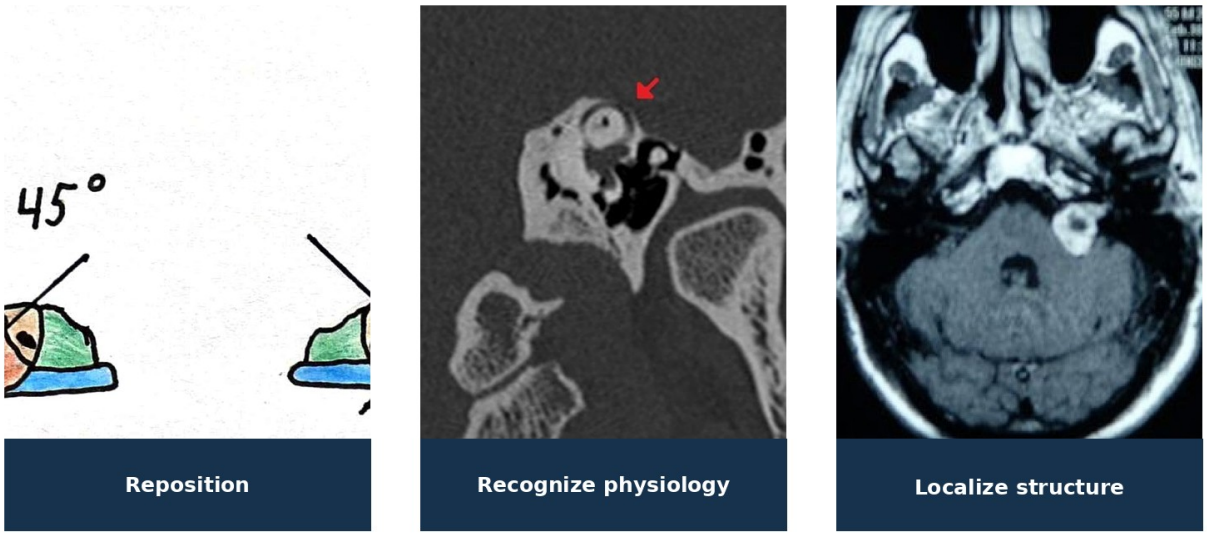
THE VESTIBULAR SYSTEM

VOLUME III

Clinical Vestibular Syndromes: Diagnosis and Management

From physiologic pattern to disease-specific treatment



Cover montage. Repositioning maneuver, superior canal dehiscence CT, and vestibular schwannoma MRI. Web images; full credits in Appendix E.

Resident study guide • July 2026

NAME THE SYNDROME BEFORE THE DISEASE

ORIENTATION. How to use this volume

Volumes I and II built the physiologic and localization framework. Volume III asks the final clinical questions: Which disease process best explains the syndrome, which alternatives are dangerous, and which intervention changes outcome? The organizing principle remains the same. A diagnosis is defensible only when the time course, triggers, examination, hearing findings, and testing all point in the same direction.

The most common error in vestibular medicine is premature closure. A positional complaint is called BPPV before positional nystagmus is characterized. Continuous vertigo is called vestibular neuritis before an acute vestibular syndrome is established and stroke is addressed. Recurrent attacks are called Ménière disease because tinnitus is present, even though the audiogram and attack duration do not fit. Chronic visual-motion intolerance is treated as persistent peripheral injury when the current disorder is PPPD. This volume is designed to slow that closure down without making the reasoning cumbersome.

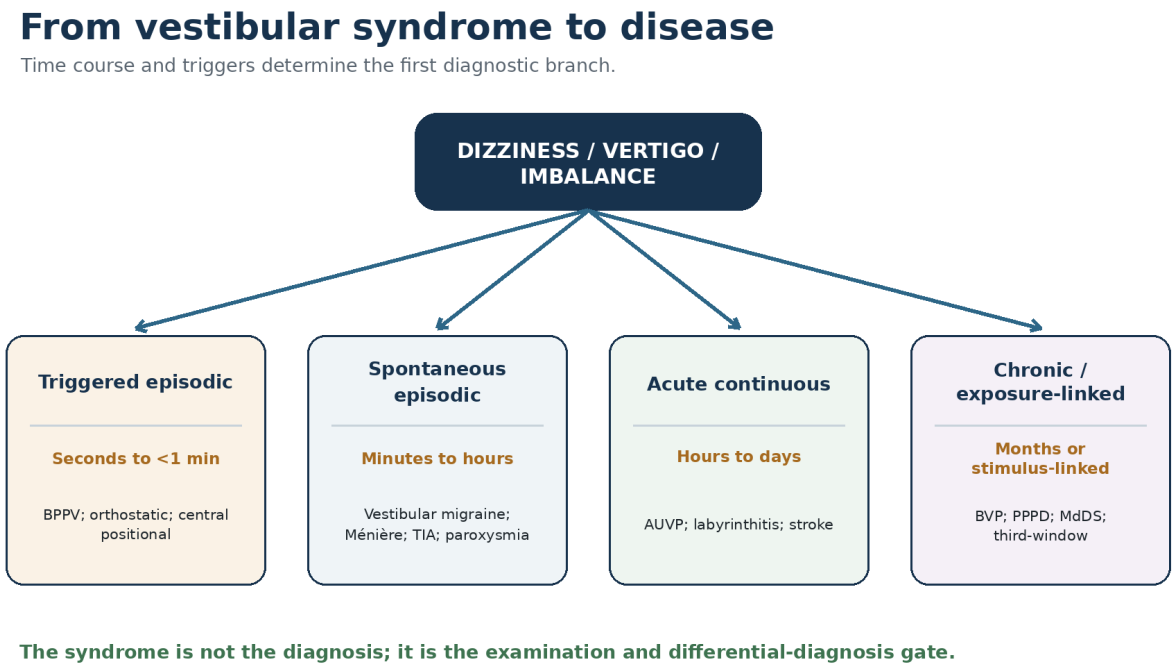


Figure 1. The syndrome-first map. Time course and triggers establish the diagnostic branch before disease labels are applied. Original synthesis.

