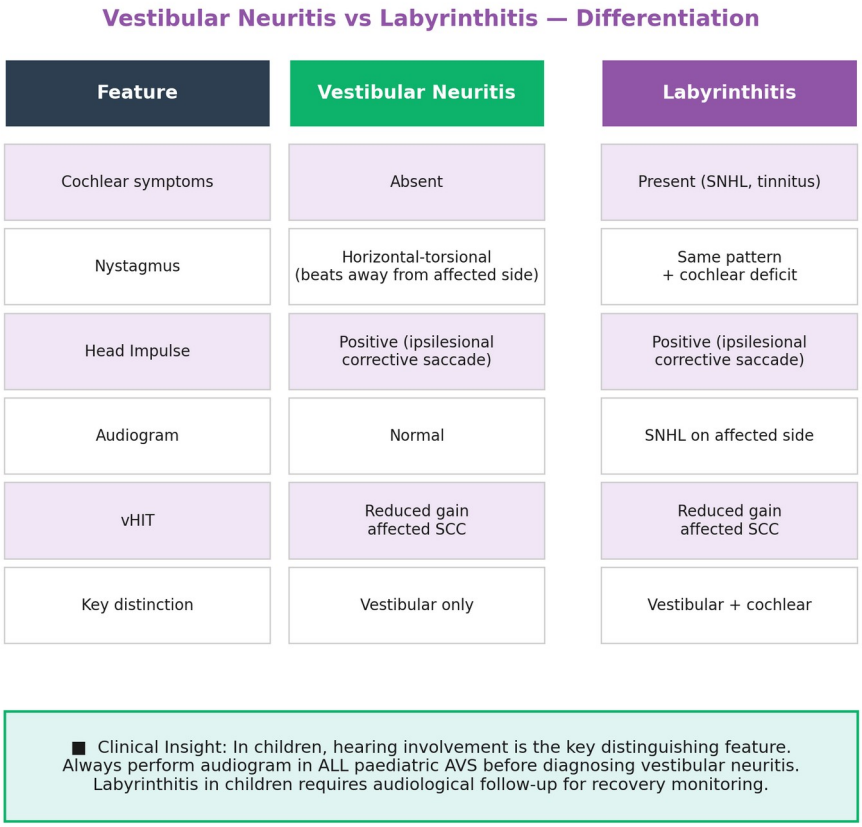

Figure 2. Vestibular Neuritis vs Labyrinthitis — differentiation by cochlear involvement, audiogram findings, and clinical features.

Source: Australian Dizziness Clinics — clinical flowchart.

□ **Clinical Insight:** In children, hearing involvement is the key distinguishing feature between neuritis and labyrinthitis. Always perform an audiogram in ALL paediatric AVS presentations before establishing the diagnosis of vestibular neuritis. A normal audiogram supports neuritis; SNHL indicates labyrinthitis and widens the differential to include AICA territory vascular disease.

V. Bedside Examination: HINTS and Adapted Paediatric Assessment

The HINTS examination (Head Impulse, Nystagmus, Test of Skew) is a bedside oculomotor battery with high sensitivity and specificity for distinguishing peripheral from central AVS in adults. It was originally described and validated in the adult emergency setting, where its sensitivity for posterior circulation stroke exceeds that of early MRI-DWI. However, its application in children requires important caveats. [13,14]

H — Head Impulse Test

The head impulse test (HIT) assesses the horizontal VOR. The clinician holds the child's head and delivers rapid, small-amplitude (15–20 degree) horizontal head rotations. A corrective saccade — observed after the head rotation — indicates that the VOR has failed to keep the eyes stable on the target, confirming ipsilesional canal hypofunction. In peripheral AVS (neuritis/labyrinthitis), the HIT is positive (corrective saccade present) on the affected side. In central AVS, the HIT is typically normal (no saccade), reflecting preserved labyrinthine function with a central lesion distorting the vestibular pathways. Paediatric adaptations include verbal coaching, using a visual target (sticker on the clinician's nose), and ensuring the head turn is rapid — slow turns do not adequately test the high-frequency VOR. [13,15]

