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Neurotology: Pathophysiology, diagnosis and management of vestibular and auditory disorders - A Contemporary Review

Abstract

Neurotology exists at the intersection of neurology, otolaryngology, and neurosurgery, encompassing disorders of the inner ear and the central pathways governing vestibular, cochlear, and auditory function. This contemporary review major neurotologic conditions encountered in clinical practice: benign paroxysmal positional vertigo (BPPV), vestibular neuritis, Ménière disease, superior semicircular canal dehiscence, autoimmune inner ear disease, vestibular schwannoma, central vestibulopathies from brainstem and cerebellar pathology, vestibular migraine, and persistent postural-perceptual dizziness (PPPD). Special emphasis is placed on the neurotologic relevance of posterior fossa stroke, advanced imaging (3T endolymphatic hydrops MRI, ultra-high-resolution CT), quantitative vestibular function testing (video head impulse testing, vestibular evoked myogenic potentials), and the evolving therapeutic landscape including intratympanic pharmacotherapy, vestibular rehabilitation, cochlear gene therapy, and radiosurgery. A structured seven-step diagnostic algorithm for the clinician managing the dizzy patient is presented, accompanied by five evidence-referenced clinical tables. This article serves as a comprehensive, clinically actionable reference for otolaryngologists, neurologists, audiologists, and allied health professionals engaged in neurotologic care.

Keywords: neurotology, vertigo, BPPV, Ménière disease, vestibular neuritis, vestibular schwannoma, HINTS examination, cochlear gene therapy, central vestibulopathy, posterior fossa stroke, persistent postural-perceptual dizziness

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Introduction

Dizziness and disequilibrium are among the most prevalent symptoms in clinical medicine, accounting for approximately 7.5 million ambulatory physician visits annually in the United States. The etiology spans peripheral vestibular dysfunction, central vestibulopathies, cardiovascular causes, psychiatric co-morbidity, and polypharmacy - demanding a structured diagnostic approach that crosses subspecialty boundaries. Neurotology occupies a uniquely cross-disciplinary territory, addressing the neurological aspects of otologic disease and the otologic manifestations of neurologic disease.[1,2]

Over the past two decades, transformative advances in high-resolution temporal bone CT, 3-Tesla gadolinium-enhanced MRI, video head impulse testing (vHIT), vestibular evoked myogenic potentials (VEMPs), molecular genetics, and cochlear gene therapy have fundamentally altered diagnostic precision and therapeutic options. Concurrently, large multicentre trials have refined the evidence base underpinning repositioning maneuvers, intratympanic pharmacotherapy, stereotactic radiosurgery, and cochlear implantation with expanded audiological criteria.[3–5]

This review provides a systems-based synthesis of contemporary neurotology, structured to serve the practising otolaryngologist, the stroke neurologist managing posterior fossa disease, and the general physician confronted by the dizzy patient. A seven-step diagnostic algorithm and five evidence-graded clinical tables facilitate direct clinical application.

Functional anatomy and neurophysiology of the vestibular system

Peripheral labyrinth

The inner ear labyrinth comprises two functional divisions: the cochlea (audition) and the vestibular labyrinth, consisting of three semicircular canals (SCCs) and two otolith organs (utricle and saccule). The SCCs detect angular acceleration via endolymph displacement and cupular deflection. The utricle senses horizontal linear acceleration and head tilt; the saccule detects vertical linear acceleration. Hair cells transduce mechanical deflection into receptor potentials, driving afferent signals along CN VIII to the four ipsilateral vestibular nuclei in the dorsal pons and lateral medulla.[6,7]

Endolymph is produced by the stria vascularis and reabsorbed by the endolymphatic sac. Disruption of this fluid homeostasis underlies endolymphatic hydrops, the pathoanatomical substrate of Ménière disease. The vestibular nuclei project to the spinal cord via vestibulospinal tracts, the cerebellum (flocculus, nodulus, uvula), and the extraocular motor nuclei via the medial longitudinal fasciculus (MLF) - the substrate of the vestibulo-ocular reflex (VOR).[6,7]

The vestibulo-ocular reflex and clinical testing

The VOR generates compensatory eye movements equal and opposite to head rotation, maintaining retinal image stability at near-unity gain (normal >0.8) across physiological head velocities (0.5–5 Hz). The head impulse test (HIT), quantified by video head impulse testing (vHIT), exploits VOR gain asymmetry to localize canal-

specific hypofunction. VOR gain is preserved in central posterior fossa lesions - this forms the critical basis of the HINTS+ [Head Impulse test, Nystagmus type, Test of Skew, plus Hearing] examination strategy for acute vestibular syndrome.[7–9]