The disease chapter template

Each major disorder is approached through the same sequence:

1. Mechanism: what physiologic signal or central computation has failed?
2. Clinical phenotype: what does the patient experience, and over what time course?

3. Expected findings: what should be present on examination, audiometry, vestibular testing, or imaging?
4. Disconfirming findings: which features should force reconsideration of the diagnosis?
5. Management: what treats the attack, what changes recurrence, and what restores function?
6. Boards bridge: what distinction is most likely to be tested?

> **A diagnostic label is a model, not a symptom synonym.**
>
> The label should predict the attack duration, provoking conditions, nystagmus, hearing phenotype, and course. When it does not, the model needs revision.

Contents

Part	Clinical question	Core disorders
1. Syndrome to disease	Which branch of the differential is active?	Triggered episodic, spontaneous episodic, acute continuous, chronic, and third-window syndromes
2. Triggered episodic	Does position produce a canal-specific response?	Posterior, horizontal, and anterior canal BPPV; central positional mimics
3. Acute continuous	Is this a stable peripheral tone imbalance or a dangerous central lesion?	AUVP, labyrinthitis/SSNHL, stroke, Ramsay Hunt
4. Spontaneous episodic	Why does the vestibular system fail in attacks?	Ménière disease, vestibular migraine, TIA, vestibular paroxysmia
5. Chronic	Why has imbalance persisted?	Uncompensated loss, BVP, presbyvestibulopathy, CANVAS, PPPD, MdDS
6. Sound/pressure linked	Is a third mobile window changing labyrinthine mechanics?	SCDS, perilymphatic fistula, enlarged vestibular aqueduct
7. Structural and toxic	Is progressive asymmetry or bilateral injury present?	Vestibular schwannoma, ototoxicity, autoimmune inner-ear disease
8. Treatment principles	How should treatment be matched to mechanism?	Suppressants, rehabilitation, fall prevention, procedural escalation
9. Cases and review	Can the framework be applied under pressure?	Integrated cases, criteria cards, boards pearls

Table 1. Narrative structure of Volume III.

THE FIRST LABEL SHOULD BE TEMPORAL

1. From syndrome to disease

A vestibular syndrome is a reproducible pattern of timing, triggers, and associated findings. It functions as a diagnostic gate. Once a patient is placed in the correct gate, many tempting but incompatible diagnoses fall away. The same symptom - “the room spins” - can describe posterior canal BPPV, vestibular migraine, acute unilateral vestibulopathy, or cerebellar stroke. The duration and circumstances make those conditions separable.

Triggered episodic vestibular syndrome

In triggered episodic vestibular syndrome, the patient is normal or near baseline until a specific action provokes a brief attack. The canonical trigger is a change in head position relative to gravity. The attack is usually measured in seconds, and the diagnostic examination is positional. BPPV dominates this branch, but orthostatic hypotension, central positional nystagmus, and occasionally mechanical cervical or vascular problems can mimic it. The key is that the trigger creates an attack from baseline rather than merely worsening continuous symptoms.

Spontaneous episodic vestibular syndrome

Here the attacks begin without an obligatory immediate trigger and typically last minutes to hours. The examination between attacks may be normal. The diagnosis therefore depends heavily on a carefully reconstructed attack phenotype: duration, headache biology, fluctuating hearing, focal neurologic symptoms, and stereotypy. Vestibular migraine and Ménière disease are common; transient ischemia is the dangerous alternative. Very brief, frequent, stereotyped attacks suggest vestibular paroxysmia rather than either of the common disorders.

Acute vestibular syndrome

Acute vestibular syndrome is continuous dizziness or vertigo with nausea or vomiting, spontaneous nystagmus, and gait or postural instability lasting days. Head motion worsens symptoms, but it does not trigger isolated attacks. The central task is to distinguish an acute unilateral peripheral vestibular tone imbalance from posterior circulation stroke. This is the branch in which the wrong use of a familiar label - “vertigo,” “neuritis,” or “labyrinthitis” - carries the highest immediate risk.

Chronic vestibular syndrome

Symptoms lasting months require a different question. Is there persistent sensory loss, failure of compensation, a neurodegenerative multisensory disorder, or a functional-perceptual pattern that outlived the inciting lesion? Bilateral vestibular hypofunction is exposed by darkness, uneven ground, and walking with head motion. PPPD is exposed by upright posture, movement, and complex visual environments. MdDS is defined by continuous rocking or swaying after passive motion and a paradoxical improvement during re-exposure to motion.

Sound-, pressure-, and exertion-induced symptoms

When sound, Valsalva, pressure changes, or exertion reliably produce vertigo, oscillopsia, or an eye movement, think in terms of altered inner-ear mechanics. A third mobile window can shunt acoustic or pressure energy away from the cochlea and toward vestibular sensors. The diagnosis is not a CT finding alone; it requires a concordant symptom, physiologic evidence of a third-window effect, and anatomically appropriate imaging.

Syndrome	Typical duration	First examination	Common diagnoses	Dangerous alternative
Triggered episodic	Seconds, occasionally <1 minute	Dix-Hallpike and supine roll	BPPV	Central positional lesion; orthostasis
Spontaneous episodic	Minutes to hours	Attack history, hearing, migraine and neurologic features	Vestibular migraine; Ménière disease	Posterior circulation TIA
Acute continuous	Days	Ocular motor exam, gait, hearing; trained HINTS when valid	AUVP	Posterior circulation stroke
Chronic	Months or longer	Dynamic visual acuity, gait, sensory conditions, vestibular lab	BVP; PPPD; incomplete compensation	Neurodegeneration; structural lesion
Sound/pressure linked	Seconds and time-locked to stimulus	Sound/pressure-provoked eye movements, audiogram, VEMPs	SCDS / third window	Other middle/inner-ear or central disease

Table 2. The syndrome as a diagnostic gate.

