Sudden Sensorineural Hearing Loss with Vertigo:

An Otological Emergency for General Clinicians

Vestibular Medicine for General Clinicians

Topic 11 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. Why This Presentation Is a True Emergency

Sudden sensorineural hearing loss (SSNHL) is an otological emergency. When accompanied by vertigo, the combination carries both a narrower therapeutic window and a higher risk of serious underlying pathology. Outcomes are strongly time-dependent: treatment initiated within 24–48 hours of onset yields the best chance of hearing recovery; the window effectively closes at around 14 days, and meaningful benefit is unlikely after 4–6 weeks [1,2].

Despite this urgency, SSNHL is among the most under-recognised emergency presentations in general practice and emergency medicine. Australian and international audit data consistently show that fewer than one in three patients with SSNHL receive appropriate corticosteroid therapy within the evidence-based 72-hour window [3,4]. The commonest reason is failure of the first-contact clinician to identify the loss as sensorineural and urgent — patients are frequently reassured, referred non-urgently, or treated for presumed eustachian tube dysfunction or wax.

□ **Key Point:** SSNHL with vertigo is an otological emergency equivalent to sudden vision loss. Oral high-dose corticosteroid should be commenced within 72 hours of onset, ideally at the first medical contact, while urgent audiology and otolaryngology review are arranged.

Prognosis is worse when SSNHL is accompanied by vertigo. Vertigo reflects labyrinthine involvement and predicts poorer hearing recovery — approximately 40–50 percent recovery compared with 60–70 percent in SSNHL without vertigo [5]. The presence of vertigo also widens the differential to include posterior circulation stroke (particularly AICA territory), vestibular schwannoma, and autoimmune inner ear disease, each of which can present identically and each of which has distinct management.

II. Definitions and Diagnostic Criteria

The American Academy of Otolaryngology–Head and Neck Surgery Foundation 2019 Clinical Practice Guideline (CPG) update defines sudden sensorineural hearing loss as [6]:

- A sensorineural hearing loss of at least 30 dB over three contiguous audiometric frequencies,
- Developing over a period of 72 hours or less,
- In the absence of a known cause after initial evaluation.

Where audiometry is not immediately available, a presumed SSNHL diagnosis may be made on the basis of unilateral hearing loss of abrupt onset plus an abnormal tuning fork examination localising the loss to the sensorineural pathway.

Sudden vs Subacute

Hearing loss developing over more than 72 hours but less than a few weeks is termed rapidly progressive sensorineural hearing loss — prognosis and workup differ slightly but the same urgent principles apply.

Unilateral vs Bilateral

SSNHL is unilateral in over 95 percent of cases. Bilateral or sequential SSNHL is a red flag for autoimmune, infective, malignant, or syndromic aetiology and mandates urgent expert workup regardless of vertigo [7].

Table 1 — Red Flags in Sudden Hearing Loss Requiring Same-Day Escalation

Red flag	Concern
Accompanying vertigo or ataxia	Labyrinthitis, AICA stroke, vestibular schwannoma
Any new neurological sign (diplopia, dysarthria, weakness, cerebellar signs)	Posterior circulation stroke — urgent stroke pathway
Severe occipital headache or neck pain	Vertebral artery dissection
Bilateral or sequential SSNHL	Autoimmune inner ear disease, meningitis, malignancy

Red flag	Concern
Fever, rash, meningism	Meningitis, viral labyrinthitis
History of head trauma or barotrauma	Perilymph fistula, labyrinthine concussion
Profound (over 90 dB) SSNHL	Worse prognosis, urgent steroid trial mandatory

Any single red flag warrants same-day ENT or emergency department assessment, not next-available outpatient review.

□ **Important:** A patient with SSNHL plus any central neurological symptom must be treated as a suspected stroke until imaging excludes it. AICA infarction commonly presents with unilateral hearing loss AND vertigo AND facial weakness — a pattern that can be mistaken for labyrinthitis. Early MRI with DWI is mandatory.