Cortical vestibular processing

Cortical vestibular processing involves the parieto-insular vestibular cortex (PIVC), temporoparietal junction, and premotor cortex, integrating vestibular, visual, proprioceptive, and somatosensory signals into a coherent spatial percept. Disruption of these higher-order networks underlies persistent postural-perceptual dizziness (PPPD) and the vestibular migraine complex. Pathological limbic gating of vestibular cortical signals in anxiety states contributes to the high comorbidity between vestibular disorders and psychiatric illness.[9]

Peripheral vestibular disorders

Benign paroxysmal positional vertigo (BPPV)

BPPV is the single most common cause of vertigo, with a lifetime prevalence of 2.4% and peak incidence in the sixth decade. The pathomechanism involves detachment of calcium carbonate otoliths (otoconia) from the utricular macula and their migration into a semicircular canal (canalolithiasis) or adherence to the cupula (cupulolithiasis). Posterior SCC BPPV accounts for 85–90% of cases; horizontal SCC for 8–10%; superior SCC is rare (Tables 1 & 2).[10,11]

Table 1 Differentiation of peripheral versus central acute vestibular syndrome (hints+ framework)

Feature	Peripheral Vertigo	Central Vertigo
Onset	Sudden, often positional	Gradual or sudden (stroke)
Nystagmus	Unidirectional, fatigable, horizontal-torsional	Direction-changing, vertical, or pure torsional; non-fatigable
HIT (vHIT)	Abnormal (catch-up saccade)	Normal - key red flag
HINTS+ exam	HIT+, no skew, unidirectional nystagmus	HIT–, skew deviation, or direction-changing - HIGH stroke risk
Hearing loss	Common (Ménière, labyrinthitis)	Uncommon; AICA infarct exception
Neurological signs	Absent	May be present (diplopia, dysarthria, ataxia)
MRI DWI	Normal	Infarct / demyelination / tumour

Table 2 BPPV canal classification, diagnostic tests, and repositioning maneuvers

Canal	Prevalence	Diagnostic Test	Treatment Maneuver
Posterior SCC	~85–90%	Dix-Hallpike	Epley (CRP); Semont liberatory
Horizontal SCC - canalolithiasis	~8–10%	Supine roll test (geotropic nystagmus)	Barbecue roll (360° rotation); Gufoni
Horizontal SCC - cupulolithiasis	~2%	Supine roll test (apogeotropic nystagmus)	Gufoni (towards affected ear); forced prolonged positioning
Superior (anterior) SCC	Rare (<1%)	Deep Dix-Hallpike / straight head-hanging	Reverse Epley; Yacovino maneuver

Diagnosis is confirmed by the Dix-Hallpike maneuver (posterior/superior SCC) and the supine roll test (horizontal SCC). The Epley canalith repositioning procedure (CRP) achieves resolution in ~80% of posterior BPPV cases at first treatment (NNT = 1.3), with the Semont liberatory maneuver as an evidence-based alternative. Horizontal canal BPPV is managed with the barbecue roll or Gufoni maneuver. Pharmacological vestibular suppressants do not treat the underlying canalolithiasis and should not be used as primary therapy.[10,11]

Vestibular neuritis

Vestibular neuritis presents as acute sustained vertigo, postural instability, and oscillopsia without hearing loss, typically peaking over hours and subsiding over days to weeks. The superior vestibular nerve (SVN) division is preferentially affected, likely reflecting reactivation of herpes simplex virus type 1 (HSV-1) within the vestibular ganglia. Horizontal canal vHIT demonstrates reduced VOR gain with corrective saccades; caloric testing confirms unilateral canal paresis.[12]

Management comprises brief vestibular suppressants in the acute phase, followed by early mobilization and individualized vestibular rehabilitation (Cawthorne-Cooksey or gaze stability programmes). High-dose oral methylprednisolone (100 mg tapered over 3 weeks) accelerates vestibular function recovery. Central compensation typically occurs within 3-6 months; incomplete compensation necessitates specialist vestibular physiotherapy.[12,13]