I — Nystagmus Direction

In peripheral AVS, nystagmus is direction-fixed — it beats in one direction regardless of gaze direction, consistent with Alexander's law. Direction-changing nystagmus (beating right on right gaze, left on left gaze) is a central alarm sign. Purely vertical nystagmus (upbeat or downbeat) is also a central alarm sign. Positional nystagmus in a child with AVS should increase suspicion for central pathology. Frenzel goggles or video-Frenzel goggles are recommended for nystagmus assessment in children to eliminate fixation suppression and increase the visibility of low-amplitude nystagmus. [2,13]

T — Test of Skew

The alternate cover test detects vertical ocular misalignment (skew deviation). The examiner alternately covers each eye while the child fixates a target. Vertical correction (one eye higher than the other) indicates a skew deviation — a central alarm sign reflecting otolith pathway damage in the brainstem or cerebellum. Skew deviation is uncommon in peripheral AVS. Its presence should prompt urgent MRI even if other HINTS features are peripheral. [13,14]

HINTS Plus: Adding Audiometry

HINTS Plus adds sudden ipsilateral SNHL to the HINTS battery as an additional central alarm sign. New ipsilateral SNHL in AVS raises concern for AICA territory ischaemia (cochlear infarction via the labyrinthine artery). This is critical: sudden vertigo + acute ipsilateral SNHL = AICA territory until proven otherwise, even if the standard HIT appears positive (peripheral). [8,14]

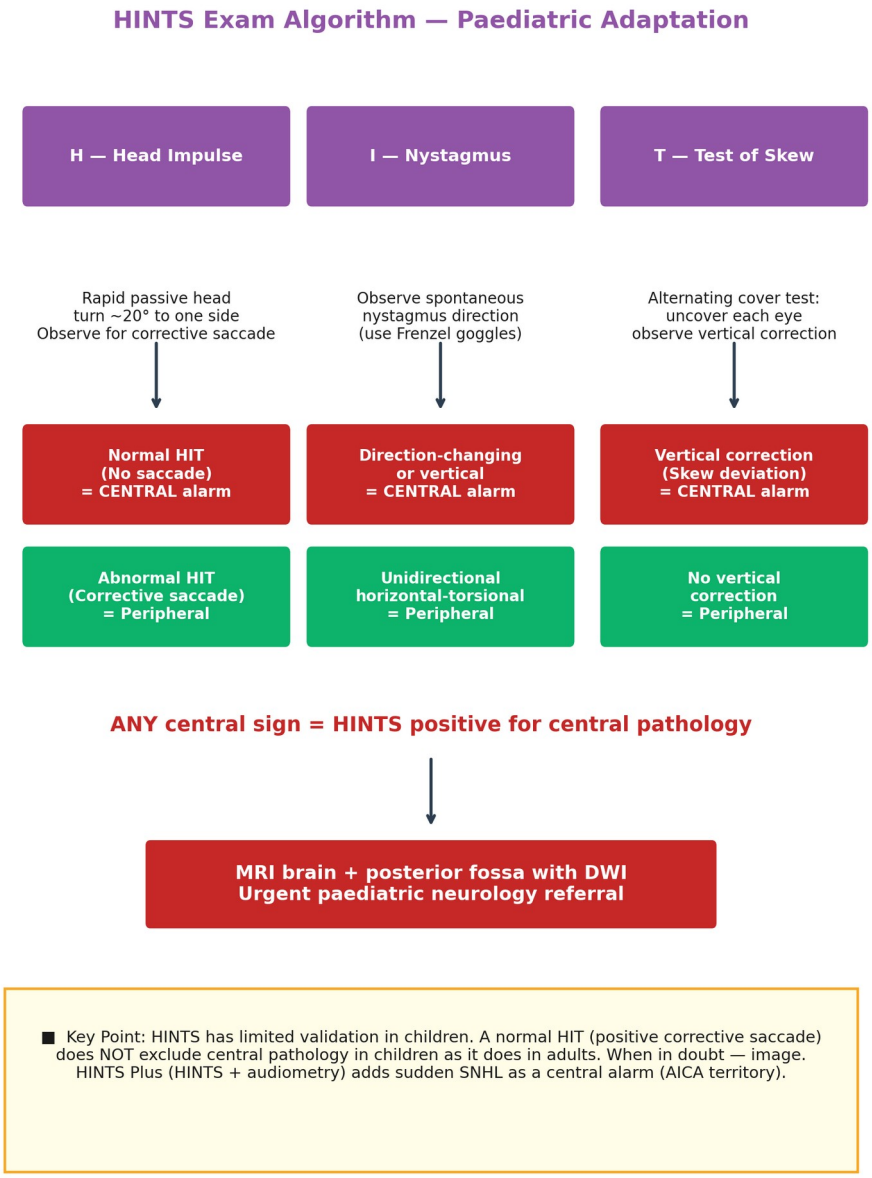

Figure 3. HINTS Exam Algorithm — Paediatric Adaptation — including component-by-component interpretation and central alarm features.

