Ménière's Disease:

Recognising and Managing Episodic Vertigo with Cochlear Symptoms

Vestibular Medicine for General Clinicians

Topic 7 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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Ménière's disease is a chronic inner ear disorder defined by recurrent attacks of episodic vertigo, fluctuating sensorineural hearing loss, tinnitus, and aural fullness on the affected side. It accounts for approximately 5–10 percent of patients seen in tertiary dizziness clinics, with an estimated prevalence of 200–500 cases per 100,000 in Western populations [1,2]. The peak age of onset is the fourth to sixth decades, with women slightly more often affected.

From a generalist perspective, the diagnosis matters because it sits squarely in the high-impact zone: episodes are disabling, the hearing loss is frequently progressive, and patients commonly present multiple times before a working diagnosis is reached. Average diagnostic delay is between two and four years from symptom onset [3]. The clinical task is to recognise the symptom pattern, document a low-frequency sensorineural hearing loss audiometrically, and refer for confirmation and longitudinal management before the disease damages residual function.

□ Ménière's disease: Episodic spontaneous vertigo (20 minutes to 12 hours), fluctuating low-frequency sensorineural hearing loss, tinnitus, and aural fullness — recurring on the same ear. Diagnosis is by Bárány Society 2015 criteria; audiometry during or close to an attack is the single most useful objective test.

Untreated, the disease tends to a vestibular and cochlear burnout phase after 5–15 years, with eventual stabilisation of vertigo at the cost of permanent hearing loss [4]. Recognising and starting treatment early reduces both the severity and frequency of attacks and improves quality-of-life outcomes.

II. Pathophysiology — Endolymphatic Hydrops

Ménière's disease is histopathologically characterised by endolymphatic hydrops — distension of the membranous labyrinth by excess endolymph. The cochlear duct (scala media) and saccule are most often affected, producing the cochlear and vestibular symptoms respectively [5]. Hydrops can now be visualised in vivo using gadolinium-enhanced delayed MRI of the inner ear, although this remains a research and tertiary tool [6].

Why hydrops produces episodic rather than continuous symptoms is incompletely understood. The leading mechanism is rupture or rapid leakage of endolymph into perilymph, transiently disrupting the ionic gradient required for hair-cell signalling and the resting potential of the cochlear partition. Recovery as the gradient is restored produces the characteristic resolution of vertigo and the partial recovery of hearing between attacks.

Anatomy in Brief

- Endolymph fills the membranous labyrinth (cochlear duct, utricle, saccule, semicircular canals); high in potassium, low in sodium.
- Perilymph fills the bony labyrinth around it; biochemically similar to cerebrospinal fluid.
- The endolymphatic sac is the site of fluid resorption — its dysfunction is implicated in hydrops.
- The mixed sensory load explains the symptom tetrad: cochlear (hearing, tinnitus), saccular (aural fullness), utricular and canal (vertigo, drop attacks).

Aetiology is multifactorial and not fully understood. Recognised contributors include genetic susceptibility (familial forms in 7–10%), autoimmune mechanisms in a subset, prior viral or otologic insult, migraine, and endolymphatic sac dysfunction [7,8]. In clinical practice the cause is rarely identified in an individual patient — diagnosis and management remain phenotypic.

□ Bilateral disease: Up to 30 percent of patients develop bilateral involvement over time. Onset is usually unilateral; bilateral disease at presentation, or rapid contralateral progression, raises suspicion for autoimmune inner ear disease, syphilis, or other systemic causes — investigate accordingly and refer urgently.

III. Clinical Features and Diagnostic Criteria

Diagnosis is clinical, established by the Bárány Society / AAO-HNS 2015 consensus criteria [9]. These criteria distinguish definite from probable Ménière's disease based on the documented combination of vertigo episodes, audiometric hearing loss, and accompanying cochlear symptoms.

Table 1. Bárány Society 2015 diagnostic criteria for Ménière's disease.