Resident checkpoint

Before naming a disease, state the syndrome in one sentence. Example: “This is a spontaneous episodic vestibular syndrome with 2-hour attacks, unilateral fluctuating low-frequency hearing loss, tinnitus, and aural pressure.” The disease label should then feel earned.

LET THE NYSTAGMUS NAME THE CANAL

2. Triggered episodic vestibular syndrome: BPPV and its mimics

Benign paroxysmal positional vertigo is a mechanical disorder. Otoconia that normally weight the utricular macula become displaced into a semicircular canal or adhere to the cupula. A change in head position relative to gravity then creates an inappropriate canal signal. The resulting vertigo and nystagmus are not arbitrary: their axis reflects the stimulated canal and their time course reflects the mechanics of moving particles or a persistently weighted cupula.

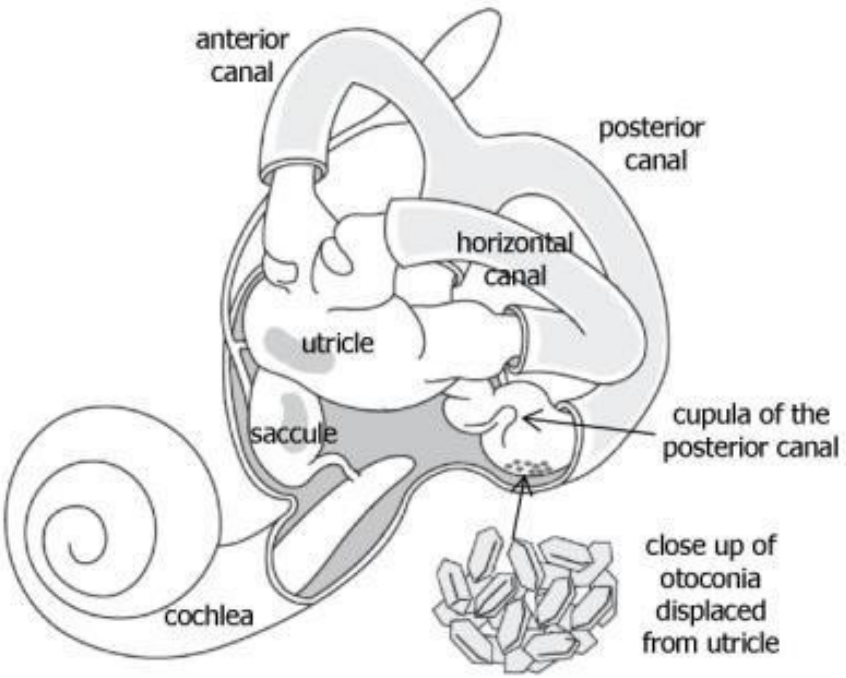


Figure 2. Otoconia displaced from the utricle into a semicircular canal create gravity-dependent endolymphatic or cupular forces. Wikimedia Commons, CC BY-SA 4.0.

Canalithiasis versus cupulolithiasis

In canalithiasis, free-floating particles move after the head is placed in a provoking position. The response typically has a brief latency, builds, and then decays as the particles settle. In cupulolithiasis, particles adhere to the cupula or otherwise create a persistent gravity-sensitive cupular deflection. Positional nystagmus may begin with less latency and persist as long as the position is maintained. Real patients do not always display textbook timing, but the distinction remains useful because it predicts the expected duration and can influence the maneuver selected.

Posterior canal BPPV

Posterior canal BPPV is the most common variant. The Dix-Hallpike maneuver brings the tested posterior canal into a gravity-sensitive plane. A typical response is transient upbeat-torsional nystagmus, with the upper poles of the eyes beating toward the affected ear. The torsional

component is easiest to see by watching the iris or a conjunctival vessel. A strong vertical component without the expected torsion, particularly if persistent or reproducible in multiple positions, should prompt concern for a central mimic.

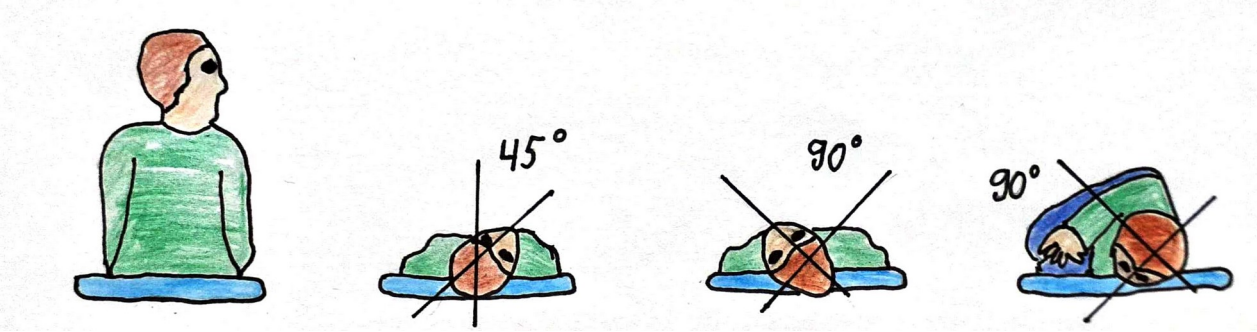


Figure 3. Canalith repositioning for posterior canal BPPV. The sequence uses gravity to move particles from the posterior canal toward the utricle. Ruhrgur via Wikimedia Commons, CC BY-SA 4.0.