Ménière's disease

Ménière's disease is characterised by episodic vertigo (20 min–12 h), fluctuating low-to-mid frequency sensorineural hearing loss (SNHL), aural fullness, and tinnitus, with an estimated prevalence of 50–200 per 100,000. The underlying pathology is endolymphatic hydrops. Diagnostic criteria follow the 2020 AAO-HNS classification (Table 3). High-resolution 3T MRI after intratympanic gadolinium injection can directly visualise endolymphatic hydrops with sensitivity and specificity exceeding 80%.[13,14]

Medical management often includes dietary sodium restriction (<1,500 mg/day), diuretics, betahistine, and trigger avoidance. Intratympanic dexamethasone reduces attack frequency. For refractory cases, intratympanic gentamicin achieves vestibular ablation with vertigo control rates of 80–90%. Endolymphatic sac decompression preserves hearing with intermediate vertigo control. Labyrinthectomy or vestibular neurectomy are reserved for end-stage disease.[14,15]

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Table 3 Ménière disease diagnostic criteria (aao-hns 2020) and investigation protocol

Category	Criteria
Definite Ménière Disease	≥2 spontaneous episodes of vertigo lasting 20 min–12 h; Low-to-mid-frequency SNHL confirmed audiometrically; Fluctuating aural symptoms (fullness, tinnitus, hearing loss).
Probable Ménière Disease	≥2 episodes of dizziness/vertigo (20 min–24 h); Fluctuating aural symptoms.
Key Investigations	Pure tone audiometry; Electrocochleography (ECoG); Gadolinium-enhanced 3T MRI endolymphatic hydrops protocol; cVEMP & oVEMP; Caloric testing / vHIT

Superior semicircular canal dehiscence (SSCD)

SSCD results from a bony defect in the roof of the superior SCC, creating a pathological third window in the inner ear. The syndrome includes sound- or pressure-induced vertigo and oscillopsia (Tullio phenomenon and Hennebert sign), low-frequency air-bone gap on audiometry without middle ear pathology, and low-threshold cVEMPs with enhanced oVEMP amplitudes. Diagnosis is confirmed by high-resolution temporal bone CT (0.625 mm slices, Pöschl and Stenvers reformats). Surgical plugging or resurfacing via middle fossa craniotomy or transmastoid approach provides excellent outcomes in appropriately selected patients.[15,16]

Autoimmune inner ear disease (AIED)

AIED presents as bilateral progressive SNHL and vestibular dysfunction, often episodic, typically affecting adults aged 20-50 years with a female predominance. It may be organ-specific or part of systemic autoimmune disorders including SLE, Cogan syndrome, Susac syndrome, and granulomatosis with polyangiitis (GPA). Rapid bilateral progressive SNHL with systemic features mandates urgent serological evaluation. Treatment with high-dose corticosteroids remains the cornerstone; steroid-responsive patients may benefit from long-term immunosuppressants. Cochlear implantation may be indicated for severe-profound SNHL refractory to medical therapy.[17]

Central Vestibular and Neurotologic Disorders

Posterior fossa stroke - a neurotologic emergency

Posterior fossa stroke affecting the cerebellum and brainstem may mimic peripheral vestibular neuritis. The anterior inferior cerebellar artery (AICA) supplies the labyrinth via the internal auditory artery in ~80% of individuals; AICA infarction therefore produces combined peripheral and central neurotologic deficits alongside brainstem signs.[18,19]

The HINTS+ bedside examination (Head Impulse test, Nystagmus type, Test of Skew, plus Hearing) reliably stratifies acute vestibular syndrome with sensitivity >96% for stroke detection - exceeding early diffusion-weighted MRI (DWI), which may be falsely negative within 48 hours of posterior fossa infarction. A "dangerous" HINTS result - normal HIT, direction-changing or vertical nystagmus, or skew deviation - mandates urgent neuroimaging and neurology input regardless of initial MRI findings. PICA infarction produces the Wallenberg (lateral medullary) syndrome and must be systematically sought.[18–20]

Vestibular migraine

Vestibular migraine is the most common cause of episodic vertigo in adults, with a prevalence of 1–3% and accounting for up to 11% of neurotology clinic referrals. The IHS/Bárány Society 2022 criteria require: ≥5 episodes of moderate-to-severe vestibular symptoms lasting 5 min–72 h; current or prior migraine history; ≥50% of episodes with migrainous features; and exclusion of other causes. Pathophysiology involves cortical spreading depression-triggered trigeminovascular activation with secondary effects on the vestibular nuclei. Treatment parallels standard migraine prophylaxis: topiramate, propranolol, amitriptyline, and CGRP antagonists.[20,21]

Persistent postural-perceptual dizziness (PPPD)