Definite Ménière's Disease (all criteria)	**1. Two or more spontaneous episodes of vertigo, each lasting 20 minutes to 12 hours.** **2. Audiometrically documented low- to medium-frequency sensorineural hearing loss in one ear, defining the affected ear, on at least one occasion before, during, or after one of the vertigo episodes.** **3. Fluctuating aural symptoms (hearing, tinnitus, fullness) in the affected ear.** **4. Not better accounted for by another vestibular diagnosis.**
Probable Ménière's Disease	1. Two or more episodes of vertigo or dizziness, each lasting 20 minutes to 24 hours. 2. Fluctuating aural symptoms (hearing, tinnitus, fullness) in the affected ear. 3. Not better accounted for by another vestibular diagnosis.
Audiometric criteria	Sensorineural hearing loss with thresholds at 2 contiguous frequencies below 2 kHz that are ≥30 dB worse on the affected side than the contralateral side.
Episode duration	20 minutes to 12 hours typical; longer duration (>12 h) should prompt review of the diagnosis. Episodes shorter than 20 minutes are not consistent with Ménière's.
Pattern over time	Recurrent — typically clusters of attacks with periods of relative quiescence. Hearing loss may fluctuate early and become progressive over years.

All criteria must be met for definite disease. Probable disease covers patients early in their course or with atypical episode duration where the diagnosis is suspected but audiometric confirmation has not yet been captured. In practice, ordering audiometry as soon as the symptom pattern is recognised — even between attacks — substantially shortens diagnostic delay.

□ Capture the audiogram: The single most useful action a generalist can take is to arrange pure-tone audiometry as soon as Ménière's is suspected. A low-frequency sensorineural hearing loss documented during a fluctuation moves the diagnosis from probable to definite — and short-circuits years of diagnostic delay.

Symptom Profile

Vertigo is spontaneous, rotational, and lasts 20 minutes to several hours, peaking within an hour. Patients are often confined to bed during attacks with severe nausea and vomiting. Between attacks they are typically symptom-free or have mild residual unsteadiness. The affected ear is the same across attacks, and is identified both by the cochlear symptoms and by audiometry.

Associated Features

- Aural fullness or pressure on the affected side, often pre-empting the attack — a useful prodromal cue.
- Roaring or low-pitched tinnitus that intensifies during attacks and partially resolves between them.
- Hearing loss starts in the low frequencies, fluctuates early, then progresses to flat sensorineural loss over years.
- Migrainous features (headache, photophobia, phonophobia) co-occur in up to one-third — overlapping syndrome with vestibular migraine is now recognised.
- Tumarkin drop attacks: sudden falls without loss of consciousness due to otolith dysfunction. Late, ominous feature requiring urgent referral.