The Epley maneuver is not a sequence to memorize as choreography. Each position advances particles along the posterior canal and common crus toward the vestibule. The treated ear begins down, the head rotates through the contralateral side, and the patient rolls so the nose points toward the floor before sitting. A Semont liberatory maneuver uses rapid side-to-side movement and may be useful when mobility or local practice favors it. Routine post-procedure restrictions are not recommended, and vestibular suppressants should not replace repositioning.

Horizontal canal BPPV

Horizontal canal disease is sought with the supine roll test, usually with the head elevated approximately 20 to 30 degrees to bring the horizontal canals closer to the earth-horizontal plane. Geotropic horizontal nystagmus usually reflects canalithiasis; the stronger side is typically affected because excitation is more powerful than inhibition. Apogeotropic horizontal nystagmus more often reflects cupulolithiasis or debris in the anterior arm; the weaker side is usually affected, but interpretation becomes difficult when responses are nearly symmetric or when central positional nystagmus is present.

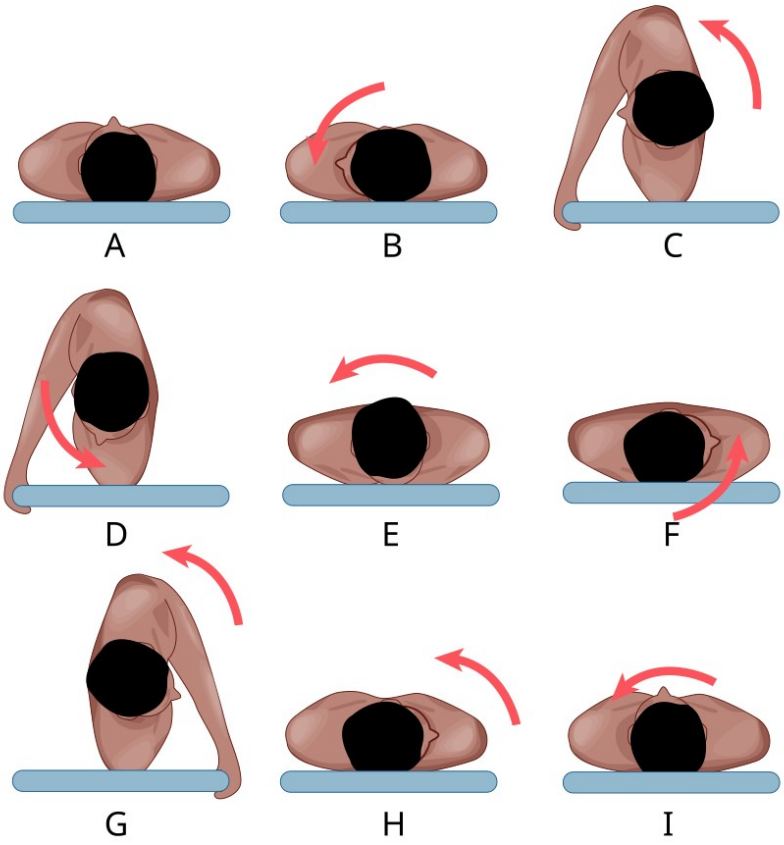


Figure 4. Lempert or "barbecue roll" maneuver for horizontal canal BPPV. Hariadhi via Wikimedia Commons, CC BY-SA 4.0.

Treatment must match the subtype. A Lempert or barbecue roll moves geotropic canalithiasis through sequential 90-degree rotations toward the utricle. Gufoni-type maneuvers use a brisk lateral movement and head turn; variants are chosen for geotropic versus apogeotropic patterns. Apogeotropic disease may first need conversion to a geotropic pattern or liberation of material from the cupula before a clearing maneuver succeeds.

Anterior canal BPPV

Anterior canal BPPV is uncommon and is easily overdiagnosed. A deep head-hanging position may provoke downbeat-torsional nystagmus. The torsional component should point toward the involved ear, but it may be subtle. Because positional downbeat nystagmus is also a classic central sign, the diagnosis requires a coherent, transient canal pattern and a low threshold to evaluate atypical or persistent cases. A Yacovino deep head-hanging maneuver can be used without first deciding the side, although side-specific variants exist.

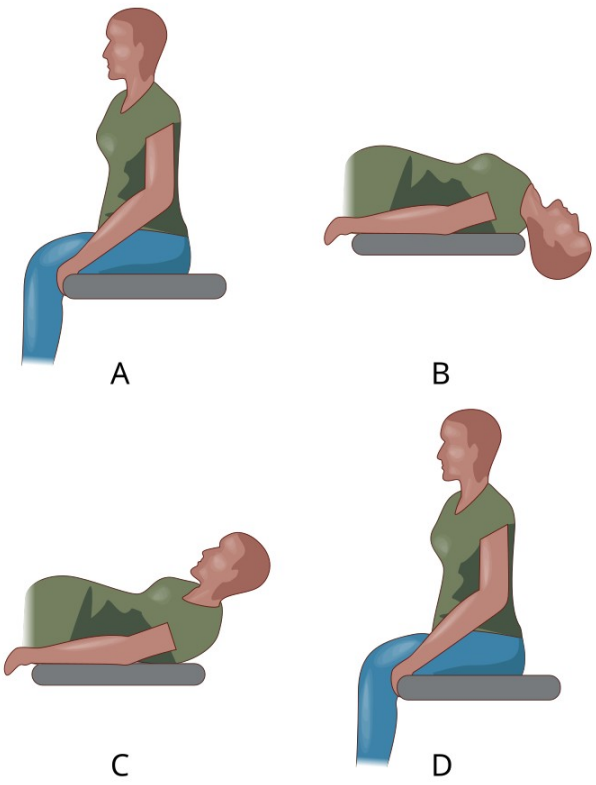


Figure 5. Yacovino deep head-hanging maneuver for suspected anterior canal BPPV. Hariadhi via Wikimedia Commons, CC BY-SA 4.0.