III. Differential Diagnosis

Fewer than 15 percent of cases of SSNHL have an identified cause at the time of presentation. The remaining 85 percent are classified as idiopathic SSNHL and managed as such. Nonetheless, the identifiable causes must be actively considered because they change management materially.

Table 2 — Differential Diagnosis of SSNHL with Vertigo

Cause	Clinical clues	Key management step
Idiopathic SSNHL (majority)	No identifiable cause after workup	High-dose corticosteroid; urgent audiology and ENT review
Viral labyrinthitis	Preceding URTI; may have fever	Corticosteroid; supportive care
AICA territory stroke	Vertigo, hearing loss, facial weakness, cerebellar signs, vascular risk	Immediate stroke pathway; MRI-DWI
Vestibular schwannoma	Progressive or stepwise hearing loss; asymmetric tinnitus	MRI IAM with gadolinium
Autoimmune inner ear disease	Bilateral or sequential loss; other autoimmune history	Corticosteroid; ENT and rheumatology review
Meningitis (bacterial, TB, fungal)	Fever, headache, meningism	Emergency admission; LP; antibiotics
Ménière's disease (first attack mimic)	Hours of vertigo, aural fullness, tinnitus; may recover	Differentiated by recovery and pattern over time
Perilymph fistula	Trauma, barotrauma, Valsalva	ENT review; bed rest; possible surgical exploration
Ototoxicity	Recent aminoglycosides, cisplatin, salicylates, loop diuretics	Cease offending agent
Multiple sclerosis	Young adult; other neurological features	MRI brain plus cord; neurology review
Syphilis / Lyme / HIV	Risk factors, systemic features	Targeted serology

A full history plus audiogram plus MRI IAM with gadolinium identifies most specific causes. Treatment with high-dose oral corticosteroid should not be delayed while awaiting these results.

□ **Clinical Insight:** Because idiopathic SSNHL is by far the most common cause and because the treatment (oral corticosteroid) is the same as for most identified causes, the decision to start steroid can almost always precede the completed workup. Start the steroid; arrange the audiogram, MRI and

ENT review in parallel.

IV. First-Contact Clinical Assessment

The immediate task of the first-contact clinician (GP, ED doctor, hospital generalist) is to (1) identify that the hearing loss is sensorineural rather than conductive, (2) identify any red flags indicating central pathology, and (3) commence oral corticosteroid within the 72-hour window unless contraindicated.

History

- Exact onset (minute, hour, day). Was it noticed on waking, on lifting the phone, or developing over hours?
- Progression since onset (stable, improving, worsening, fluctuating).
- Vertigo: duration, spontaneous or triggered, currently present?
- Tinnitus or aural fullness in the affected ear.
- Preceding URTI, headache, neck pain, trauma, barotrauma, loud noise exposure.
- Past hearing or vestibular problems; family history of hearing loss.
- Vascular risk factors, medications (especially ototoxic), autoimmune history.

Examination

- Otoscopy — exclude wax, effusion, perforation, cholesteatoma. A normal drum in the presence of hearing loss points to SSNHL.
- Tuning fork tests — Rinne and Weber with 512 Hz fork (see below).
- Vestibular screen — spontaneous nystagmus, head impulse test, gaze-evoked nystagmus, test of skew (HINTS).
- Cerebellar examination — finger-nose, heel-shin, tandem gait.
- Cranial nerves — particularly facial nerve (AICA involves CN VII).
- Blood pressure plus pulse plus general cardiovascular examination.

Tuning Fork Interpretation

Table 3 — Tuning Fork Tests in Suspected SSNHL

Test	SSNHL (sensorineural)	Conductive loss
Weber (512 Hz on forehead)	Lateralises to the GOOD ear	Lateralises to the AFFECTED ear
Rinne (affected ear)	Air conduction louder than bone (positive)	Bone conduction louder than air (negative)
Rinne (good ear)	Air conduction louder than bone (positive)	Air conduction louder than bone (positive)

Tuning fork findings are imperfect but, combined with otoscopy, reliably distinguish sensorineural from conductive loss at the bedside.

