Ménière's Disease:

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

Vestibular Medicine for Vestibular Physicians

Peripheral Vestibular Pathology — Module 2.5

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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, History and Epidemiology

Ménière's disease (MD) is a chronic, episodic inner-ear disorder defined clinically by spontaneous attacks of vertigo, fluctuating sensorineural hearing loss, tinnitus, and aural fullness in the affected ear [1,2,13]. It was first delineated as an inner-ear disorder in 1861 by the French physician Prosper Ménière, who reported on a young patient who died after a haemorrhage into the labyrinth, at a time when episodic vertigo was widely attributed to apoplexy or cerebral congestion [1,3]. Ménière's central insight, that vertigo could arise from labyrinthine pathology, reshaped the discipline and gave the disease its eponymous label [1].

The condition was further elaborated by Hallpike and Cairns in 1938, who linked the clinical syndrome to the pathological finding of endolymphatic hydrops on temporal-bone histology [5]. Over the subsequent decades, inconsistent use of terms such as 'Ménière's disease', 'Ménière's syndrome' and 'cochlear/vestibular Ménière's' fragmented diagnosis and research [3]. The American Academy of Otolaryngology - Head and Neck Surgery (AAO-HNS) introduced standardised criteria in 1972 and 1995, but these were superseded by the 2015 Bárány Society / international consensus criteria, now embedded in the 2020 AAO-HNS Clinical Practice Guideline [13,14].

Ménière's disease is uncommon yet disproportionately disabling. Reported prevalence varies widely with case definition and population sampled, ranging from approximately 3.5 to 513 per 100 000 [2,4]. The most rigorous community-based estimates cluster between 17 and 200 per 100 000 in Western populations, with annual incidence in the order of 10-15 per 100 000 [2,4,9,28,29,35,36,37]. In specialty dizziness clinics, MD accounts for 5-10% of new vertigo referrals [9,11].

Table 1. Epidemiology of Ménière's disease at a glance.

Measure	Adult value	Notes
Prevalence (reported)	3.5 - 513 / 100 000	Wide range; depends on criteria [2,4]
Best-estimate prevalence	17 - 200 / 100 000	Community-based, Western populations [2,4]
Annual incidence	10 - 15 / 100 000	Higher with definite criteria [2,9]
Peak age of onset	40 - 60 years	Rare under 18 and over 75 [2,7]
F:M ratio	approx 1.3 : 1	Slight female predominance [5,8]

Onset typically occurs between the fourth and sixth decades, with a peak in the fifth decade [2]. Paediatric MD exists but is rare, accounting for under 3% of all cases [7]. There is a slight female preponderance in most cohorts [5,8], and the disease is more frequently reported in white populations, though this likely reflects diagnostic access rather than true biological difference [8,38]. A well-described association with migraine, present in 40-60% of patients with definite Ménière's versus 12-15% of the general population, argues for shared pathophysiology in a substantial subgroup [4,21,22].

Bilateral disease is one of the most clinically important epidemiological features. At presentation, fewer than 10% have bilateral symptoms, but the cumulative bilateral conversion rate rises to 25-45% at 20 years of follow-up [2,8,10]. Bilateral disease carries far greater functional, audiological, and treatment implications, and shifts management strategy away from ablative options. Familial clustering is reported in 5-15% of cases, and rare monogenic forms have been mapped to genes such as COCH, FAM136A, DTNA, PRKCB and DPT [6].

□ **Clinical Insight:** MD is a chronic, lifelong condition with a strong tendency toward fluctuation, partial remission, and eventual stabilisation. Clinicians must counsel patients early about the realistic trajectory: most vertigo attacks 'burn out' within 5-10 years, but hearing loss is typically progressive and is the feature most likely to define long-term disability [10].

II. Pathophysiology - Endolymphatic Hydrops and Multifactorial Aetiology

The defining histopathological feature of Ménière's disease is endolymphatic hydrops (EH), abnormal expansion of the endolymphatic compartment of the membranous labyrinth [5,11]. Endolymph is the

potassium-rich fluid bathing the apical surfaces of cochlear and vestibular hair cells; its volume and ionic composition are tightly regulated by the stria vascularis, dark cells of the vestibular labyrinth, and the endolymphatic sac [5,11]. In MD, this homeostasis fails, the cochlear duct and saccule distend, and Reissner's membrane bulges into the scala vestibuli [5].

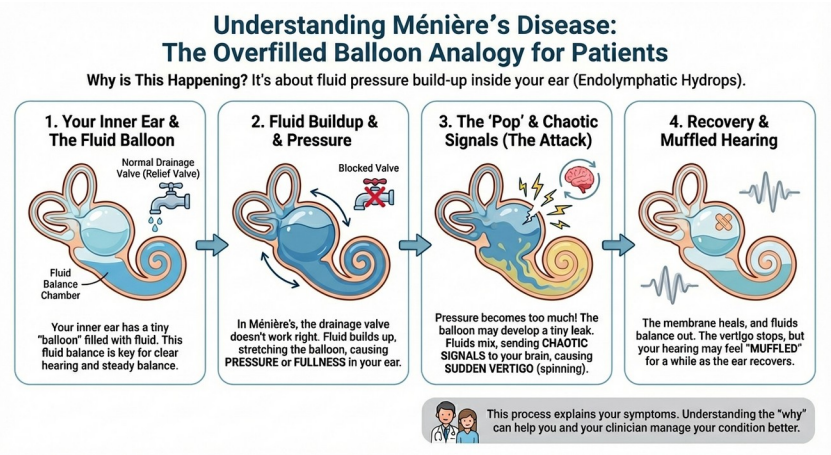

Figure 1. Schematic representation of endolymphatic hydrops - the balloon analogy.
Source: Australian Dizziness Clinics educational schematic.

Hallpike and Cairns first demonstrated EH in temporal bones from patients with Ménière's disease in 1938, and Schuknecht's subsequent work established hydrops as the canonical pathological substrate [5]. However, hydrops is not synonymous with the clinical disease. Asymptomatic hydrops has been found in temporal bones of patients without any Ménière history, while some patients with classical Ménière symptoms show only mild hydrops at autopsy [5,11]. This dissociation has driven the modern view: endolymphatic hydrops is necessary but not sufficient for MD; additional triggers and individual susceptibility factors determine clinical expression [33].