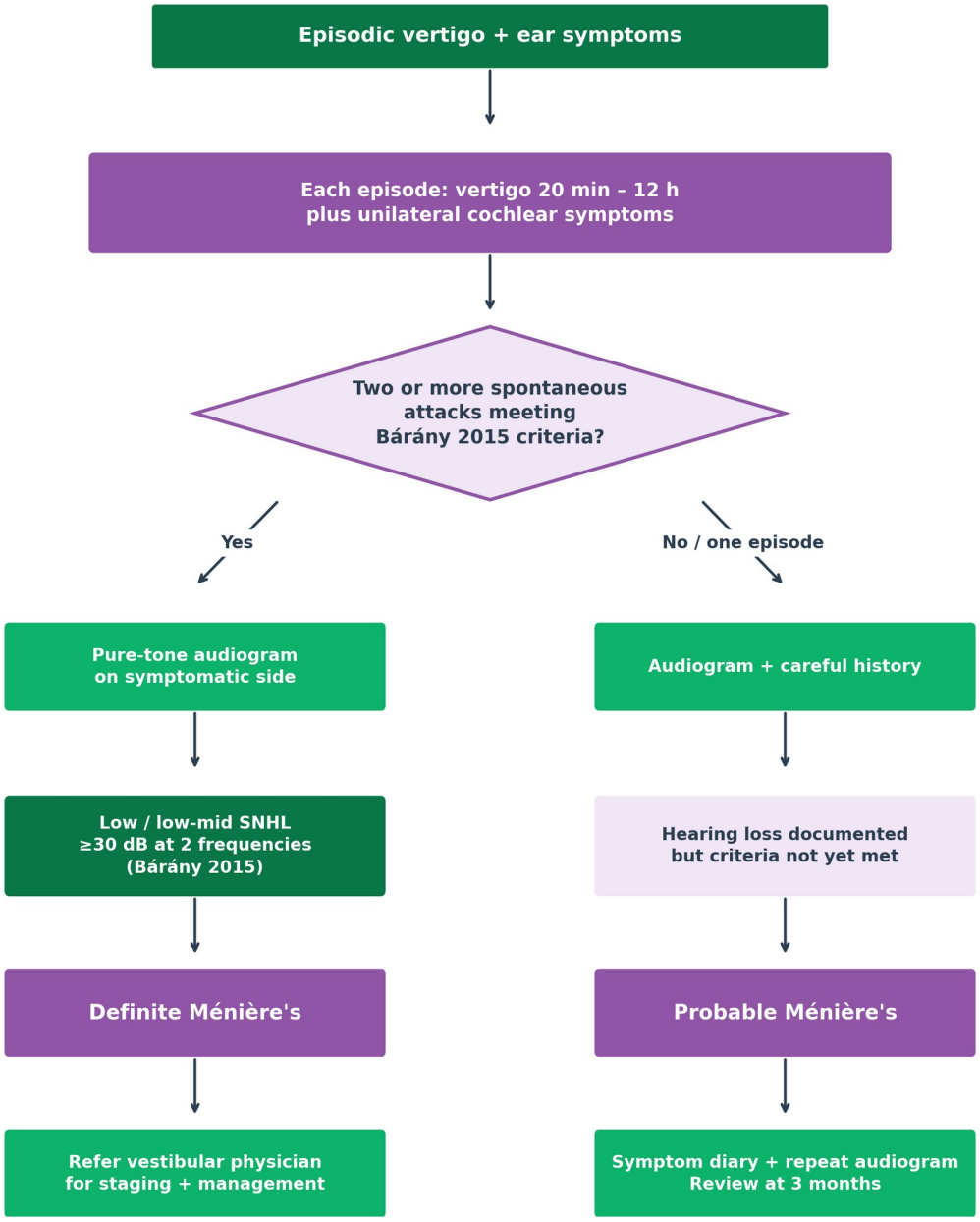

Figure 1. Diagnostic pathway for episodic vertigo with cochlear symptoms — definite vs probable Ménière's disease (Bárány 2015 framework).

Source: Australian Dizziness Clinics — clinical flowchart.

IV. Differential Diagnosis

Ménière's is one of several causes of episodic vertigo. The diagnostic differentiation rests on episode duration, presence of cochlear symptoms, audiometric pattern, and triggers. Vestibular migraine is the most commonly confused diagnosis — and the two coexist in up to a third of patients.

Table 2. Differential diagnosis of episodic vertigo encountered in primary care.

Diagnosis	Episode duration	Cochlear symptoms	Distinguishing features
Ménière's disease	20 min – 12 h	Yes — fluctuating low-frequency SNHL, tinnitus, aural fullness	Audiogram shows low-tone SNHL on affected side; same ear repeatedly affected
Vestibular migraine	Minutes – days	Usually no audiometric loss	Migraine history; photophobia / phonophobia during attacks; visual

Diagnosis	Episode duration	Cochlear symptoms	Distinguishing features
			triggers
BPPV	Seconds (<60 s)	No	Provoked by head position changes; positive Dix-Hallpike or supine roll test
Vestibular paroxysmia	Seconds – minutes	Occasional	Frequent brief attacks; carbamazepine-responsive; MRI may show neurovascular contact
TIA / posterior stroke	Minutes – hours	Possible (AICA territory)	Vascular risk factors; focal neurology; central HINTS pattern; do not miss

□ Three-question triage: (1) How long do the attacks last? (Seconds = BPPV; 20 min – 12 h = Ménière's; minutes-to-days = vestibular migraine.) (2) Is hearing affected? (Yes, fluctuating, same ear = Ménière's.) (3) Are there migraine features? (Yes = vestibular migraine, possibly co-existing.) Three minutes of focused history will sort the majority.

Where the differential cannot be resolved on history alone, the audiogram is the next-best discriminator. A normal audiogram during or shortly after an attack effectively rules out Ménière's; a unilateral low-frequency sensorineural pattern is highly suggestive.