Pattern	Likely site	Typical positional nystagmus	Common treatment	Major caution
Dix-Hallpike positive	Posterior canal	Transient upbeat-torsional; upper poles toward affected ear	Epley or Semont	Pure vertical or persistent response suggests central disease
Roll test geotropic	Horizontal canal canalithiasis	Horizontal, toward ground; usually stronger on affected side	Lempert/barbecue or geotropic Gufoni	Large asymmetry helps lateralization; small asymmetry is less reliable
Roll test apogeotropic	Horizontal cupula/anterior arm	Horizontal, away from ground; often weaker on affected side	Apogeotropic Gufoni/Appiani or conversion strategy	Central positional nystagmus may mimic
Deep head hanging	Anterior canal	Downbeat with canal-consistent torsion	Yacovino/deep head-hanging	Central downbeat nystagmus must be excluded

Table 3. Canal-specific patterns and maneuvers.

When “refractory BPPV” is not simple BPPV

Persistent symptoms after an appropriate maneuver can reflect residual particles, incorrect canal or side assignment, multicanal disease, cupulolithiasis, canal conversion, or a second diagnosis. The patient may also have transient residual disequilibrium even after positional nystagmus resolves. Reassess the actual eye movement rather than repeating the same maneuver reflexively. Atypical direction, absent latency, lack of fatigability, severe truncal ataxia, new headache, focal neurologic findings, or failure to fit a canal plane should reopen the central differential.

Boards bridge

BPPV is diagnosed by a canal-specific positional nystagmus pattern, not by the sentence “dizzy when I move.” Imaging, vestibular laboratory testing, and chronic suppressants are generally unnecessary when the pattern is classic and no contradictory features are present.

PERIPHERAL TONE IMBALANCE OR CENTRAL EMERGENCY?

3. Acute continuous vestibular syndrome

The acute vestibular syndrome is a high-stakes pattern: sudden or rapidly progressive continuous vertigo or dizziness lasting at least a day, spontaneous nystagmus, nausea or vomiting, and marked gait or postural instability. The most important differential is acute unilateral vestibulopathy versus posterior circulation stroke. The absence of weakness, aphasia, or obvious dysmetria does not make stroke unlikely enough to dismiss; small brainstem and cerebellar lesions can present with isolated vestibular findings.

Acute unilateral vestibulopathy

Acute unilateral vestibulopathy, often called vestibular neuritis, is the sudden loss of tonic and dynamic vestibular input from one vestibular nerve or labyrinth without acute hearing loss or other focal otologic or neurologic signs. At rest, the intact side continues to fire while the affected side falls silent. The brain interprets the asymmetry as rotation toward the intact side. The eyes drift slowly toward the lesion and reset with fast phases away from it, producing unidirectional peripheral vestibular nystagmus. Head impulses toward the injured side generate corrective saccades because the VOR cannot keep the target on the fovea.

The syndrome is usually maximal over hours, remains continuous for days, and then improves through peripheral recovery and central compensation. Symptoms worsen with movement, but movement does not create discrete attacks. Hearing should be preserved. New unilateral hearing loss changes the problem: it may indicate labyrinthine involvement, sudden sensorineural hearing loss, or AICA territory ischemia and must not be casually folded into “neuritis.”

A practical management sequence

1. Establish that the patient truly has acute vestibular syndrome and document hearing, ocular motor findings, cranial nerves, limb coordination, stance, and gait.
2. Address stroke using a trained bedside strategy when applicable and appropriate imaging or stroke evaluation when the examination is central, equivocal, incomplete, or performed by an untrained examiner.
3. Use antiemetics and vestibular suppressants only long enough to permit hydration, sleep, and mobilization. Prolonged use can slow compensation.
4. Discuss corticosteroids as an optional early treatment with uncertain patient-centered benefit. Evidence suggests possible short-term improvement in peripheral test recovery, but durable symptomatic benefit is not established.
5. Begin graded mobility and vestibular rehabilitation early once vomiting and medical instability permit.

Labyrinthitis and sudden hearing loss

The word labyrinthitis is often used imprecisely for any acute vertigo. A more useful clinical meaning is an acute peripheral vestibular syndrome accompanied by cochlear dysfunction. That combination

deserves urgent audiometry and attention to sudden sensorineural hearing loss pathways. It also raises the possibility of vascular inner-ear injury, especially when hearing loss is abrupt and the patient has vascular risk or other subtle central signs. The inner ear is supplied by an end artery; simultaneous cochlear and vestibular loss can be ischemic even when the initial MRI is unrevealing.

Posterior circulation stroke

Stroke can imitate every component of peripheral acute vestibular loss. Central clues include a normal head impulse in a patient with strong spontaneous nystagmus, direction-changing gaze-evoked nystagmus, skew deviation, vertical or pure torsional nystagmus, impaired smooth pursuit or saccades, severe truncal ataxia, new hearing loss, and other neurologic findings. None of these should be interpreted in isolation. HINTS is a targeted examination for a specific patient population - continuous AVS with spontaneous nystagmus - and its performance depends on training. It is not a screening acronym for all dizziness.