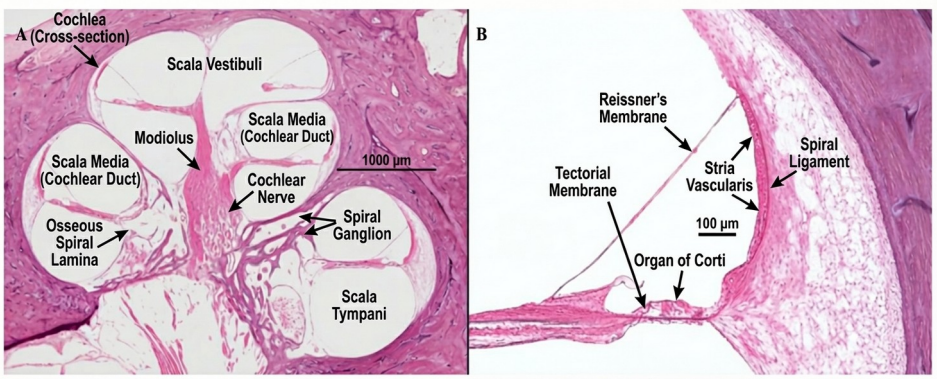

Figure 2. Histological cross-section of the cochlea in Ménière's disease showing enlarged scala media.
Source: temporal-bone histopathology, reproduced for educational use.

Two complementary mechanisms have been proposed to translate hydrops into the clinical episode. Schuknecht's classical 'membrane rupture' hypothesis posits that progressive dilatation eventually ruptures the membranous labyrinth, most often Reissner's membrane or the saccular wall, releasing potassium-rich endolymph into the perilymphatic space [5]. The resulting potassium intoxication of vestibular and cochlear nerve fibres produces the abrupt vertigo, hearing change, and tinnitus, and membrane healing terminates the attack within hours [5,11]. Histological evidence of fibrous scarring from healed ruptures, particularly in the saccule, supports this mechanism [5].

A second, complementary model, the 'drainage' or 'pressure-relief failure' theory, emphasises dysfunction of the endolymphatic sac and duct as the upstream lesion [5,11]. Animal models in which the endolymphatic sac is surgically obliterated reliably produce hydrops, and human temporal bones from MD patients show sac hypoplasia and peri-sac fibrosis disproportionately often [5,11]. The endolymphatic sac acts as a pressure-relief organ; chronic failure of resorption raises endolymph volume slowly, while acute resorption failure may precipitate attacks [33].

Beyond hydrops itself, multiple aetiological factors converge on the endolymphatic sac or stria vascularis. Anatomical predisposition includes congenital sac hypoplasia, a short or narrow vestibular aqueduct, and altered angulation of the petrous bone [5,11]. Genetic factors are increasingly recognised: familial Ménière's accounts for 5-15% of cases, and pathogenic variants in COCH, FAM136A, DTNA and PRKCB cause autosomal-dominant disease [6]. Autoimmune mechanisms are supported by elevated rates of systemic autoimmunity, circulating anti-inner-ear antibodies, and HLA-DRB1 associations in subgroups [6].

Viral aetiology, most often herpes simplex, remains plausible but unproven, with viral DNA detected in endolymphatic sac tissue in some series [11]. Vascular and migraine-related mechanisms have gained traction given the strong epidemiological overlap: a vestibular-migraine-like vasospastic insult on a vulnerable inner ear may unmask hydrops, and this may explain the indistinguishable clinical phenotype seen in many migraineurs with Ménière features [21,22]. Trauma, allergy, hormonal flux, and hypothyroidism are each weakly but reproducibly associated [21,27].

□ **Key Point: Endolymphatic hydrops is the histopathological hallmark of Ménière's disease but not its sole cause. Clinical MD reflects an interaction between an unstable inner-ear fluid system and a constellation of genetic, immunological, vascular, viral, and migrainous co-factors. This explains why MD is a heterogeneous, lifelong syndrome rather than a single biochemical lesion.**

III. Clinical Features and the Classical Tetrad

Ménière's disease classically presents with a tetrad of episodic spontaneous vertigo, fluctuating sensorineural hearing loss, tinnitus, and aural fullness, all referable to one ear in the early years [2,13]. The episodic nature of attacks is the single most important diagnostic anchor; a continuous, non-fluctuating vestibular complaint should always raise alternative diagnoses [13,21].

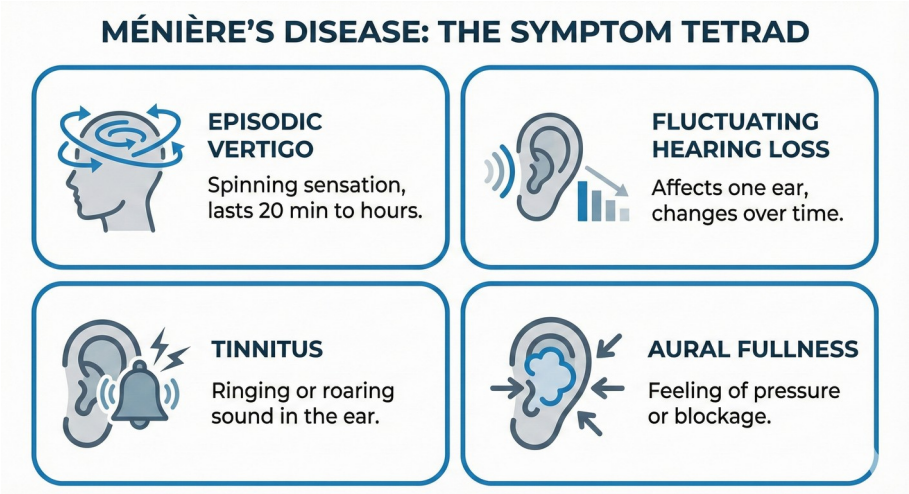

Figure 3. The classical Ménière's tetrad - vertigo, fluctuating hearing loss, tinnitus, and aural fullness.
Source: Australian Dizziness Clinics educational schematic.

Vertigo is the dominant symptom in most patients. Attacks are spontaneous, intense rotational vertigo lasting 20 minutes to 12 hours, characteristically accompanied by autonomic features such as nausea, vomiting, sweating, pallor, and prostration [13]. The Bárány criteria explicitly bracket vertigo duration: less than 20 minutes argues against MD and toward vestibular migraine, transient ischaemic attack, or BPPV; greater than 24 hours suggests an alternative diagnosis such as vestibular neuritis or a central event [13,21]. After the acute attack, patients commonly experience days of motion sensitivity and unsteadiness that gradually resolve [10].

Fluctuating sensorineural hearing loss is the second pillar. Early in the disease, hearing loss predominantly affects the low frequencies, producing the characteristic 'peak' or upsloping audiogram and a sensation of muffled, bass-poor hearing [2,12]. Crucially, hearing improves between attacks in the first years, a feature that distinguishes MD from progressive sensorineural disorders [24]. With time, a flat

or peaked audiometric loss develops, speech discrimination deteriorates disproportionately, and a sense of distortion or recruitment dominates [2,8,24].