Source: Australian Dizziness Clinics — clinical flowchart.

□ **Important:** HINTS has limited validation in children. A normal corrective saccade on HIT does NOT exclude central pathology in children as it does in adults. When in doubt, always image. A child with AVS and any of the following requires MRI: direction-changing nystagmus, skew deviation, vertical nystagmus, normal HIT, sudden SNHL, or any neurological sign.

VI. Investigations and Differential Diagnosis

Audiogram

An audiogram is mandatory in all paediatric AVS presentations. It differentiates vestibular neuritis (normal hearing) from labyrinthitis (SNHL). A normal audiogram substantially reduces the probability of AICA territory vascular disease and expands the confidence in a peripheral diagnosis. In children under 5, audiogram may require behavioural or objective testing (ABR/ASSR) rather than pure-tone audiometry. [12,15]

Video Head Impulse Test (vHIT)

vHIT provides quantitative assessment of horizontal SCC function (VOR gain) and documents the presence of overt or covert corrective saccades. In vestibular neuritis, vHIT demonstrates reduced VOR gain on the affected side with catch-up saccades. A normal vHIT (preserved VOR gain) in a patient with AVS is a central alarm sign. Normative vHIT data in children are available from approximately 6 years of age; testing in younger children may be challenging due to cooperation. [15,16]

Caloric Testing

Caloric testing (bithermal caloric irrigation) documents the degree of unilateral canal paresis. In vestibular neuritis, caloric testing demonstrates reduced or absent response on the affected side (unilateral canal paresis). Caloric testing is poorly tolerated in young children and is less commonly performed acutely; vHIT is preferred as the initial vestibular function test. [15]

MRI Brain and Posterior Fossa

MRI with DWI (diffusion-weighted imaging) is the definitive investigation to exclude central pathology. In paediatric AVS, MRI should be obtained within 24–48 hours of presentation — or urgently if any central alarm signs are present. DWI has a false negative rate of approximately 20–50% in posterior fossa stroke in the first 24–48 hours, so repeat imaging at 48–72 hours is indicated if clinical suspicion persists despite initial normal DWI. MRI sequences should include DWI (stroke), FLAIR (demyelination), and T1 with gadolinium (tumour/abscess). [11,17]