□ **Clinical Pearl:** A normal eardrum on otoscopy combined with a Weber that lateralises to the GOOD ear is sufficient to commence steroid therapy at first contact in suspected SSNHL. Do not wait for formal audiometry to start treatment.

HINTS Examination in the Setting of SSNHL

In a patient with SSNHL plus vertigo, the HINTS examination is used to assess the vestibular component, but is interpreted with caution. AICA infarction can produce a peripheral HINTS pattern via labyrinthine artery involvement — so a peripheral pattern in this setting does not exclude central pathology.

V. Treatment — Oral Corticosteroid Within 72 Hours

The 2019 AAO-HNSF CPG update makes the following strong recommendations for idiopathic SSNHL [6]:

- Offer corticosteroid therapy to patients presenting within 2 weeks of onset.
- Oral prednisolone 1 mg/kg/day (maximum 60 mg/day) for 7–14 days, followed by a short taper.
- Offer intratympanic steroid as a salvage option to patients with incomplete recovery at 2–6 weeks despite initial oral steroid.
- Offer hyperbaric oxygen therapy within 3 months as an adjunct in centres where available (weaker recommendation).

Practical Australian Prescribing

- **First-line:** Prednisolone 1 mg/kg/day orally (capped at 60 mg) in the morning for 7 days.
- **Taper:** 50 mg x 2 days, 40 mg x 2 days, 30 mg x 2 days, 20 mg x 2 days, 10 mg x 2 days. Shorter tapers (5–7 days) are acceptable.
- **Gastroprotection:** Co-prescribe a proton-pump inhibitor for the duration of steroid treatment.
- **Glycaemic monitoring:** Advise diabetic patients to increase home glucose monitoring; liaise with GP/endocrinology if insulin adjustment required.
- **Infection screen:** Consider TB risk in at-risk populations; screen for active infection.

Contraindications and Cautions

- Active systemic infection — seek urgent expert ENT advice.
- Poorly controlled diabetes — use lower dose or intratympanic route; liaise with ENT.
- Active peptic ulcer, severe osteoporosis, uncontrolled psychiatric illness — discuss risk-benefit.
- Pregnancy — prednisolone is used where benefit outweighs risk; consult obstetric team.

□ **Important:** Do NOT withhold systemic corticosteroid on the basis of relative cautions such as well-controlled diabetes or hypertension without first discussing with the on-call ENT team. The evidence for corticosteroid in SSNHL is strong, and the consequence of delay is permanent hearing loss.

Intratympanic Steroid

Intratympanic dexamethasone or methylprednisolone is used in three contexts: as primary therapy where oral steroid is contraindicated, as combined (concurrent) therapy with oral steroid in severe or profound loss, and as salvage therapy for incomplete recovery. It is administered by ENT and requires same-week access [12,13].

□ **Clinical Insight:** In Australia, most tertiary ENT services accept same-day referrals for SSNHL. When phoning the registrar, specifically state "sudden sensorineural hearing loss with vertigo, needing urgent audiogram and consideration of intratympanic steroid". This phrasing substantially shortens time to review.

VI. Investigations

The AAO-HNSF 2019 guideline is explicit that routine laboratory workup is NOT recommended in idiopathic SSNHL [6]. Investigations should be targeted by history and examination.

Mandatory

- **Pure-tone audiogram —** within 14 days; confirms diagnosis and severity; baseline for response assessment.
- **MRI internal auditory meati with gadolinium —** for ALL patients with SSNHL, to exclude vestibular schwannoma (identified in 3–5 percent of otherwise idiopathic cases [8]) and retrocochlear pathology.

Same-Day Imaging (MRI brain with DWI plus MRA)

- Any neurological sign beyond the vertigo itself.
- New occipital headache or neck pain.

- Central features on HINTS examination (normal head impulse, direction-changing nystagmus, skew deviation).
- Significant vascular risk profile in a patient presenting acutely.