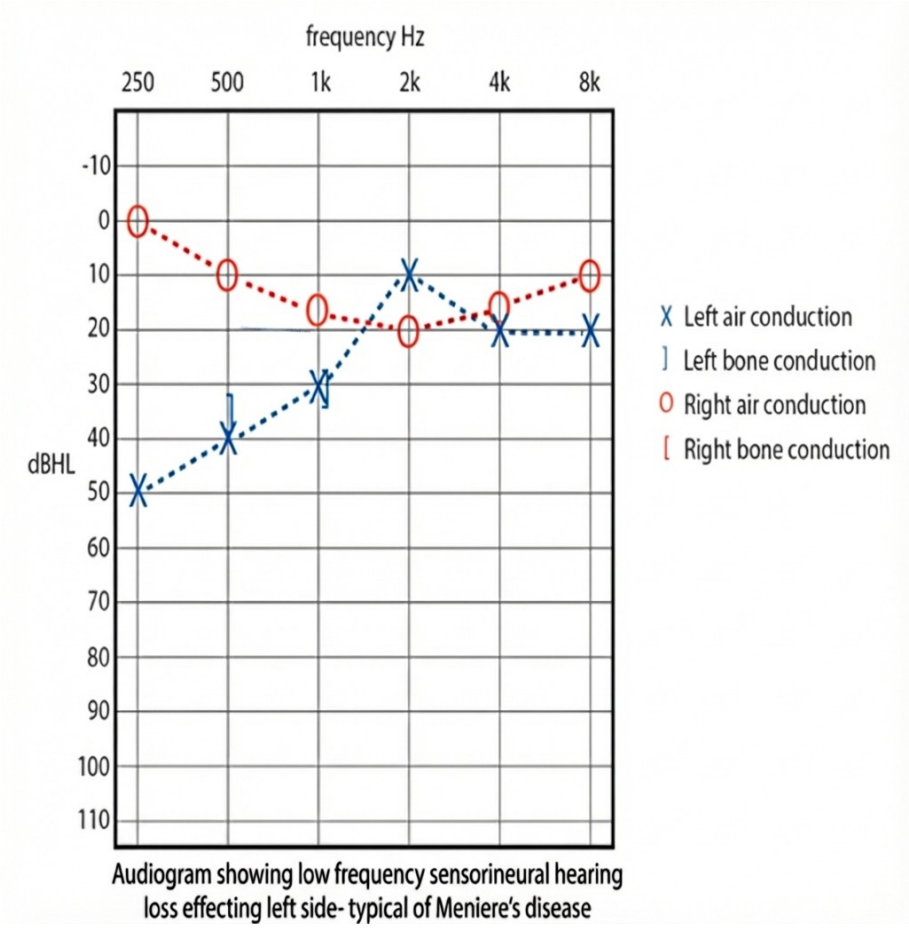

Figure 4. Initial pure-tone audiogram in left-sided Ménière's disease showing low-frequency sensorineural hearing loss.

Source: Australian Dizziness Clinics patient audiogram, anonymised.

Tinnitus is typically low-pitched, roaring or ocean-like, and intensifies before or during attacks [2]. Aural fullness, a pressure or blockage sensation in the affected ear, is the most under-reported of the four cardinal symptoms but is highly characteristic when sought, and is now retained as a diagnostic criterion in the 2015 Bárány definition [13]. Patients frequently describe a 'crescendo' of tinnitus and fullness over minutes to hours preceding an attack, providing a window for early abortive treatment in some [30].

Several additional clinical features warrant familiarity. Tumarkin's otolithic crises ('drop attacks') are sudden, brief falls without warning or loss of consciousness, attributed to instantaneous otolithic deflection from a hydropic saccule or utricle [12,23]. They occur in 5-10% of MD patients, may cluster late in the disease, and are highly disabling [23]. Lermoyez's syndrome, vertigo with paradoxical improvement of hearing, is a rare but classical variant [27]. Recovery is typically slow over days, and many patients describe a baseline of low-grade motion sensitivity between attacks even after vertigo control [10].

On examination during an acute attack, otoscopic findings are normal; nystagmus is horizontal-torsional and may switch direction across the attack (initial 'irritative' beats toward the affected ear, then 'paretic' beats away as the canal function falls) [11,20]. Between attacks the examination is often unremarkable, although headshake or vibration-induced nystagmus is common in the chronically affected ear, and the head impulse test may demonstrate covert saccades indicative of cumulative vestibular hypofunction [20,42].

□ Clinical Pearl: The single most useful historical question in suspected Ménière's is: 'Do attacks last between 20 minutes and a few hours, with hearing change in the same ear?' A 'yes' is highly specific. Brief (seconds) episodes argue for BPPV; sub-20-minute or migraine-coloured attacks argue for vestibular migraine; protracted single events argue for vestibular

neuritis or stroke.

IV. Diagnostic Criteria and the Bárány Society Position

Diagnosis of Ménière's disease is clinical. The current standard, the 2015 Bárány Society / Japan Society for Equilibrium Research / European Academy of Otology and Neurotology / Equilibrium Committee of the AAO-HNS criteria, replaced the 1995 AAO-HNS framework and is now embedded in the 2020 AAO-HNS Clinical Practice Guideline [13,14,48]. The 2015 criteria define two categories: definite and probable Ménière's, eliminating the older 'possible' and 'certain' tiers [13].

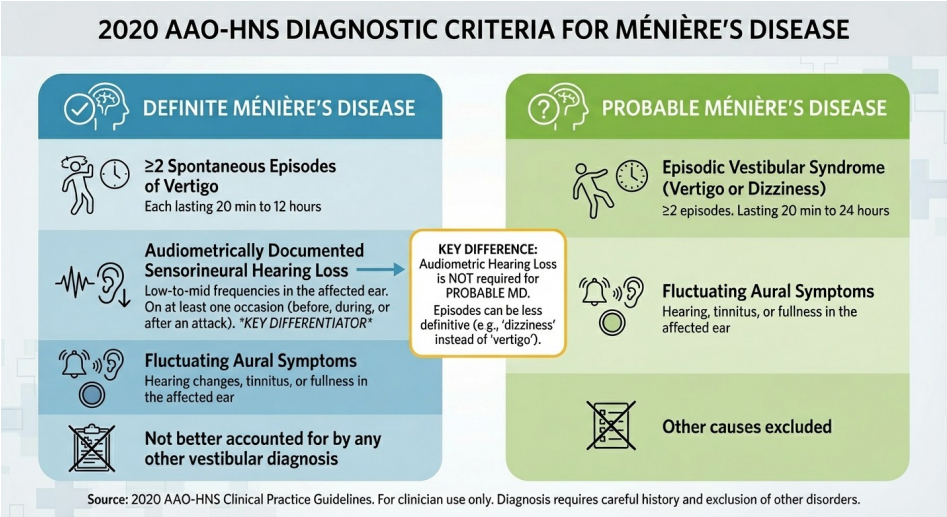

Figure 5. Bárány Society / 2020 AAO-HNS diagnostic criteria for Ménière's disease.
Source: Australian Dizziness Clinics infographic adapted from López-Escámez et al. 2015 [13].