PPPD is defined by the Bárány Society as non-spinning dizziness or unsteadiness on ≥15 days/month for ≥3 months, worsened by upright posture, active/passive motion, and visual stimulation. It arises as a maladaptive high-gain threat-avoidance postural control strategy following an acute vestibular trigger. Neuroimaging and vestibular function testing are normal or non-specifically abnormal. Management includes psychoeducation, vestibular rehabilitation with progressive desensitisation, CBT, and SSRIs/SNRIs - venlafaxine has the strongest pharmacological evidence base.[21,22]

Vestibular schwannoma (Acoustic neuroma)

Vestibular schwannoma (VS), a benign Schwann cell neoplasm arising from CN VIII, accounts for ~8% of intracranial tumours (incidence 1.2–1.7 per 100,000/year). Bilateral VS is pathognomonic of Neurofibromatosis Type 2. The classic presentation is unilateral progressive SNHL with or without tinnitus; vertigo is often mild due to slow-growth central compensation. Gadolinium-enhanced MRI of the internal auditory meati is the investigation of choice (sensitivity >95%).[22,23]

Management follows the Koos grading system (Table 4), with options of active surveillance, stereotactic radiosurgery (Gamma Knife - GKRS), and microsurgical resection. GKRS achieves tumour control in 93–97% of Koos I–III tumours at 5 years with hearing preservation in ~50%, avoiding the morbidity of open surgery. Management choice must integrate tumour growth rate, hearing status, age, NF2 status, and patient preference in multidisciplinary discussion.[22–24]

Table 4 Koos grading of vestibular schwannoma and management framework

Koos Grade	Description	Management Preference
I	Purely intracanalicular tumour	Observation or SRS
II	Small intracranial extension; does not reach brainstem	SRS (Gamma Knife) or observation
III	Reaches brainstem; no compression	SRS or microsurgery
IV	Brainstem compression ± hydrocephalus	Microsurgery preferred

Neurotologic aspects of auditory disorders

Auditory neuropathy spectrum disorder (ANSD)

ANSD is characterised by present otoacoustic emissions (OAEs) with absent or grossly abnormal auditory brainstem responses (ABRs), indicating disrupted neural synchrony despite intact

outer hair cell function. Etiologies include OTOF gene mutations (otoferlin), DIAPH1 mutations, and perinatal hypoxia. Speech perception is disproportionately worse than pure tone thresholds. Cochlear implantation often achieves excellent outcomes in OTOF-related ANSD, making genetic testing mandatory in the evaluation pathway.[24,25]

Auditory processing disorder (APD)

APD refers to deficits in central auditory nervous system processing despite normal peripheral hearing thresholds. Diagnosis requires a central auditory processing battery and must be performed by a highly trained audiologist. Neurologic associations include epilepsy, traumatic brain injury, multiple sclerosis, and neurodevelopmental disorders including ADHD and dyslexia.[25]

Sudden sensorineural hearing loss (SSNHL)

SSNHL (≥30 dB hearing loss across ≥3 consecutive audiometric frequencies over ≤72 hours) is a neurotologic emergency with annual incidence of 5-27 per 100,000. Etiology is idiopathic in ~80–90%; identifiable causes include viral labyrinthitis, AICA infarct, autoimmune disease, and retrocochlear pathology. MRI with gadolinium is useful to exclude suspected VS. High-dose systemic corticosteroids (prednisolone 1 mg/kg/day with taper) are standard of care; intratympanic dexamethasone offers comparable cochlear drug levels with superior systemic safety.[26]

Emerging diagnostic technologies and therapeutic innovations

Advanced vestibular function testing

Video head impulse testing (vHIT) provides quantitative VOR gain measurements across all six semicircular canals (dynamic range 0.01–3.0 Hz), detecting overt and covert saccades to enable canal-specific localization of hypofunction. Coupling vHIT with the HINTS+ algorithm has transformed bedside acute vestibular syndrome assessment. cVEMPs and oVEMPs evaluate saccular and utricular function, complementing vHIT across different neurotologic diagnoses.[27]

Molecular biomarkers and cochlear gene therapy

Next-generation sequencing (NGS) panels enable identification of causative variants in over 150 genes associated with hereditary hearing loss, transforming the etiological workup of non-syndromic SNHL. The OTOF gene (DFNB9) is the most clinically actionable target: Phase I/II clinical trials (2023–2025) using AAV vectors delivering wild-type OTOF to cochlear hair cells have demonstrated hearing restoration in pediatric patients with OTOF-related ANSD - a paradigm shift in inner ear therapeutics.[28,29]