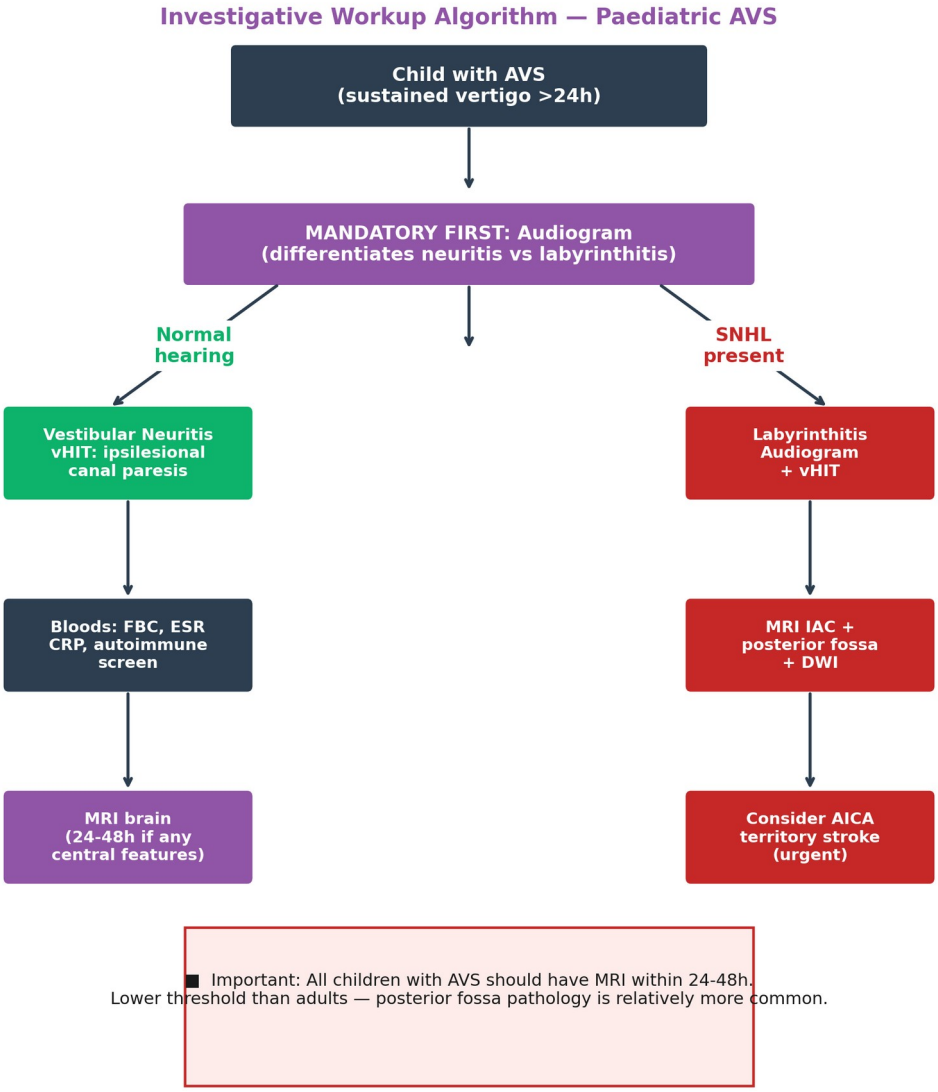

Figure 4. Investigative Workup Algorithm for Paediatric AVS — structured investigation by cochlear involvement, central alarm features, and clinical context.

Source: Australian Dizziness Clinics — clinical flowchart.

Differential Diagnosis

The differential diagnosis of paediatric AVS includes: (1) posterior fossa stroke (AICA, PICA, basilar artery territory); (2) posterior fossa tumour (medulloblastoma, ependymoma, pilocytic astrocytoma) — important in children presenting with insidious or progressive AVS; (3) demyelination (first demyelinating event, ADEM); (4) vestibular migraine (episodic rather than sustained, headache history); (5) BPPV (positional trigger, brief episodes); (6) autoimmune labyrinthitis (Cogan syndrome, vasculitis). The distinction between these conditions drives both urgency of investigation and treatment approach. [18]

□ **Clinical Pearl:** Acute onset vertigo + ipsilateral deafness + sudden onset = AICA territory until proven otherwise. This combination mandates urgent MRI with DWI regardless of bedside HINTS findings. The labyrinthine artery is the terminal branch of the AICA and has no collateral supply — cochlear infarction is the vestibular equivalent of a retinal artery occlusion.

VII. Acute Management

Vestibular Suppressants

Vestibular suppressants reduce the acute severity of vertigo and nausea but do not modify the underlying disease process and — critically — delay vestibular compensation if used for prolonged periods. Current evidence supports their use for a maximum of 2–3 days. Appropriate agents in children include promethazine (12.5–25 mg orally or intramuscularly, age-appropriate dosing) and prochlorperazine. These agents carry extrapyramidal side effect risk in children, and clinicians should be alert to dystonic reactions. [19]

Anti-emetics

Ondansetron is the preferred anti-emetic in children for acute vestibular nausea — it has a favourable side effect profile, is well tolerated, and is available in oral dispersible tablet form for children unable to swallow. Metoclopramide may also be used but carries higher extrapyramidal risk. Anti-emetics should be prescribed for symptomatic control of vomiting but titrated down as the acute phase settles. [19]

Corticosteroids

In adults, a course of methylprednisolone (beginning at 100 mg/day and tapering over 22 days) has been shown to improve recovery of peripheral vestibular function in vestibular neuritis — the Strupp 2004 RCT remains the definitive evidence. Antiviral therapy (valaciclovir) did not add benefit in the same trial. In children, direct evidence for corticosteroids is lacking, but extrapolation from adult data is reasonable in severe paediatric cases, particularly labyrinthitis with SNHL, where treatment of the cochlear component is a priority. The decision to use corticosteroids should be made on a case-by-case basis. [20]

Early Mobilisation and Vestibular Exercises

Early active mobilisation — from day 2 of illness — is critical to initiating vestibular compensation. Prolonged bed rest significantly delays compensation and should be avoided. From day 2–3, simple gaze stabilisation exercises (VOR x1 and x2 protocols — fixating a target during head movement) should be introduced. These exercises drive cerebellar adaptive plasticity and accelerate VOR gain recovery. Return to school should be guided by symptom control, typically within 5–10 days.