Definite Ménière's disease requires: at least two spontaneous episodes of vertigo lasting 20 minutes to 12 hours; audiometrically documented low- to mid-frequency sensorineural hearing loss in one ear before, during or after one of the episodes; fluctuating aural symptoms (hearing, tinnitus, or fullness) in the affected ear; and exclusion of better-fitting alternative diagnoses [13]. Probable Ménière's requires the same temporal vertigo pattern with fluctuating aural symptoms, but without an objective audiometric requirement [13]. Both categories explicitly require active exclusion of competing diagnoses [13,21].

Table 2. Comparison of the 2015 Bárány / 2020 AAO-HNS criteria with the 1995 AAO-HNS framework.

Category	2015 Bárány / 2020 AAO-HNS	1995 AAO-HNS
Definite MD	Two or more vertigo episodes (20 min to 12 h); audiometric low/mid-frequency SNHL in affected ear; fluctuating aural symptoms; alternatives excluded.	Two or more definitive vertigo episodes (20 min or longer); audiometrically confirmed hearing loss on at least one occasion; tinnitus or aural fullness; histological confirmation not required.
Probable MD	Two or more vertigo episodes (20 min to 24 h); fluctuating aural symptoms in affected ear; alternatives excluded. No audiometric requirement.	One definitive vertigo episode (20 min to 24 h); audiometrically confirmed hearing loss on at least one occasion; tinnitus or aural fullness.
Possible / Certain	Removed in 2015. Tiers consolidated to Definite and Probable.	'Possible': vertigo without confirmed hearing loss, or hearing loss without confirmed vertigo. 'Certain': definite MD plus histopathological confirmation.

The 2015 simplification eliminated the 'possible' tier, which had encompassed a heterogeneous group of patients with vertigo without hearing loss or vice versa; many of these are now recognised as vestibular migraine, autoimmune inner-ear disease, or early MD pre-conversion [13,21]. The 'certain' tier (requiring histopathology) was redundant in clinical practice and has also been removed [13]. Critically, the duration window for definite MD was narrowed to 20 minutes to 12 hours (from up to 24 hours in 1995) to improve specificity against vestibular migraine and protracted single events [13,48].

Several diagnostic caveats are now widely accepted. First, the vertigo duration cap is most useful in differentiating from vestibular migraine (often shorter and migraine-coloured) and vestibular neuritis (typically protracted single events) [13,21]. Second, the requirement for low- to mid-frequency audiometric loss in definite MD is the single most discriminating objective feature; a high-frequency or symmetric flat loss in a patient with episodic vertigo should prompt consideration of presbycusis-plus-vestibular-migraine, vestibular schwannoma, or sudden sensorineural hearing loss with vertigo [13,21,24]. Third, exclusion of vestibular schwannoma, superior canal dehiscence, and central vestibular disorders by appropriate imaging and examination is required before a Ménière diagnosis is locked in [21,32].

Subtype classification has also evolved. López-Escámez and colleagues proposed five clinical subgroups based on cluster analysis: classical sporadic, familial, migraine-associated, autoimmune-associated, and delayed-hydrops phenotypes [6,21]. This subtype framework is not diagnostic but is gaining traction for stratifying prognosis and selecting candidates for research-grade interventions, particularly immune-modulating treatments [6].

□ **Important: Ménière's disease remains a clinical diagnosis of exclusion. An MRI of the internal auditory meatus is required at first presentation to exclude vestibular schwannoma, endolymphatic-sac tumour, and intracranial pathology - these conditions can mimic the full Ménière phenotype and must not be missed [21,32].**

V. Investigations: Audiometry, Vestibular Tests, and MRI Hydrops Imaging

No single test confirms Ménière's disease; investigations serve to objectively document inner-ear involvement, characterise hearing and vestibular function, and exclude mimics [14,21]. Pure-tone audiometry is mandatory in every suspected case and is the only investigation explicitly required by the 2015 criteria for definite MD [13,14].

Table 3. Key investigations in suspected Ménière's disease.

Investigation	Purpose	Indication / yield
Pure-tone audiometry (PTA)	Document the characteristic low/mid-frequency SNHL; track fluctuation and progression.	Mandatory at presentation and serially; required for definite MD diagnosis [13,14].
Speech audiometry	Detect disproportionate speech-discrimination loss suggestive of cochlear hydrops.	Routine; aids assessment of functional disability [12,24].
Tympanometry	Confirm middle-ear function is normal; exclude conductive overlay.	Routine first-visit screening [14].
Caloric / video head impulse (vHIT)	Quantify ipsilesional canal paresis; serially track vestibular function.	Recommended in atypical, bilateral, or refractory cases [20,42].
Cervical and ocular VEMPs (cVEMP/oVEMP)	Assess saccular and utricular function; characteristic asymmetries support MD.	Useful adjunct, particularly in early or atypical disease [20,42].
Electrocochleography (ECochG)	SP/AP ratio elevation supports cochlear hydrops; specificity modest.	Selected cases; sensitivity 60-70%, specificity 80-90% [11].
MRI IAM with gadolinium	Exclude vestibular schwannoma, ELST, central pathology; delayed	Mandatory at first presentation; delayed-FLAIR if available

	3D-FLAIR can visualise hydrops.	[21,33].

Pure-tone audiometry is the single highest-yield test. The classical finding is a low-frequency (125-500 Hz) sensorineural loss with relative preservation of higher frequencies, producing the 'peak' or 'tent' audiogram when mid-frequencies are slightly better than both ends [12,24]. With disease progression, the pattern flattens and may evolve into a downsloping high-frequency loss indistinguishable from presbycusis [2,12]. Crucially, hearing thresholds should fluctuate in early disease; serial audiograms are therefore more informative than any single study [24].

Caloric testing classically shows unilateral canal paresis on the affected side in 50-75% of patients with established disease, though normal calorics do not exclude MD, particularly early [20,42]. Video head impulse testing has gained ground as a portable, attack-friendly complement to caloric testing; an interesting dissociation is reported in which calorics show paresis but vHIT remains normal, a pattern relatively specific for hydrops and presumed to reflect preserved high-frequency canal function despite low-frequency caloric weakness [20,42]. Bilateral testing is essential because bilateral conversion changes management substantially [8,20].

Vestibular evoked myogenic potentials (VEMPs) provide indirect evidence of saccular (cVEMP) and utricular (oVEMP) involvement [20,42]. Reduced or absent cVEMP amplitude in the affected ear, with tuning shifts toward higher frequencies, supports saccular hydrops, while preserved VEMPs argue against advanced disease [20]. Electrocochleography (ECochG) measures the summating potential / action potential ratio; an elevated SP/AP ratio supports cochlear hydrops, but sensitivity is modest (60-70%) and the test has largely been superseded by MRI hydrops imaging where available [11].