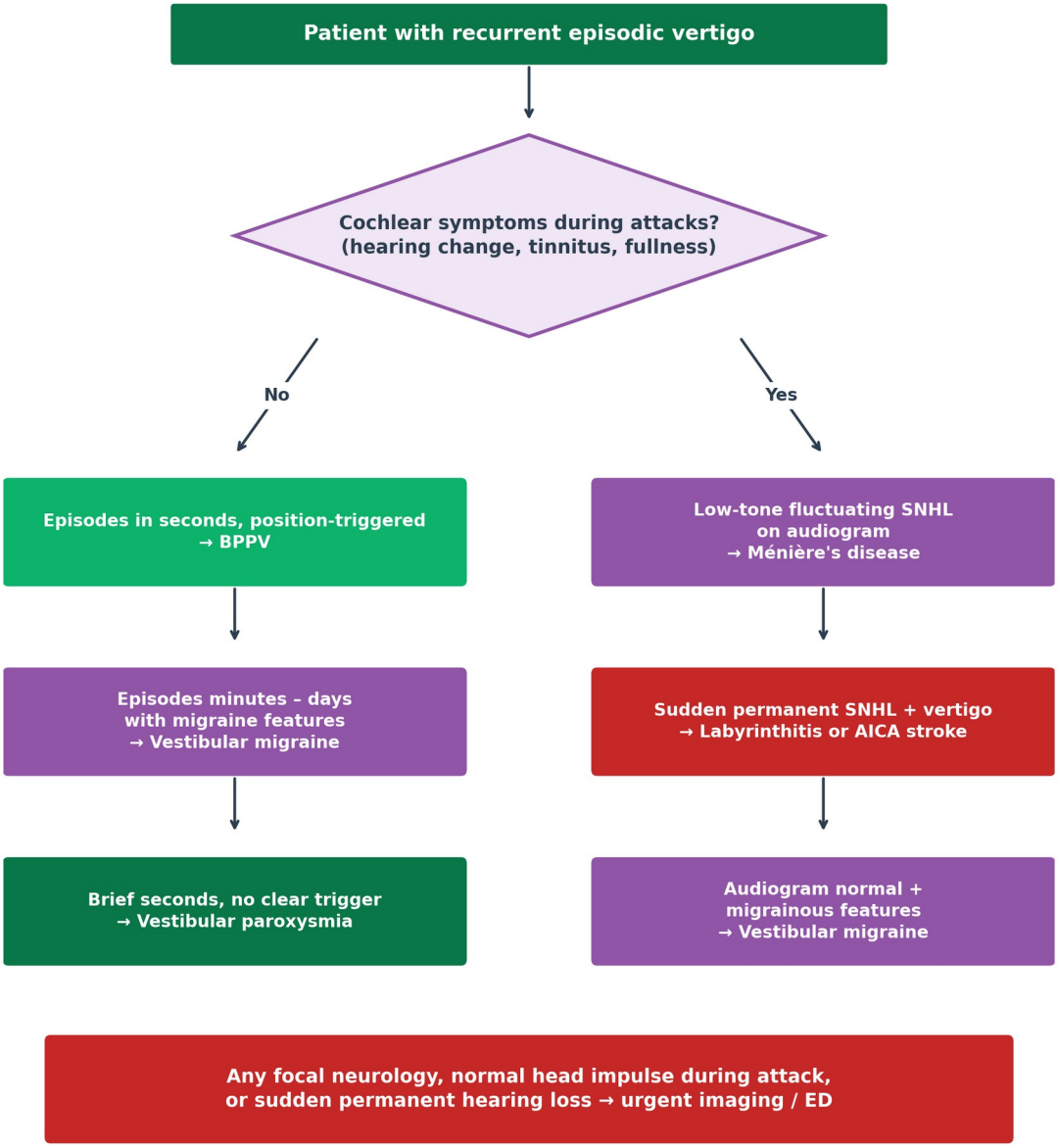

Figure 2. Triage of recurrent episodic vertigo — first gate is presence of cochlear symptoms during attacks.
Source: Australian Dizziness Clinics — clinical algorithm.