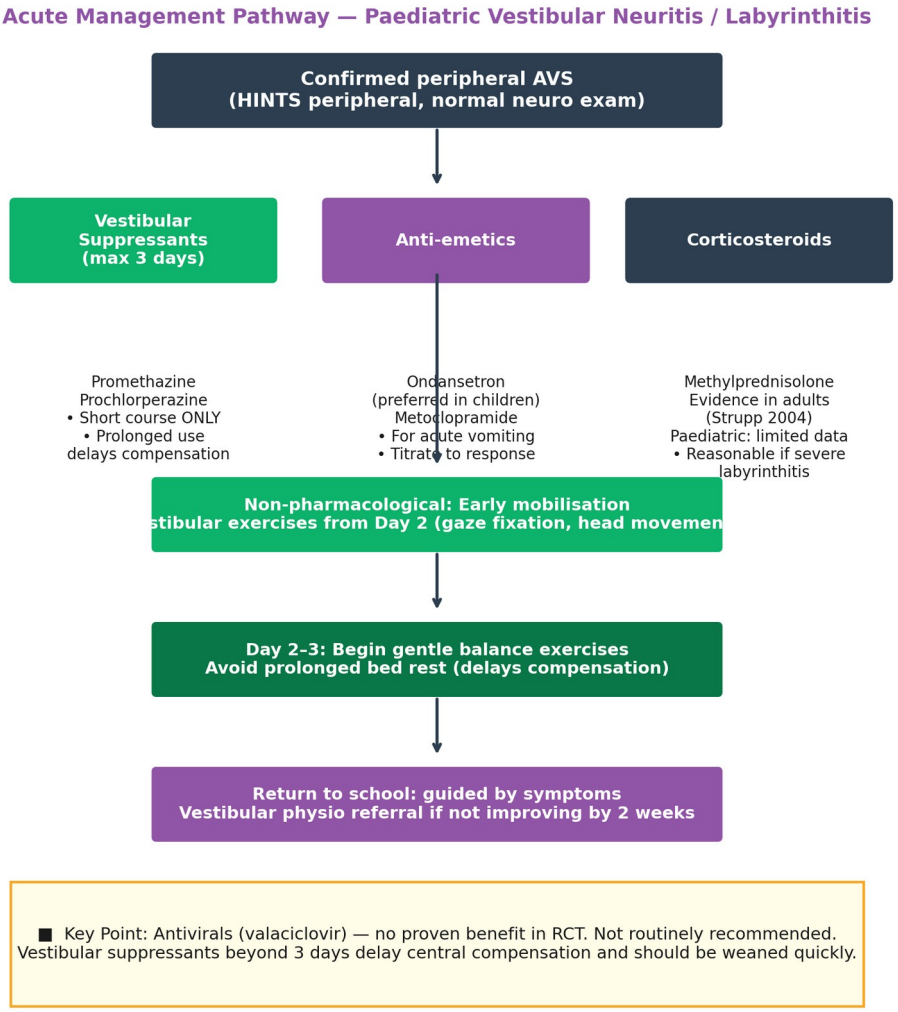

Figure 5. Acute Management Pathway for Paediatric Vestibular Neuritis and Labyrinthitis — pharmacological and non-pharmacological strategies.
Source: Australian Dizziness Clinics — clinical flowchart.