MRI of the internal auditory meatus is mandatory at the first MD-suspect presentation, primarily to exclude vestibular schwannoma, endolymphatic-sac tumour, and intracranial pathology [21,32]. Delayed post-gadolinium 3D-FLAIR sequences (4 hours after intravenous or intratympanic contrast) now allow direct visualisation of the endolymphatic compartments and can demonstrate cochlear or vestibular hydrops with reasonable sensitivity in expert centres [11,33,34]. This is moving toward becoming a confirmatory rather than purely exclusionary investigation, particularly in atypical or unilateral cases where the differential includes vestibular migraine [11,33].

□ Clinical Insight: A normal MRI excludes structural mimics but does not confirm Ménière's. Diagnosis remains clinical, anchored on the temporal pattern of vertigo and audiometric fluctuation. Over-investigation is common and rarely changes management - order tests with a specific question in mind, particularly before considering ablative treatment.

VI. Differential Diagnosis

The differential diagnosis of Ménière's disease encompasses every cause of recurrent episodic vertigo, fluctuating sensorineural hearing loss, or both [21,32]. Misdiagnosis is common; specialist re-evaluation of community MD diagnoses re-categorises 30-50% of patients as having vestibular migraine, BPPV, or other peripheral or central disorders [21,26].

Vestibular migraine (VM) is the single most important differential and is the most frequent misdiagnosis. VM produces episodic vertigo lasting minutes to days, often with auditory features (transient tinnitus, fullness) but without sustained low-frequency hearing loss [21,22]. Migraine history (with or without headache during attacks), photophobia, phonophobia, and a normal audiogram point toward VM [22]. Critically, MD and VM can coexist in 30-40% of patients with definite MD, and the migrainous subtype may represent a distinct prognostic group [21,22].

Benign paroxysmal positional vertigo (BPPV) produces brief (under one minute) vertigo on positional change without hearing loss and is easily distinguished by Dix-Hallpike or supine roll testing [21]. Vestibular neuritis presents as a single protracted (24-72 hours) attack of vertigo without hearing loss; the protracted single-event pattern is decisive [21]. Labyrinthitis adds hearing loss to vestibular neuritis but is typically a one-off illness rather than recurrent [21,32].

Vestibular schwannoma must always be excluded. The classical presentation is progressive (not fluctuating) unilateral sensorineural hearing loss with disequilibrium, but in 15-20% of cases schwannomas can present with episodic vertigo or sudden hearing loss that mimics MD [21,32].

Asymmetric tinnitus or hearing loss without typical Ménière fluctuation is an indication for MRI regardless of the working diagnosis [32]. Endolymphatic-sac tumours are rare but can produce a Ménière phenotype, particularly in von Hippel-Lindau disease [32].

Table 4. Differential diagnosis of Ménière's disease - key distinguishing features.

Differential	Distinguishing features
Vestibular migraine	Migraine history; photophobia / phonophobia during attacks; transient or absent objective hearing loss; attacks often shorter or longer than MD's 20 min - 12 h window [21,22].
Benign paroxysmal positional vertigo	Brief (under one minute) positional vertigo without hearing loss; characteristic Dix-Hallpike or supine roll nystagmus [21].
Vestibular neuritis	Single protracted (24-72 h) vertigo episode without hearing loss; unilateral canal paresis on caloric / vHIT testing [21].
Vestibular schwannoma	Progressive (not fluctuating) unilateral SNHL with or without episodic vertigo; confirmed on gadolinium MRI [21,32].
Autoimmune inner-ear disease	Rapidly progressive bilateral SNHL with or without systemic autoimmunity; steroid-responsive [4,6].
Superior canal dehiscence	Sound- or pressure-induced vertigo; conductive hyperacusis; characteristic CT temporal bone [32].
Vertebrobasilar TIA	Brief episodes with brainstem features (diplopia, dysarthria, hemiparesis); vascular risk factors; MRI / CT angiography [21].

Autoimmune inner-ear disease (AIED) produces rapidly progressive bilateral SNHL over weeks to months and may include vertigo; its corticosteroid responsiveness is the key clinical clue [4,6]. Cogan's syndrome, autoimmune audiovestibular disease with interstitial keratitis, is a rare but important differential, particularly in younger patients [4]. Otosyphilis, though uncommon, can produce a near-perfect Ménière phenocopy and is a treatable cause that must not be missed; serological screening is appropriate in atypical, bilateral, or younger cases [4,21,26].

Third-window syndromes, superior canal dehiscence, enlarged vestibular aqueduct, perilymph fistula, produce sound- or pressure-induced vertigo (Tullio phenomenon, Hennebert's sign) and conductive hyperacusis; CT of the temporal bone is diagnostic [32]. Posterior circulation transient ischaemic attacks can mimic vertiginous episodes, particularly in older patients with vascular risk factors, and require urgent vascular imaging when suspected [21]. Finally, persistent postural-perceptual dizziness (PPPD) is increasingly recognised as a secondary functional consequence of MD attacks and should be considered when chronic non-vertiginous dizziness dominates the inter-attack period [21,22].

□ Clinical Pearl: Whenever the 'Ménière' diagnosis does not fully fit - younger patient, bilateral hearing loss at onset, predominant migrainous features, normal audiogram between attacks, or progressive hearing decline without episodes - re-interrogate the differential. Most 'refractory Ménière's' is mis-labelled vestibular migraine, autoimmune disease, or a third-window syndrome.

VII. Medical Management - Lifestyle, Pharmacology, and Intratympanic Therapy

Management of Ménière's disease is stepwise and tailored. The therapeutic goals are: control of vertigo attacks, preservation of hearing and balance function, and improvement of quality of life [14,18]. There is no curative therapy; treatment is staged from conservative measures through pharmacological prophylaxis, intratympanic therapy, and finally surgery in refractory cases [14,16,17]. Approximately 80% of patients achieve adequate vertigo control with conservative and medical measures alone [16,39].