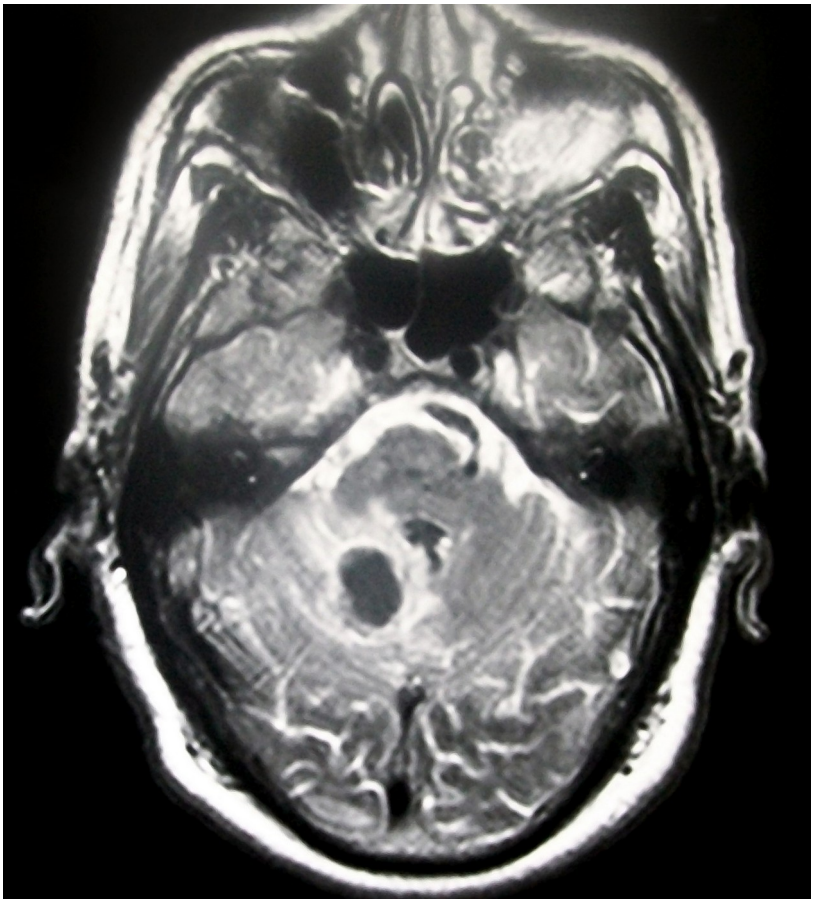


Figure 6. Cerebellar hemorrhage as an example of a central destructive lesion producing acute imbalance and vestibular signs. Bobjgalindo via Wikimedia Commons, CC BY-SA 4.0.

Imaging caveat

A negative early MRI does not erase a central bedside phenotype. Conversely, an abnormal head impulse does not guarantee a peripheral lesion because AICA and selected brainstem strokes can injure vestibular pathways. Reconcile the entire syndrome and repeat evaluation when the course is discordant.

Ramsay Hunt syndrome and cranial polyneuropathy

Herpes zoster oticus can involve the facial nerve and adjacent cranial nerves. Severe otalgia, auricular or canal vesicles, lower motor neuron facial weakness, hearing loss, tinnitus, and vertigo may occur in varying combinations. Vesicles can appear after weakness, and zoster sine herpete can occur. The acute priorities are eye protection for incomplete closure, prompt combined antiviral and corticosteroid treatment when clinically appropriate, pain control, audiovestibular assessment, and recognition of broader cranial neuropathy or central complications.

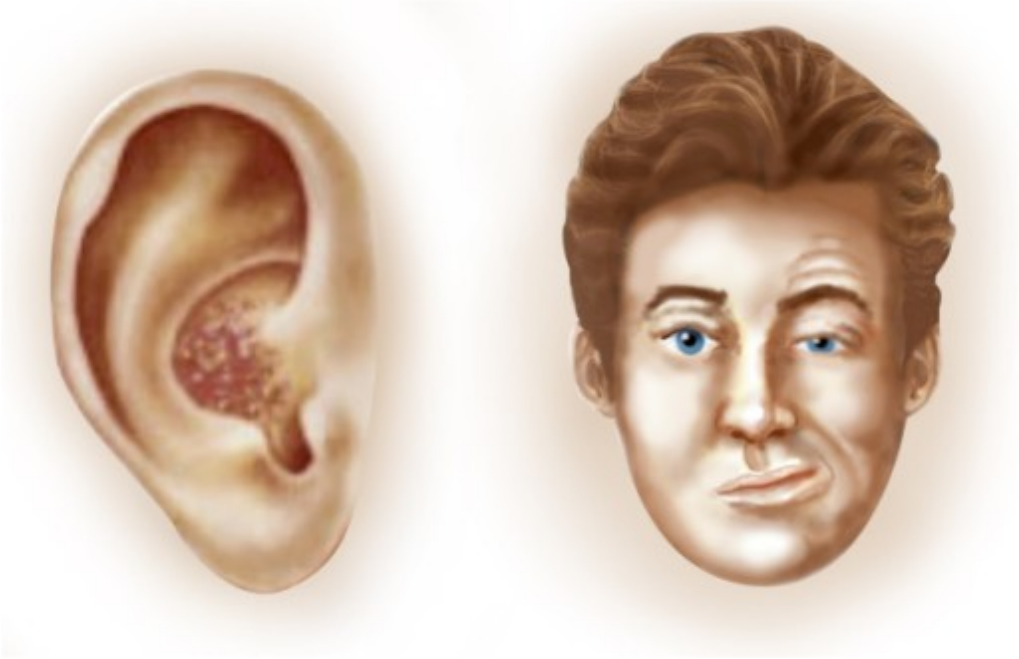


Figure 7. Ramsay Hunt syndrome with facial weakness and zoster oticus. Worme et al. via Wikimedia Commons, CC BY 2.0.