Additional Targeted Workup (ENT-led)

- Full blood count, ESR, CRP — screen for infection, inflammation.
- Autoimmune screen (ANA, ANCA, anti-cardiolipin) — bilateral / sequential / young patient.
- Syphilis serology, HIV, Lyme serology — risk factors.
- Fasting lipids plus HbA1c — vascular risk management.
- Vestibular function tests — after the acute phase, where persistent imbalance.

□ **Key Point:** Every patient with SSNHL must have an MRI IAM with gadolinium at some point in the pathway. Do not assume normal hearing recovery rules out vestibular schwannoma — it does not.

VII. Central Causes — Recognising AICA Stroke

Anterior inferior cerebellar artery (AICA) infarction is the principal stroke pattern that presents with sudden hearing loss plus vertigo. The AICA supplies the inner ear (via the labyrinthine artery), the lateral pons, the cerebellar flocculus, and part of the lateral cerebellar hemisphere. An AICA infarct therefore produces [9]:

- Sudden unilateral hearing loss (the feature that brings the patient to hearing attention).
- Vertigo and nausea (from labyrinthine plus vestibular nucleus involvement).
- Ipsilateral facial weakness (CN VII nucleus, lower motor neuron pattern).
- Ipsilateral facial sensory loss (CN V nucleus).
- Ipsilateral limb ataxia (cerebellar).
- Horner's syndrome, contralateral limb sensory loss (may occur).

The labyrinthine symptoms can dominate the presentation, especially if the facial and cerebellar features are subtle. An AICA infarct masquerading as labyrinthitis is a well-described diagnostic pitfall and a major source of missed stroke in emergency medicine [10].

HINTS Examination in SSNHL with Vertigo

In a patient with SSNHL and vertigo, HINTS is used to assess the vestibular component:

- **Head impulse test NORMAL plus** direction-changing nystagmus OR skew deviation = central (AICA) until proven otherwise.
- **Head impulse test ABNORMAL (catch-up saccade) plus** unidirectional nystagmus plus no skew = peripheral (labyrinthitis pattern) — but does NOT rule out AICA, because AICA can produce a peripheral HINTS pattern via labyrinthine artery involvement.

□ **Important:** Unlike in isolated acute vestibular syndrome, a peripheral HINTS pattern in the presence of sudden hearing loss does NOT reliably rule out AICA infarction. Any patient with SSNHL plus vertigo plus vascular risk factors warrants MRI brain with DWI regardless of HINTS findings.