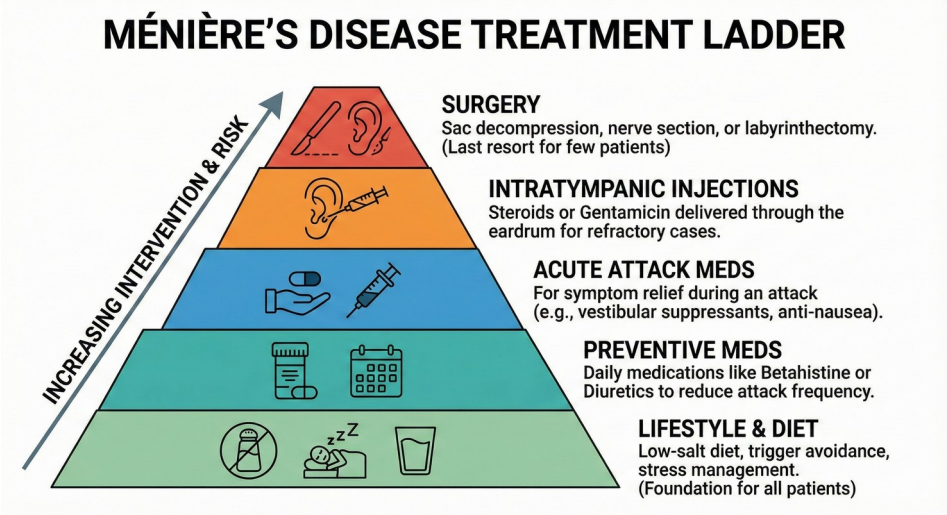

Figure 6. Treatment pyramid for Ménière's disease - stepwise escalation from lifestyle to surgical options.
Source: Australian Dizziness Clinics educational schematic.

Lifestyle and dietary measures

A sodium-restricted diet (aim under 1500-2000 mg/day) remains a long-standing first-line measure, based on the rationale that reducing systemic sodium load may stabilise endolymph homeostasis [14,15]. The high-quality evidence for salt restriction is modest, but observational data and consensus opinion support its use as a benign, sustainable intervention [14,15]. Caffeine, alcohol, and nicotine reduction are similarly low-cost and consistently recommended despite limited trial evidence [14,30]. Hydration discipline (regular fluid intake rather than alternating dehydration and bolus drinking) is emphasised in patient education [30].

Stress, fatigue, and sleep deprivation are widely reported triggers; cognitive behavioural therapy and stress-reduction programmes have small but consistent benefits in attack frequency and patient-reported handicap [30,44,46]. Vestibular rehabilitation has a defined role between attacks and after ablative treatment, supporting central compensation when vestibular hypofunction is established [16,43].

Acute attack management

Acute attacks are managed pragmatically: vestibular suppressants (prochlorperazine 5-10 mg, diazepam 2-5 mg, or promethazine 25 mg) for the duration of the attack; antiemetics for nausea; and reassurance that the attack will self-terminate within hours [14,16,49,50]. Patients should be given a written 'rescue plan' early in care and supplied with rapidly dissolving or buccal antiemetics for use at attack onset. Vestibular suppressants must be used short-term only; continuous use blocks central compensation and worsens chronic disequilibrium [14,49].

Preventive pharmacotherapy

Betahistine (16-48 mg three times daily, commonly 48 mg three times daily in Australian practice) is the most widely prescribed maintenance agent in Europe and Australasia, though it is not FDA-approved in the United States [16,20]. Mechanistic rationales include stria vascularis vasodilatation and central vestibular modulation [16]. The high-quality BEMED trial showed no superiority over placebo, but meta-analyses of older trials and pragmatic clinical experience continue to support a modest benefit in attack frequency reduction, particularly at higher doses [16,20]. Side effects are generally mild (headache, dyspepsia) [20].

Diuretics, most commonly hydrochlorothiazide-triamterene or acetazolamide, have been a traditional prophylactic option for decades, on the rationale that systemic diuresis may reduce endolymph volume [15,16]. Evidence is again modest, with no high-quality RCT showing clear superiority over placebo, but observational data and clinical consensus support a trial in patients with frequent attacks [15,16,39]. Electrolyte monitoring is required, particularly in older patients on other antihypertensives [49]. Some practitioners use acetazolamide 250 mg twice daily as an alternative carbonic-anhydrase-inhibitor approach.

Migraine prophylaxis (propranolol, topiramate, amitriptyline) has emerged as an important consideration when migrainous features are present, given the high MD-VM overlap [21,22]. In a patient with definite Ménière's plus migraine history, a trial of migraine prophylaxis may yield better attack control than escalating MD-specific therapy [21,22,25]. Conversely, in patients with purely classical MD without migrainous features, migraine prophylaxis is unlikely to add value.

Intratympanic therapy

Intratympanic (IT) therapy delivers pharmacological agents directly to the affected inner ear via round-window absorption from the middle ear [17,18,19]. Two agents dominate: corticosteroids and gentamicin. IT therapy is indicated when oral pharmacological prophylaxis fails to control attacks, before considering surgical options [17,18].

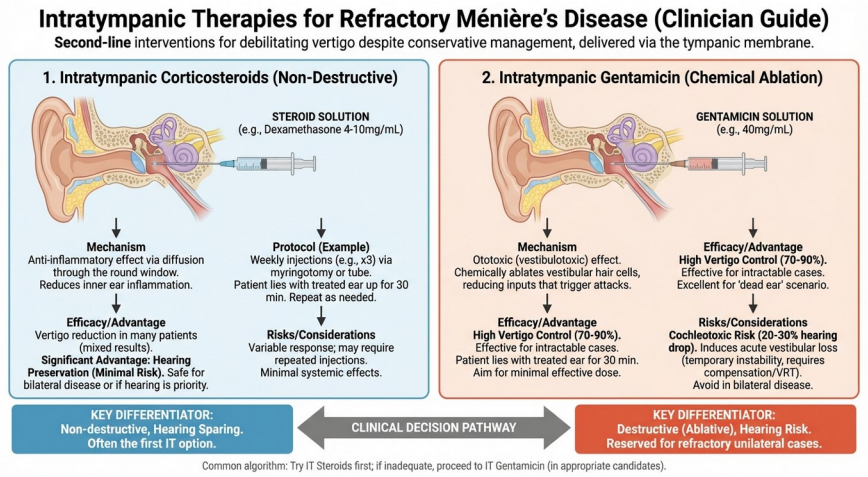

Figure 7. Intratympanic injection technique - round-window delivery via the tympanic membrane.
Source: Australian Dizziness Clinics educational schematic.

Intratympanic dexamethasone (4-24 mg/mL, weekly or three-doses-over-three-weeks regimens) is non-destructive and preserves hearing [17,19]. Attack-control rates of 60-80% are reported, though the placebo-controlled evidence is limited [17,19]. IT dexamethasone is the preferred first-line IT therapy when hearing preservation is a priority, particularly in only-hearing-ear disease, bilateral disease, or younger patients [17,19].

Intratympanic gentamicin (typically 26.7-40 mg/mL, low-dose titrated regimens) achieves ablative chemical vestibulectomy of the affected ear, with vertigo-control rates of 80-95% in the BEMED, low-dose, and titrated literature [17,18]. The trade-off is the risk of hearing loss: older single-dose protocols produced 25-30% rates of severe hearing decline, but modern low-dose and titrated regimens have reduced this to under 10-15% while preserving high vertigo-control rates [17,18]. IT gentamicin is now considered the most effective non-surgical option for refractory unilateral Ménière's [17,18,40]. It is contraindicated in only-hearing-ear disease and should be used with extreme caution in bilateral disease [17,18].