V. Investigations

Investigations in suspected Ménière's disease serve four purposes: (1) confirm the diagnosis through documented hearing loss, (2) exclude alternative or coexisting pathology, (3) characterise vestibular function for management planning, and (4) establish baseline status for monitoring.

Audiometry

Pure-tone audiometry is the single most important investigation. The classic finding is a unilateral low- to mid-frequency (250 Hz – 1 kHz) sensorineural hearing loss with normal high-frequency thresholds early in disease. Over years the pattern flattens, then descends, eventually producing a flat or descending profile of moderate-to-severe loss. Repeated audiograms during fluctuations are highly informative and should be arranged as soon as suspicion is raised.

Vestibular Function Testing

Caloric testing classically shows reduced response on the affected side, present in 50–80 percent of patients with established disease [10]. Video head impulse testing (vHIT) is normal in the majority of

Ménière's patients — the dissociation between abnormal calorics and normal vHIT is a recognised hallmark. Cervical and ocular VEMPs are often abnormal, reflecting saccular involvement. These tests are not required for diagnosis but support disease characterisation in tertiary care.

Imaging

Routine MRI is recommended in any patient with unilateral SNHL to exclude vestibular schwannoma and other retrocochlear pathology. Gadolinium-enhanced inner-ear MRI to visualise hydrops directly is currently a research and selected-case investigation, not a routine step. Imaging is not required to make the diagnosis where Bárány criteria are clearly met.

Bloods and Other Tests

Targeted bloods may be appropriate when the clinical picture suggests a secondary cause: thyroid function, autoimmune screen (ANA, ENA, ESR/CRP), serology for treponemal disease, and FBE/U&E if diuretic therapy is anticipated. Routine inflammatory and metabolic screening in classical cases adds little.

□ Sudden permanent hearing loss is not Ménière's: Acute, severe, non-fluctuating sensorineural hearing loss developing over hours-to-days is sudden sensorineural hearing loss until proven otherwise. This is an ENT emergency requiring oral high-dose steroids within 72 hours and same-day audiology — do not pigeonhole into Ménière's.

VI. Conservative and Lifestyle Management

First-line management is non-pharmacological and applies to every patient regardless of severity. Although the evidence base is observational, the burden of intervention is low, side effects are negligible, and many patients achieve substantial reduction in attack frequency on lifestyle measures alone [11].

Patient education is central. Explaining the disease as recurrent inner-ear pressure-related disturbance — rather than something dangerous — significantly reduces avoidance behaviour and anxiety, both of which independently worsen outcomes.

- Low-salt diet: target less than 2 g sodium per day. Patients should be taught to read labels and to avoid processed foods. This is the single most-supported lifestyle intervention.
- Reduce caffeine, alcohol, and nicotine — common precipitants. A trial of complete elimination for 6–8 weeks is a reasonable first step.
- Sleep hygiene and stress management — fatigue and stress are reproducible triggers; consider written prevention plans and CBT referral when persistent.
- Symptom and trigger diary: track attack frequency, duration, severity, and associated factors. Useful for both monitoring response to therapy and for vestibular physician review.

□ Set realistic expectations: Most patients achieve a 50–70 percent reduction in attack frequency on diligent lifestyle measures, but few become attack-free on diet alone. Frame the goal as control rather than cure — and explain that medication is added when lifestyle alone is insufficient, not as failure.

VII. Pharmacological Management — Stepwise Approach

Pharmacotherapy is added when lifestyle measures fail to control attacks adequately, typically defined as more than one disabling attack per month or interference with work or driving. Therapy aims to reduce attack frequency and severity; it does not cure the underlying hydrops. Australia-wide prescribing patterns favour betahistine and thiazides; international practice is similar [12].

First-Line Agents

- Betahistine 16 mg three times daily is the most widely used first-line agent. Higher doses (up to 48 mg tds) may be effective in resistant cases. Evidence is mixed but a meta-analysis of randomised trials shows modest benefit in attack frequency [13]. Side effects are minor.
- Hydrochlorothiazide 25 mg daily, often combined with amiloride or triamterene to mitigate potassium loss, is a long-established option. Mechanism is reduction of endolymph volume.