Feature	AUVP / neuritis	Labyrinthine or cochleovestibular injury	Posterior circulation stroke
Time course	Continuous days	Continuous days	Continuous days
Hearing	Preserved by definition	Acute SNHL may be present	May be preserved or acutely reduced, especially AICA
Nystagmus	Usually unidirectional, mixed horizontal-torsional, fixation suppressed	Peripheral pattern possible	Direction-changing, vertical/torsional, or even peripheral-appearing
Head impulse	Abnormal toward lesion	Often abnormal toward lesion	Often normal in PICA/cerebellar stroke; may be abnormal in AICA/vestibular pathway lesions
Skew / central ocular signs	Absent	Absent unless second lesion	May be present
Gait	Unsteady but often able to stand with support	Variable	Severe truncal instability can dominate
Immediate priority	Exclude stroke, symptom control, early rehab	Urgent hearing assessment and stroke consideration	Stroke pathway and vascular imaging as indicated

Table 4. Acute vestibular syndrome differential.

ATTACK DURATION IS A BIOMARKER

4. Spontaneous episodic vestibular syndrome

When the patient is normal or near baseline between spontaneous attacks, the current examination may reveal little. Diagnosis becomes an exercise in reconstructing the attack. A useful history sounds almost like a video: how the attack begins, what the patient can and cannot do, whether hearing changes in one ear, whether light or sound becomes intolerable, whether headache biology appears, and how long untreated symptoms persist.

Ménière disease

Ménière disease is an episodic cochleovestibular syndrome. Definite 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 the affected ear, fluctuating aural symptoms in that ear, and no better explanation. Probable disease allows attacks up to 24 hours and does not require audiometrically documented hearing loss. The criteria intentionally privilege a coherent phenotype rather than a single test result.

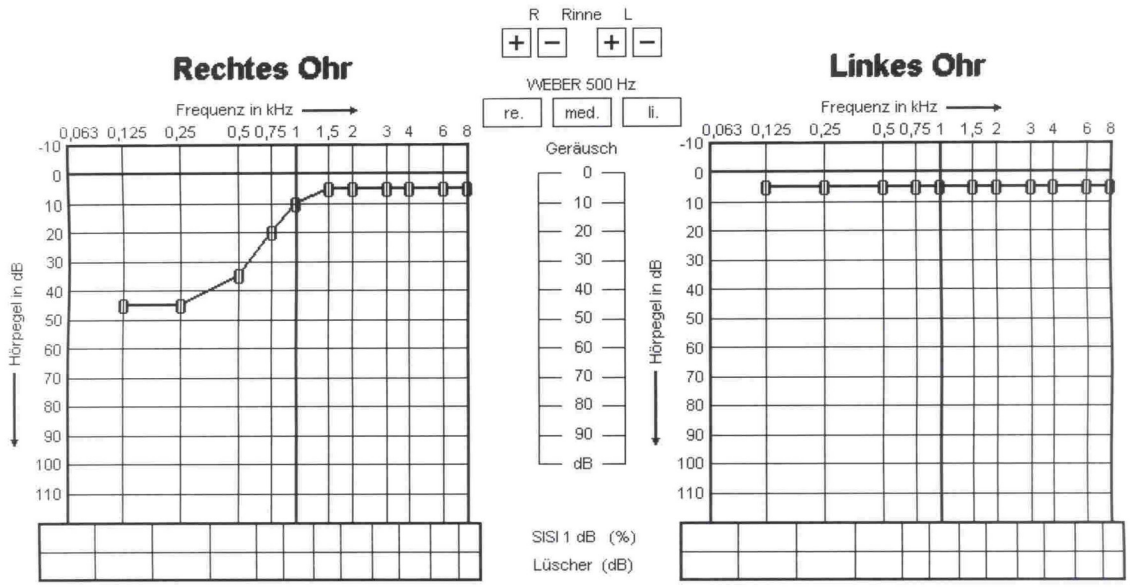


Figure 8. Example of low-frequency hearing loss, a classic early audiometric pattern in Ménière disease. Klaus D. Peter via Wikimedia Commons, CC BY 3.0 DE.

Early in the disease, hearing may fluctuate and recover between attacks. Over time, hearing loss can become less reversible and involve more frequencies. Tinnitus and fullness often fluctuate with the affected ear, but neither is specific. Bilateral symptoms, migraine features, autoimmune disease, and retrocochlear asymmetry should be considered rather than forced into a unilateral Ménière model.

Management as a ladder

Management separates attack treatment from prevention and from rehabilitation of chronic deficits. A limited vestibular suppressant or antiemetic can be used during attacks, but chronic use does not

prevent disease and may impair compensation. Education, safety planning, and individualized dietary or lifestyle modification form the foundation. A diuretic and/or betahistine may be offered for maintenance, acknowledging variable evidence and practice patterns. Persistent active disease can be escalated to intratympanic steroid, then intratympanic gentamicin when nonablative therapy fails. Destructive surgery is reserved for refractory disease and selected hearing status. Vestibular rehabilitation is useful for chronic imbalance but not as a treatment for an active vertigo attack.