□ **Key Point:** The medical management pyramid: lifestyle and dietary measures at the base, betahistine and diuretics on the second tier, intratympanic dexamethasone as the conservative IT step, and intratympanic gentamicin as the most effective non-surgical option for refractory unilateral disease - at the cost of measurable hearing risk.

VIII. Adjuncts, Refractory Disease, and Surgical Options

A minority of Ménière's patients, under 10% in modern series, remain refractory to lifestyle modification, oral pharmacotherapy, and intratympanic therapy [14,17,18]. For these patients, surgical options range from non-destructive endolymphatic-sac procedures to hearing-preserving neurectomy and definitive

labyrinthectomy [18,40,41]. Patient counselling about expected outcomes, hearing risk, and persistent imbalance after surgery is essential [40].

Endolymphatic sac decompression or shunt surgery aims to decompress the endolymphatic sac and duct without destroying inner-ear function [20,40]. Long-running controversy surrounds its efficacy: Thomsen's classical 1981 sham-controlled trial showed no benefit over placebo, but subsequent meta-analyses and re-analyses suggest meaningful vertigo control in 70-85% of selected patients with low morbidity and minimal hearing risk [20,40]. The procedure remains an option in patients seeking a hearing-preserving alternative to ablative therapy, particularly those reluctant to accept gentamicin's hearing risk [40,41].

Table 5. Surgical and ablative options for refractory Ménière's disease.

Procedure	Vertigo control	Hearing preservation	Notes
Endolymphatic sac decompression / shunt	60-85%	Largely preserved	Long-standing controversy; favoured when hearing preservation paramount [20,40].
Intratympanic gentamicin (chemical vestibulectomy)	80-95%	Hearing loss 10-25% (low-dose protocols)	Most effective non-surgical option; modern low-dose regimens improve safety [17,18].
Vestibular nerve section	approx 95%	Preserved	Hearing-sparing definitive option; neurosurgical morbidity; only-hearing-ear caution [40,41].
Labyrinthectomy	Up to 99%	Complete hearing loss	Definitive; reserved for unilateral disease with non-serviceable hearing [40].

Vestibular nerve section (vestibular neurectomy) is a definitive surgical cure for vertigo while preserving cochlear function [40,41]. Performed via retrosigmoid or middle-fossa approaches, it selectively sections the vestibular division of the eighth nerve, achieving vertigo-control rates approaching 95% with preservation of useful hearing in 70-90% of cases [40,41]. The procedure is neurosurgically demanding, requires hospital admission, and carries small but real risks of facial nerve injury, CSF leak, and persistent imbalance during central compensation [40]. It is particularly suited to patients with serviceable hearing on the affected side who have failed all medical management [41].

Labyrinthectomy, surgical destruction of the inner ear via transmastoid or translabyrinthine approach, is the most definitive procedure, with vertigo-control rates approaching 99% but necessitating complete hearing loss in the operated ear [40]. It is reserved for unilateral disease with severe, non-serviceable hearing where vestibular ablation is the primary goal [40]. Recovery requires vestibular rehabilitation to support central compensation, and outcomes are generally excellent in well-selected patients [40,43].

Hearing rehabilitation should run in parallel with vertigo-focused interventions throughout the disease course. Modern digital hearing aids, programmable for fluctuating losses, transform communication outcomes in the affected ear [16]. Cochlear implantation is appropriate for patients with bilateral severe-to-profound loss; while traditionally regarded as a late-stage option, outcomes are favourable and the option should be raised proactively rather than reserved as a last resort [16]. CROS (contralateral routing of signal) and BiCROS hearing aids are useful in unilateral severe loss with normal contralateral hearing [16].

□ Important: Bilateral Ménière's (25-45% of patients at 20-year follow-up) fundamentally changes management. Ablative therapy in either ear must be avoided unless one ear is clearly inactive. Bilateral vestibular hypofunction produces severe oscillopsia and chronic imbalance - outcomes that outweigh any potential vertigo benefit. In bilateral disease, prioritise non-destructive therapy: betahistine, diuretics, IT dexamethasone, and sac surgery in selected

cases.

IX. Prognosis, Recurrence, Bilaterality, and Special Populations

The natural history of Ménière's disease is one of progressive vertigo attenuation against a background of progressive sensorineural hearing loss [10,39]. Vertigo attack frequency typically peaks in the first 3-7 years, then gradually declines as cumulative vestibular hypofunction establishes a stable, hypofunctional baseline ('vestibular burnout') [10,31,39]. By 10-20 years from onset, the majority of patients have largely vertigo-free intervals, though many retain motion intolerance and chronic mild disequilibrium [10].

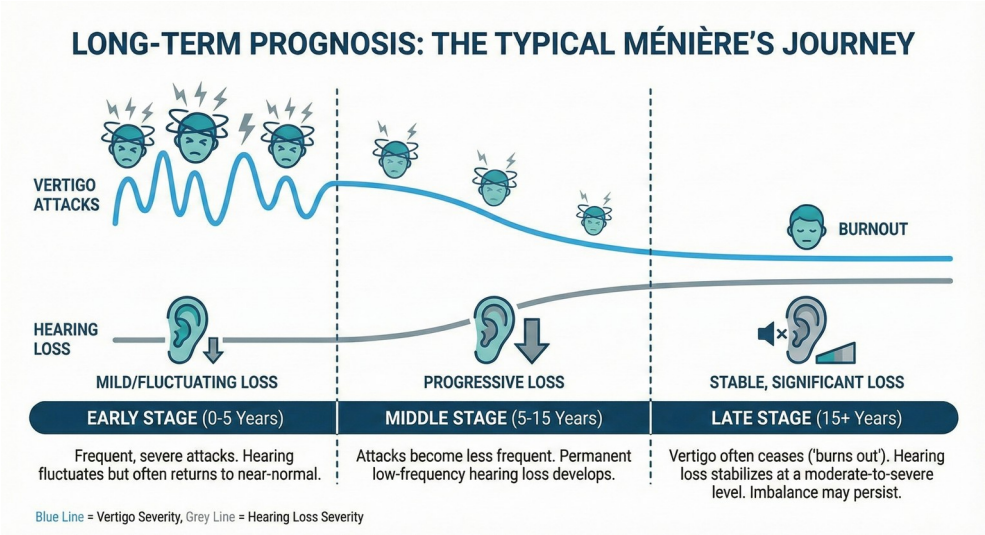

Figure 8. Natural history of Ménière's disease - vertigo attack frequency declines while sensorineural hearing loss progresses.

Source: Australian Dizziness Clinics educational schematic.

Hearing in the affected ear follows a different trajectory. Early fluctuation gives way to a progressive, stepwise loss that stabilises typically in the moderate-to-severe range by 10-15 years from onset [10,24]. Speech discrimination often deteriorates disproportionately to pure-tone thresholds and is the principal driver of communication handicap in long-standing disease [12,24]. Late hearing recovery is rare; loss is generally permanent and incremental [10].