- Combination therapy (betahistine + diuretic) is reasonable for patients with persisting attacks on monotherapy, although evidence for additive benefit is limited.
- Trial duration: each agent or combination should be tried for at least 8–12 weeks before declaring failure. Lifestyle measures must be optimised in parallel.

Acute Attack Management

During an attack, prochlorperazine 12.5 mg IM or buccal 3 mg, or oral promethazine 25 mg, controls vertigo and nausea. Ondansetron is an alternative for nausea. These agents must be used short-course only — never beyond 48–72 hours — as prolonged use delays central compensation and predisposes to PPPD. This is identical to the suppressant-trap principle in vestibular neuritis.

□ Stop suppressants early: Vestibular suppressants are for the acute attack only. Routine prescription of prochlorperazine, promethazine, or betahistine for 'maintenance' anti-vertigo cover beyond 72 hours is the single commonest avoidable error in primary-care Ménière's management — it directly worsens long-term outcomes.

Figure 3. Stepwise management of Ménière's disease — lifestyle and medical therapy first; intratympanic and surgical options for refractory disease.

Source: Australian Dizziness Clinics — clinical flowchart.

VIII. Procedural and Surgical Options

When optimised lifestyle and pharmacological therapy fail to control attacks (typically defined as ongoing disabling vertigo despite three months of adequate therapy), procedural options are considered. These are vestibular physician or otolaryngology decisions; the generalist's role is timely referral.

Intratympanic Steroids

Intratympanic dexamethasone is increasingly the first procedural step. It is hearing-sparing, can be repeated, and has a favourable safety profile. Approximately 60–70 percent of patients achieve substantial vertigo reduction [14]. Multiple courses may be required.

Intratympanic Gentamicin

Intratympanic gentamicin chemically ablates vestibular hair cells and provides robust vertigo control in around 80 percent of patients. The trade-off is a 20–30 percent risk of further hearing loss, requiring careful patient selection and counselling. It is generally reserved for refractory unilateral disease where useful hearing on the contralateral side is preserved [15].

Surgical Options

Endolymphatic sac decompression is hearing-preserving but the evidence for benefit beyond placebo is contested. Vestibular nerve section (transmastoid or middle fossa) and labyrinthectomy are reserved for refractory disease with non-serviceable hearing on the affected side. These are tertiary-centre procedures with significant operative considerations.

□ Early referral matters: Refer all suspected Ménière's to a vestibular physician or otolaryngology service for diagnostic confirmation and longitudinal staging. Timely referral preserves access to hearing-sparing intratympanic options before disease progression mandates more destructive procedures.

IX. Red Flags and Referral Indications

Red Flags Requiring Urgent Same-Day Referral

- Sudden permanent (non-fluctuating) sensorineural hearing loss — ENT emergency requiring high-dose oral steroids within 72 hours.
- Acute focal neurology accompanying vertigo — diplopia, dysarthria, limb weakness, sensory loss, ataxia disproportionate to vertigo.
- New severe headache with vertigo — particularly occipital — suggests posterior circulation pathology.
- Tumarkin drop attacks (sudden falls without loss of consciousness) — late and disabling feature of advanced disease.
- Bilateral simultaneous symptoms or rapid progression to bilateral disease — consider autoimmune, syphilitic, or systemic cause.
- Acute SNHL with vertigo — consider AICA territory stroke, particularly with vascular risk factors.
- Severe psychological distress, suicidality, or inability to function — mental health input alongside vestibular review.

Indications for Routine Vestibular Physician Referral

- All suspected Ménière's disease — for diagnostic confirmation, audiometric staging, and longitudinal management.
- Diagnostic uncertainty between Ménière's, vestibular migraine, and overlap syndromes.
- Inadequate control on lifestyle and first-line pharmacotherapy after 3 months — for consideration of intratympanic options.
- Progressive hearing loss requiring audiology and rehabilitation input.
- Patient request for prognosis discussion, second opinion, or treatment planning.
- Specific occupational or recreational concerns — pilots, drivers of heavy vehicles, divers, professional athletes.

□ Two messages at every review: (1) Stop vestibular suppressants between attacks — they delay compensation and worsen long-term outcome. (2) Start lifestyle measures and obtain an audiogram now — these are the two interventions that materially shorten diagnostic delay and start meaningful management.