Cochlear implantation: expanding indications and vestibular implants

Cochlear implant (CI) candidacy has expanded to include single-sided deafness, ANSD, hybrid electro-acoustic stimulation, and AIED. In the UK, NICE guidance (2019) broadened adult CI criteria to include moderately severe SNHL when audiological rehabilitation with hearing aids is insufficient. The vestibular prosthesis - delivering electrical stimulation to the ampullary nerves to restore VOR function - is currently in Phase I clinical trials for bilateral severe vestibulopathy, with demonstrated VOR gain restoration and subjective improvement in oscillopsia.[29,30]

Diagnostic algorithm for the dizzy patient

The evaluation of the dizzy patient requires integration of symptom characterization, bedside neurotologic examination, targeted vestibular function testing, and selective neuroimaging. The seven-step algorithm below (Table 5) guides the clinician from initial presentation to diagnosis and specialist referral.

Table 5 Structured seven-step diagnostic algorithm for the dizzy patient

Step	Action / Decision
Step 1 - Onset & timing	Acute onset (<72 h): apply HINTS+ exam immediately. Episodic: characterise duration (sec = BPPV; min–h = Ménière/TIA; h = vestibular migraine)
Step 2 -HINTS+ exam	HIT normal + direction-changing/vertical nystagmus + skew = CENTRAL (stroke until proven otherwise). Urgent MRI DWI. Note: early MRI DWI may be false-negative in posterior fossa infarct — repeat at 24–48 h.
Step 3 - Audiological screen	Bedside tuning fork (Weber/Rinne) + same-day pure tone audiometry. SNHL + vertigo: consider Ménière, AICA stroke, autoimmune IED, labyrinthitis.
Step 4 - Positional testing	Dix-Hallpike: posterior BPPV (upbeating-torsional, fatigable, latency). Supine roll: horizontal canal BPPV.
Step 5 - Vestibular function	vHIT (canal-specific VOR gain), cVEMP/oVEMP, caloric testing. Unilateral hypofunction: vestibular neuritis vs schwannoma. Bilateral: ototoxicity, CANVAS, autoimmune.
Step 6 - Neuroimaging	MRI internal auditory meati (IAM) with gadolinium for unilateral SNHL, focal signs, or retrocochlear suspicion. 3T hydrops protocol for Ménière.
Step 7 - Specialist referral	Multidisciplinary: Neurotology/ENT, Neurology (central signs), Audiology (CI candidacy), Vestibular rehabilitation physiotherapy.

Critical red flags mandating emergency neuroimaging include: (i) acute onset severe headache; (ii) normal vHIT with atypical nystagmus; (iii) direction-changing or purely vertical nystagmus; (iv) skew deviation; (v) any focal neurological sign; (vi) inability to stand; (vii) new-onset hearing loss in a patient with vascular risk factors. The HINTS+ examination, performed competently, outperforms MRI DWI within the first 24-48 hours for detection of posterior fossa infarction.

Medicolegal considerations: driving and vestibular disorders

In the United Kingdom, DVLA regulations stipulate: Group 1 (car/motorcycle) drivers with BPPV must not drive during symptomatic episodes; Ménière disease requires notification and specialist assessment for both Group 1 and Group 2 (bus/lorry) licences; sudden incapacitating vertigo mandates a minimum 12-month episode-free

period before Group 2 licence restoration. Clinicians bear a duty to advise patients clearly and document this advice. Failure to do so carries significant medicolegal exposure. In jurisdictions outside the UK, locally applicable driving authority regulations should be consulted.[31]

Conclusion

Neurotology encompasses a clinically demanding spectrum of disorders whose accurate diagnosis and management requires an integrative approach informed by audiology, otolaryngology and neurologic expertise. The evolution of quantitative vHIT, 3T hydrops MRI, NGS genetic panels, and cochlear gene therapy necessitates continuous updating of clinical practice frameworks.

The HINTS+ examination remains the most powerful bedside tool for stratifying acute vestibular syndrome and directing timely neuroimaging. The expanding genetic and molecular evidence base is transforming the management of hereditary hearing loss, with cochlear gene therapy poised to become a clinical reality within the current decade. The structured seven-step diagnostic algorithm and five clinical tables presented herein provide a practical, evidence-grounded reference for the practising otolaryngologist, neurologist, and allied health professional.

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Conflicts of interest

The authors declare that there are no conflicts of interest.

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