Table 6. Long-term natural history of unilateral Ménière's disease.

Time from onset	Vertigo course	Hearing course
0-2 years	Attacks frequent, often clustered; high disability.	Fluctuating low-frequency SNHL; recovery typical between attacks [10].
2-5 years	Attack frequency variable; some plateau, others escalate.	Progressive thresholds; reduced inter-attack recovery [10,39].
5-10 years	Gradual vertigo decline in most; up to 80% improvement [10].	Flat or peaked moderate SNHL; mid/high frequencies affected [10].
10-20 years	'Burnout' in 60-80%; chronic motion intolerance common [10].	Severe SNHL in most; speech discrimination markedly reduced [10,24].
Bilateral conversion	Up to 25-45% by 20 years; alters management profoundly [2,8].	Bilateral SNHL; cochlear-implant candidacy may arise [16].

Bilateral disease is the most important prognostic variable. Cumulative bilateral conversion rates rise from under 10% at presentation to 25-45% at 20 years [2,8]. Predictors of bilateral conversion include longer disease duration, autoimmune comorbidity, and migrainous features [6,8]. Once bilateral, management

strategy shifts away from ablative therapy and toward systemic treatment, immune modulation in selected cases, and hearing rehabilitation including cochlear implantation [16].

Tumarkin otolithic crises - sudden, brief falls without warning or loss of consciousness - occur in 5-10% of MD patients and are particularly devastating, especially in older patients at risk of fragility fractures [12,23]. They tend to cluster either early or late in the disease and are an absolute indication for aggressive vertigo control, often with intratympanic gentamicin or surgical intervention [12,17,23].

Psychological comorbidity is the rule rather than the exception. Anxiety, depression, and agoraphobic avoidance are highly prevalent in MD and significantly worsen patient-reported outcomes [44,45,46]. Anticipatory anxiety around triggering scenarios (travel, driving, work) often outlasts active vertigo control and requires explicit psychological support, including cognitive behavioural therapy and selective serotonin reuptake inhibitor pharmacotherapy where indicated [46].

Special populations warrant tailored consideration. Paediatric MD (under 18 years) is rare but described; it is more frequently familial, more often bilateral, and may have a stronger autoimmune component [7,47]. Older patients (over 70) often have superimposed presbycusis and presbystasis, complicating audiometric interpretation and increasing fall risk; conservative management is generally preferred [16,43]. Pregnancy modifies management options - diuretics and betahistine are best avoided where possible, and migrainous co-morbidity often improves during pregnancy [16].

□ **Clinical Pearl:** Tell patients the truth early: vertigo attacks are likely to diminish over years, but hearing is unlikely to fully recover. Frame management as a long-term partnership focused on attack reduction, hearing optimisation, and psychological resilience rather than cure. Realistic expectations transform adherence and outcomes.

X. Guidelines, Controversies and Future Directions

Despite over 160 years of clinical observation, Ménière's disease remains an area of active controversy. The 2020 AAO-HNS Clinical Practice Guideline integrated the 2015 Bárány criteria and provides the most current evidence-based framework, but several aspects of management remain contested [14,48].

The relationship between Ménière's disease and vestibular migraine is the most clinically consequential debate. The substantial phenotypic overlap, the high rate of migraine comorbidity in MD, and the response of some 'MD' patients to migraine prophylaxis raise the possibility that a subgroup currently labelled Ménière's is actually severe vestibular migraine with auditory involvement, or vice versa [21,22,25]. The 2015 criteria explicitly require exclusion of better-fitting alternatives, and many vestibular physicians now trial migraine prophylaxis early in migraine-coloured MD presentations [21,22].

Betahistine remains a transatlantic divide. European and Australasian practice endorses it as standard first-line prophylaxis, while the United States, where it is not FDA-approved, does not [16,20]. The BEMED trial showed no superiority over placebo at standard or high doses, yet real-world data and earlier meta-analyses continue to support a modest effect [16,20]. The controversy is unlikely to resolve without further trials, and pragmatic practice continues with betahistine in most non-US settings.

Endolymphatic sac surgery is similarly contested. The Thomsen 1981 sham-controlled trial famously showed no benefit over placebo mastoidectomy, but subsequent observational data, re-analyses, and meta-analyses have consistently reported 70-85% vertigo control with low morbidity [20,40]. The 2020 AAO-HNS Guideline lists it as a reasonable option for refractory disease, with the caveat that evidence quality remains modest [14,40]. In Australian practice, it remains an option in selected patients prioritising hearing preservation over absolute vertigo control [41].

Dietary salt restriction, despite its long tradition, is supported by limited high-quality evidence [15,16]. Some commentators argue that its purported benefit may reflect general fluid-balance discipline rather than specific endolymphatic effect, while others maintain it should remain first-line given its safety and low cost [30]. The 2020 Guideline retains it as a reasonable option [14,15]. Practice patterns vary widely across regions.

Several emerging directions warrant attention. MRI hydrops imaging, 4-hour delayed 3D-FLAIR after intravenous or intratympanic gadolinium, is moving from research curiosity toward clinical adjunct in

expert centres [11,33]. Its diagnostic role remains adjunctive, but it may stratify atypical or research cohorts more precisely [11,33]. Genomic stratification, building on the López-Escámez subgroup framework, may eventually permit precision treatment, particularly for the autoimmune and familial subtypes [6,21].

Biologic and immune-modulating therapies are under investigation for the autoimmune subgroup of MD. Small studies of IL-1 beta blockade, anti-TNF agents, and tailored corticosteroid regimens have shown encouraging results in selected patients [4,6]. Inner-ear drug delivery via micro-pump or sustained-release matrix is being explored to optimise the gentamicin therapeutic-toxic balance, and gene-based approaches targeting COCH and other identified loci may emerge over the next decade [6,11].

Finally, the conceptual framing of Ménière's may itself evolve. Some experts argue that 'Ménière's disease' is best understood as a common endpoint - endolymphatic hydrops with episodic symptom expression - reached by multiple aetiological routes [6,21]. This 'final common pathway' model may eventually lead to disaggregation of MD into mechanistic subgroups (autoimmune, migrainous, vascular, genetic), each with its own optimal treatment [6]. For now, MD remains a clinical diagnosis defined by phenotype, but the next decade is likely to reshape that framework substantially.

□ Key Point: Ménière's disease in 2026 is best managed as a chronic, multifactorial inner-ear disorder using a stepwise pyramid: lifestyle and diet, betahistine and diuretics, intratympanic dexamethasone, intratympanic gentamicin, and surgery in the small refractory minority. The future likely belongs to subgroup-stratified, mechanism-targeted therapy.

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