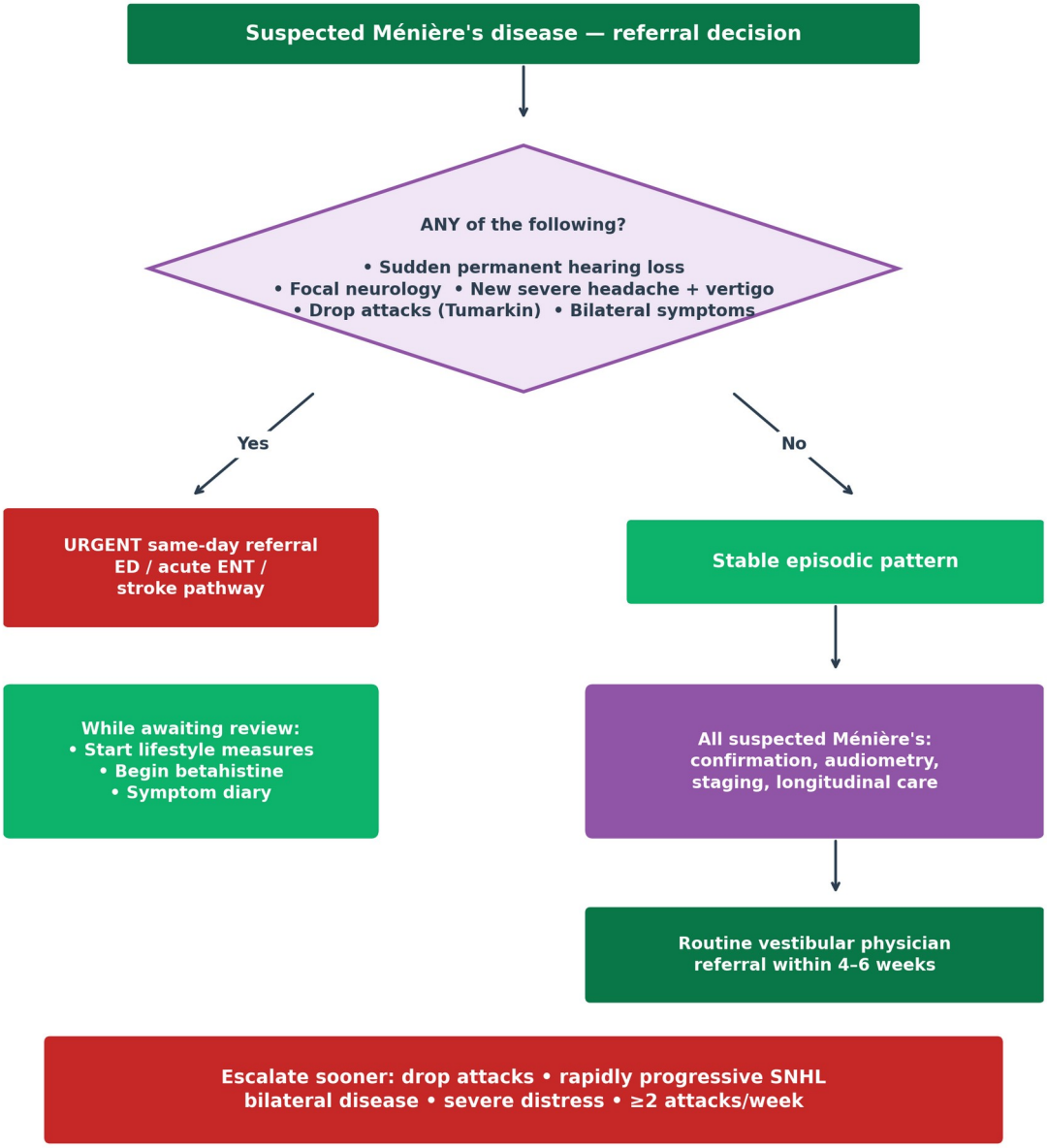

Figure 4. Referral decision pathway — urgent versus routine pathways and primary-care holding measures while awaiting vestibular physician review.
Source: Australian Dizziness Clinics — clinical algorithm.

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