VIII. Vestibular Compensation and Rehabilitation

Vestibular compensation is the process by which the central nervous system recalibrates itself following unilateral vestibular deafferentation, restoring postural stability and gaze control at rest and during head movement. It proceeds in two stages: static compensation (restoration of resting neural symmetry, abolishing spontaneous nystagmus) occurring over days to weeks; and dynamic compensation (recovery of VOR gain and postural reflexes) occurring over weeks to months. Children, with their superior neuroplasticity, typically compensate faster than adults. [10,21]

Role of Sensory Substitution

Compensation relies on the reweighting of visual and somatosensory inputs to substitute for the reduced vestibular input. This process is disrupted by: (1) prolonged vestibular suppression medication; (2) visual or proprioceptive impairment; (3) high levels of anxiety (which activates the amygdala and disrupts cerebellar adaptive mechanisms); and (4) central pathology (which may impair the cerebellum's adaptive capacity). Recognition of these barriers is important for guiding rehabilitation. [10,22]

Cawthorne-Cooksey and Gaze Stabilisation Exercises

The Cawthorne-Cooksey programme is a structured sequence of head, eye, and balance exercises designed to promote vestibular compensation. More specifically, gaze stabilisation exercises (VOR × 1 protocol: fixate a target while moving the head) are the evidence-based core of vestibular rehabilitation post-neuritis. These exercises have been demonstrated in RCTs to accelerate VOR gain recovery and

postural stability. In children, exercises should be introduced by a vestibular physiotherapist at an age-appropriate level from approximately 7–8 years. [21]

Return to School and Sport

Return to school should be guided by symptom control, ability to tolerate head movement, and confidence with gait. Most children are able to return to school within 5–10 days if the acute illness is uncomplicated. Return to sport requires vestibular rehabilitation to an appropriate level of balance and gaze stability. Contact sports should be deferred until dynamic balance has been restored under physiotherapist guidance.

□ **Key Point:** Early mobilisation from day 2 and gaze stabilisation exercises are the cornerstones of vestibular compensation. Vestibular suppressants beyond 3 days delay compensation and should be weaned. Anxiety is a major barrier to compensation in adolescents — early psychological support reduces the risk of persistent postural-perceptual dizziness (PPPD) as a post-neuritis complication.

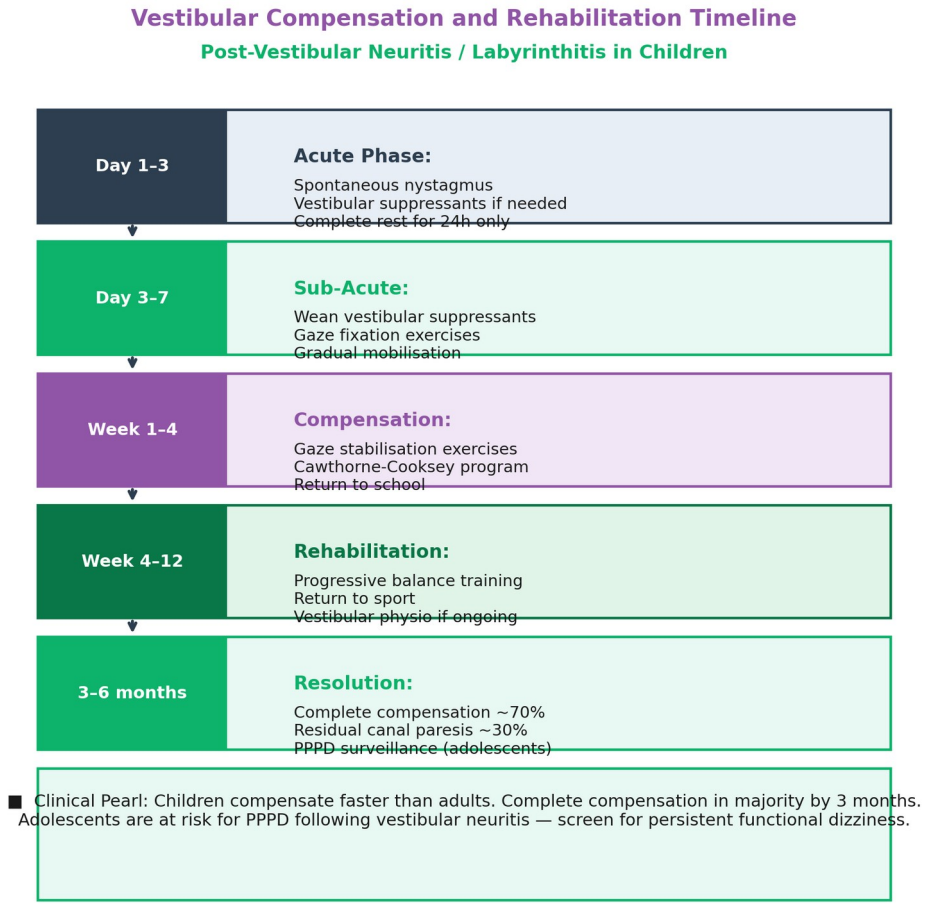

Figure 6. Vestibular Compensation and Rehabilitation Timeline — phase-by-phase recovery pathway post-vestibular neuritis in children.