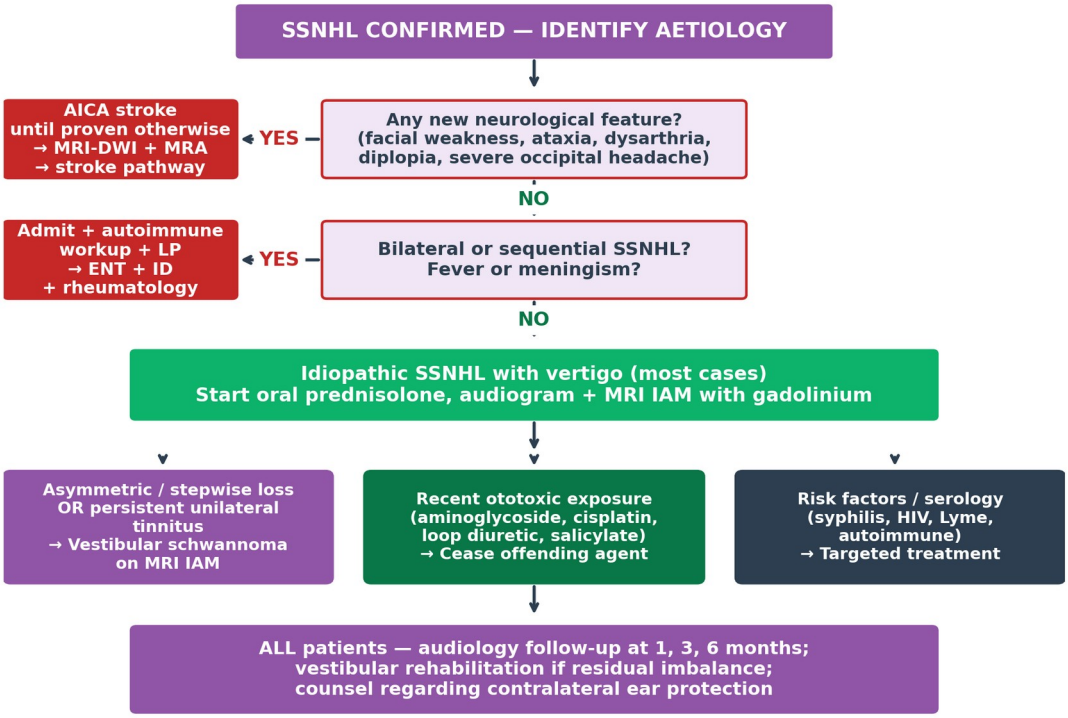

Figure 2. Aetiological branching after SSNHL is confirmed — central first, then specific peripheral causes.
Source: Australian Dizziness Clinics — clinical algorithm.

VIII. Referral and Follow-Up Pathway

SSNHL with vertigo requires the coordination of three services in the first 72 hours: an audiology service to document the loss, an otolaryngology service to confirm the diagnosis and consider intratympanic steroid, and (where central features exist) a neurology/stroke service to exclude posterior circulation pathology.

Same-Day Actions

- Commence oral prednisolone 1 mg/kg/day unless contraindicated.
- Arrange urgent audiometry within 48 hours.
- Phone on-call ENT registrar at the nearest tertiary service.
- If any central feature: direct transfer to ED with neurology/stroke capacity for MRI-DWI.
- Counsel patient to return immediately if any new neurological symptom develops.

Within 2 Weeks

- ENT review with repeat audiogram.
- Consider intratympanic steroid salvage if incomplete recovery.
- MRI internal auditory meati with gadolinium (if not already obtained).
- Vestibular assessment if vertigo persists.

Beyond the Acute Phase

- Audiology follow-up at 1, 3 and 6 months.
- Hearing aid fitting where residual loss affects function.
- Vestibular rehabilitation if residual imbalance or oscillopsia.

- Counselling regarding protection of the contralateral ear (noise avoidance, prompt review of any hearing change).
- Tinnitus management where relevant.

□ **Clinical Insight:** Residual vestibular hypofunction after SSNHL with vertigo is common and responds well to vestibular rehabilitation. Do not assume the vertigo has gone when the patient stops falling over — formal assessment and rehabilitation measurably improve long-term balance and gaze-stability outcomes [11].