Source: Australian Dizziness Clinics — clinical flowchart.

IX. Prognosis and Long-Term Outcomes

The prognosis of vestibular neuritis in children is generally excellent. The majority of children achieve complete or near-complete functional compensation within 3–6 months. In contrast to adults, children have superior central neuroplasticity, and complete functional recovery is the expected outcome. [5,22]

Residual Canal Paresis

Objective vestibular function tests demonstrate persistent canal paresis (reduced VOR gain on vHIT) in approximately 30–40% of cases at 6 months, even when symptoms have resolved. This dissociation

between objective paresis and clinical compensation reflects the effectiveness of central adaptation. Persistent canal paresis does not preclude full return to activity but may predispose to reduced tolerance of challenging vestibular environments.

Hearing Recovery in Labyrinthitis

Hearing recovery in labyrinthitis is variable. In viral labyrinthitis, partial or complete recovery occurs in the majority of cases within 6–12 weeks. Early corticosteroid treatment may improve hearing outcomes. Audiological monitoring at 6 weeks, 3 months, and 12 months is recommended for any child with labyrinthitis-associated SNHL. If hearing does not recover, audiological rehabilitation (hearing aids, cochlear implant consideration) should be discussed.

PPPD as a Post-Neuritis Complication

Persistent postural-perceptual dizziness (PPPD) — a functional vestibular disorder characterised by chronic non-spinning dizziness, unsteadiness, and visual vertigo — is increasingly recognised as a post-neuritis complication in adolescents. Anxiety and maladaptive behavioural responses to acute vestibular illness (avoidance of head movement, reduced activity) are key risk factors. Early psychological support, active rehabilitation, and avoidance of prolonged rest can reduce the risk of PPPD developing. Vestibular physiotherapy with a biopsychosocial framework is the primary treatment for established PPPD. [22]

□ **Clinical Pearl:** Adolescents with vestibular neuritis are at risk for PPPD as a post-neuritis complication — particularly if anxiety is prominent, or if recovery is being managed with prolonged rest and activity restriction. Screening for chronic non-spinning dizziness, visual hypersensitivity, and avoidance behaviour at the 4–6 week review is recommended.

X. Summary and Key Clinical Takeaways

Vestibular neuritis and labyrinthitis are the most common peripheral causes of acute vestibular syndrome in children. The following ten clinical takeaways summarise the core messages of this review:

1. AVS in a child is a posterior fossa lesion until proven otherwise — always perform HINTS and consider MRI before discharging.

2. HINTS has limited paediatric validation. A normal corrective saccade does NOT exclude central pathology in children as it does in adults — err on the side of imaging.

3. Audiogram is mandatory in all paediatric AVS. Normal hearing = neuritis. SNHL = labyrinthitis (and widens the differential).

4. Sudden vertigo + ipsilateral SNHL = AICA territory until proven otherwise. Urgent MRI required regardless of HIT findings.

5. Vestibular suppressants should not be used beyond 2–3 days — they delay compensation. Wean early and start mobilisation from day 2.

6. Corticosteroids may be considered for severe cases or labyrinthitis with SNHL. Evidence is extrapolated from adult data (Strupp 2004). Antivirals have no proven benefit.

7. Early mobilisation and gaze stabilisation exercises are the cornerstones of vestibular compensation. Vestibular physiotherapy should be commenced within the first 1–2 weeks.

8. Children compensate faster than adults — complete recovery is expected in the majority within 3–6 months.

9. Residual canal paresis on vHIT in an asymptomatic child is common (30–40%) and does not require further treatment if functionally compensated.

10. Adolescents are at risk for PPPD post-neuritis. Screen at 4–6 weeks for persistent functional dizziness and institute early psychological support if indicated.

This review is the fifth in the Vestibular Medicine in Children series. The next review (PVM06) covers Central Causes of Vestibular Dysfunction in Children, including posterior fossa tumours, posterior circulation stroke, and demyelinating disease.

References

[1] Edlow JA, Newman-Toker DE, Savitz SI. Diagnosis and initial management of cerebellar infarction. Lancet Neurol. 2008;7(10):951–64.

[2] Brandt T, Dieterich M. Vertigo and dizziness: common complaints. Springer; 2004.

[3] Arbusow V, Schulz P, Strupp M, et al. Distribution of herpes simplex virus type 1 in human geniculate and vestibular ganglia: implications for vestibular neuritis. Ann Neurol. 1999;46(3):416–9.

[4] Strupp M, Brandt T. Vestibular neuritis. Semin Neurol. 2009;29(5):509–19.

[5] Russell G, Abu-Arafeh I. Paroxysmal vertigo in children — an epidemiological study. Int J Pediatr Otorhinolaryngol. 1999;49 Suppl 1:S105–7.

[6] Choung YH, Park K, Moon SK, Kim CH, Ryu SJ. Various causes and clinical characteristics in vertigo in children with normal eardrums. Int J Pediatr Otorhinolaryngol. 2003;67(8):889–94.

[7] Balatsouras DG, Kaberos A, Assimakopoulos D, Katotomichelakis M, Economou NC, Korres SG. Etiology of vertigo in children. Int J Pediatr Otorhinolaryngol. 2007;71(3):487–94.

[8] Kim JS, Lee H. Inner ear disease and Bayesian approach to determine the locus of damage. Otol Neurotol. 2012;33(4):e23–5.

[9] Murofushi T, Halmagyi GM, Yavor RA, Colebatch JG. Absent vestibular evoked myogenic potentials in vestibular neurolabyrinthitis: an indicator of inferior vestibular nerve involvement? Arch Otolaryngol Head Neck Surg. 1996;122(8):845–8.

[10] Lacour M, Tighilet B. Plastic events in the vestibular nuclei during vestibular compensation: the brain orchestration of a "deafferentation" code. Restor Neurol Neurosci. 2010;28(1):19–35.

[11] Chalela JA, Kidwell CS, Nentwich LM, et al. Magnetic resonance imaging and computed tomography in emergency assessment of patients with suspected acute stroke: a prospective comparison. Lancet. 2007;369(9558):293–8.

[12] Baloh RW. Clinical practice. Vestibular neuritis. N Engl J Med. 2003;348(11):1027–32.

[13] Kattah JC, Talkad AV, Wang DZ, Hsieh YH, Newman-Toker DE. HINTS to diagnose stroke in the acute vestibular syndrome: three-step bedside oculomotor examination more sensitive than early MRI diffusion-weighted imaging. Stroke. 2009;40(11):3504–10.

[14] Newman-Toker DE, Kerber KA, Hsieh YH, et al. HINTS outperforms ABCD2 to screen for stroke in acute continuous vertigo and dizziness. Acad Emerg Med. 2013;20(10):986–96.

[15] Dhondt C, Dhooge I, Maes L. Paediatric vestibular assessment. Acta Otolaryngol. 2020;140(4):310–6.

[16] Valente M. Maturational effects of the vestibular system: a study of rotary chair, computerized dynamic posturography, and vestibular evoked myogenic potentials with children. J Am Acad Audiol. 2007;18(6):461–81.

[17] Tarnutzer AA, Berkowitz AL, Robinson KA, Hsieh YH, Newman-Toker DE. Does my dizzy patient have a stroke? A systematic review of bedside diagnosis in acute vestibular syndrome. CMAJ. 2011;183(9):E571–92.

[18] Bronstein AM, Lempert T. Dizziness: a practical approach to diagnosis and management. Cambridge University Press; 2007.

[19] Seemungal BM, Bronstein AM. A practical approach to acute vertigo. Pract Neurol. 2008;8(4):211–21.

[20] Strupp M, Zingler VC, Arbusow V, et al. Methylprednisolone, valacyclovir, or the combination for vestibular neuritis. N Engl J Med. 2004;351(4):354–61.

[21] Hillier SL, McDonnell M. Vestibular rehabilitation for unilateral peripheral vestibular dysfunction. Cochrane Database Syst Rev. 2011;(2):CD005397.

[22] Staab JP. Chronic subjective dizziness. Continuum (Minneap Minn). 2012;18(5 Neuro-otology):1118–41.

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