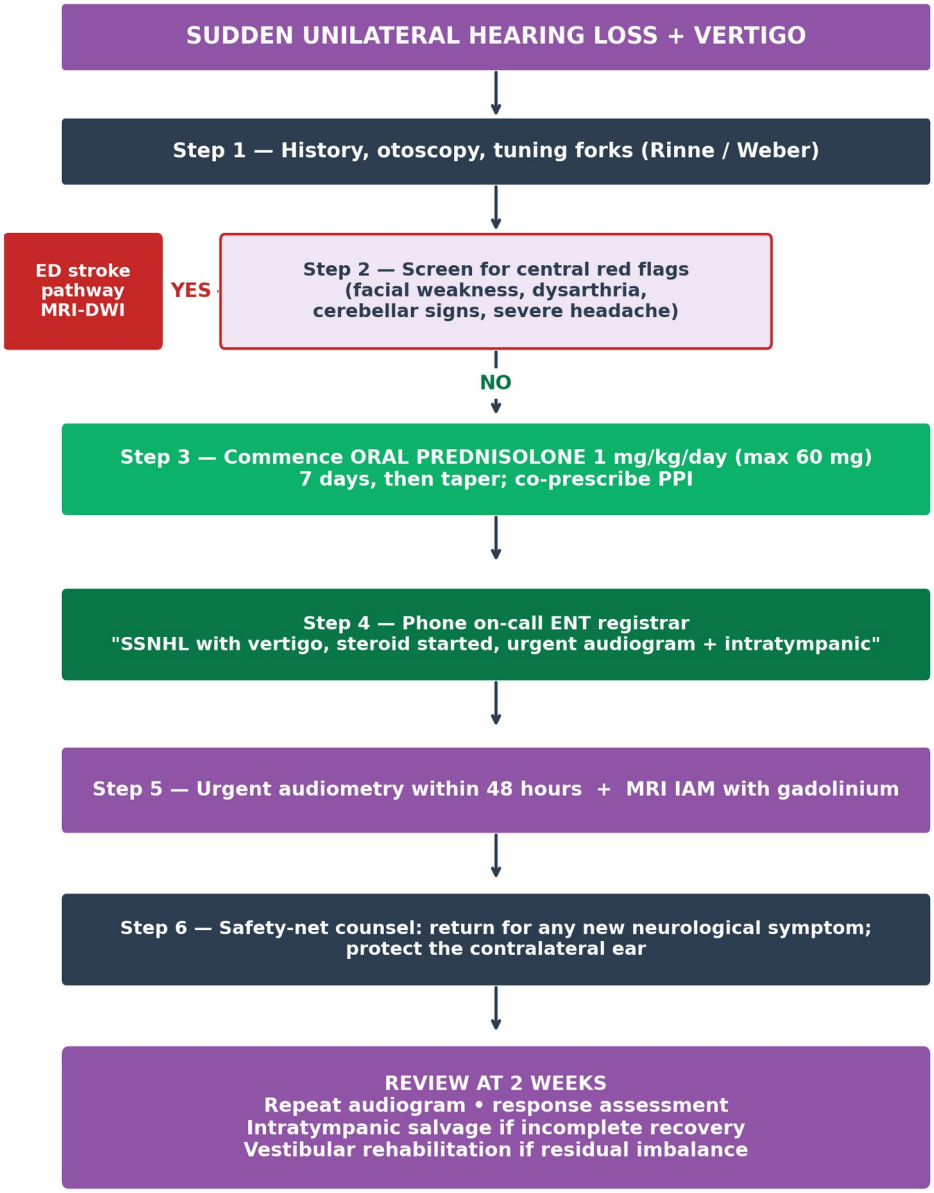

Figure 1. First-72-Hour Pathway for Sudden Sensorineural Hearing Loss with Vertigo — for any first-contact clinician.

Source: Australian Dizziness Clinics — clinical flowchart.

IX. Prognosis and Patient Counselling

Across pooled data, approximately 32–65 percent of patients with idiopathic SSNHL recover some hearing spontaneously, and systemic corticosteroid within the 72-hour window increases this to 50–75 percent [1,6]. The presence of vertigo reduces recovery rates by approximately 10–20 percentage points.

Prognostic Factors — Unfavourable

- Severe or profound loss at presentation (90 dB or worse).
- Presence of vertigo.
- Down-sloping audiometric pattern (high-frequency loss).
- Delay to treatment beyond 7–14 days.
- Advanced age.
- Diabetes mellitus.
- Cardiovascular disease.

Counselling Points

- The window for optimal treatment is the first 72 hours; every day of delay reduces the chance of recovery.
- Most recovery occurs in the first 2 weeks; further recovery up to 6 months is possible but less common.
- The contralateral (good) ear must be protected — avoid loud noise, prompt review of any change.
- Steroid side effects (insomnia, mood change, glycaemic rise, fluid retention) are usually self-limiting.
- Residual tinnitus or vertigo may require dedicated rehabilitation; most patients improve with time.

□ **Key Point:** The single most important message for patients, family, and first-contact clinicians: any sudden hearing loss, with or without vertigo, is an otological emergency on a par with sudden vision loss. Same-day medical contact and same-week expert review are standard of care — not aspirational.

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