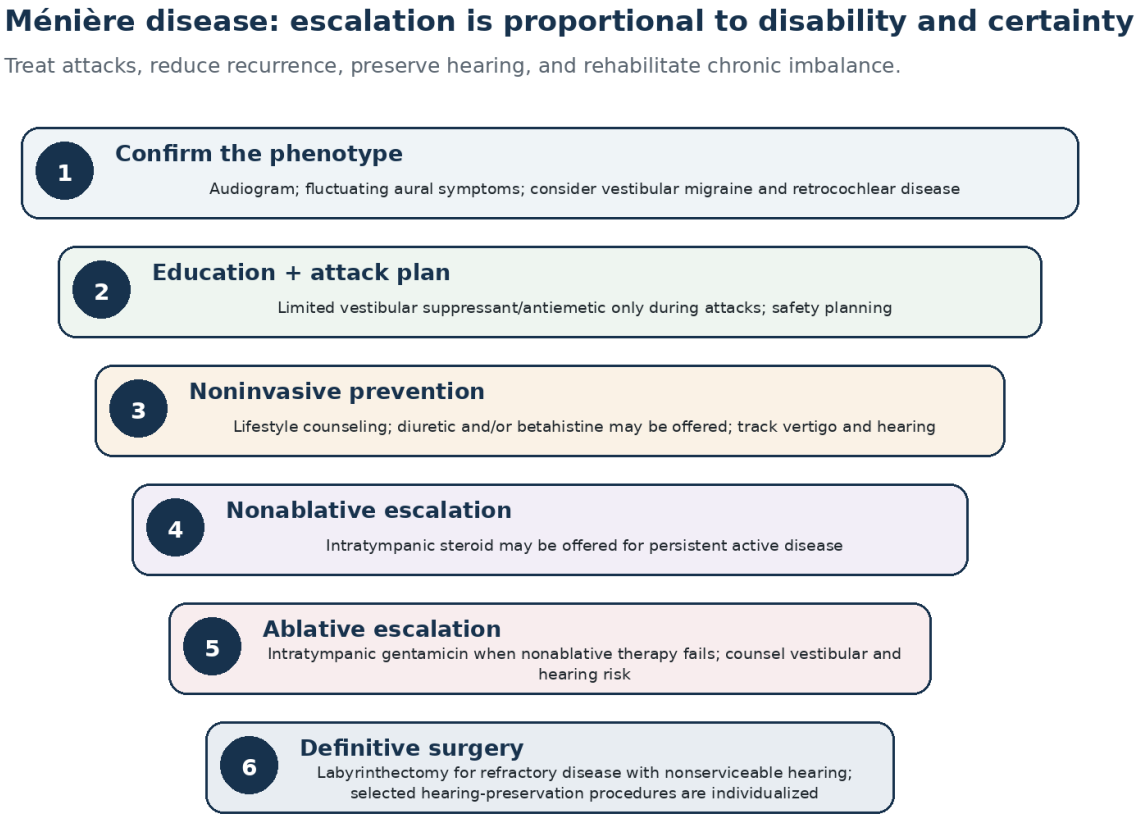


Figure 9. A mechanism-conscious escalation ladder for Ménière disease, adapted to AAO-HNS guideline principles. Original synthesis.

What not to order reflexively

Routine vestibular testing or electrocochleography is not required to establish a classic Ménière diagnosis. Obtain audiometry. MRI of the internal auditory canals/posterior fossa may be offered when asymmetric sensorineural hearing loss raises a retrocochlear question.

Vestibular migraine

Vestibular migraine is a clinical diagnosis that links recurrent vestibular episodes to migraine biology. Definite vestibular migraine requires at least five episodes of moderate or severe vestibular symptoms lasting 5 minutes to 72 hours, a current or previous history of migraine, migraine features during at least half of the vestibular episodes, and no better diagnosis. The qualifying migraine features are migraine-type headache, photophobia and phonophobia, or visual aura. Headache need not accompany every attack and may be absent in the dominant vestibular phenotype.

The disorder can generate spontaneous vertigo, positional vertigo, head-motion induced dizziness, visual-motion sensitivity, or a prolonged postdrome. Audiometry is usually normal or does not display the stereotyped unilateral fluctuating low-frequency pattern expected in Ménière disease. Patients can have both disorders, and migraine is common enough that its presence alone should not erase objective unilateral cochlear findings.

Treatment borrows from migraine care because vestibular-specific trials remain limited. Begin with regular sleep, hydration, meals, exercise, management of medication overuse, and identification of reproducible rather than mythical food triggers. Preventive choices are individualized to comorbidity and reproductive considerations; commonly used options include beta-blockers, topiramate, venlafaxine, tricyclic agents, calcium-channel blockers, and increasingly migraine-specific therapies in selected refractory patients. Evidence for aborting the vertigo component with triptans is weak. Vestibular rehabilitation can help persistent motion sensitivity or visual dependence but should be graded to avoid an unproductive boom-and-bust cycle.

Posterior circulation TIA

Transient ischemia is the diagnosis that must not be missed in spontaneous episodic vestibular syndrome. Abrupt attacks lasting minutes, especially with diplopia, dysarthria, unilateral weakness or numbness, severe gait instability, new headache or neck pain, or recent similar attacks, require urgent vascular evaluation. Isolated vertigo can be ischemic. A normal examination after the attack does not exclude it, because the pathologic signal has ended. The clinical goal is not to prove that every brief attack is a TIA, but to recognize when the consequence of delay is unacceptable.

Vestibular paroxysmia

Vestibular paroxysmia produces very brief, frequent, stereotyped attacks attributed to pathologic excitation of the eighth nerve, often in the setting of neurovascular contact. Definite criteria require at least ten spontaneous attacks lasting less than one minute, a stereotyped phenotype, response to carbamazepine or oxcarbazepine, and no better explanation. Probable disease allows at least five attacks lasting less than five minutes and may include head-movement provocation. Neurovascular contact on MRI is supportive but common in asymptomatic people; the clinical pattern and treatment response remain central.

Feature	Ménière disease	Vestibular migraine	Posterior circulation TIA	Vestibular paroxysmia
Typical duration	20 min-12 h definite; up to 24 h probable	5 min-72 h	Usually minutes; variable	Seconds to <1 min definite
Hearing	Fluctuating unilateral low/mid-frequency SNHL	Usually no fixed unilateral cochlear pattern	May have sudden hearing loss in AICA ischemia	Possible unilateral tinnitus but not defining
Migraine biology	May coexist	Required history and/or attack features per criteria	Not defining	Not defining
Key discriminator	Documented cochlear phenotype	Migraine features in at least half of episodes	Vascular context or focal neurologic features	Very brief stereotyped attacks and medication response

Table 5. Spontaneous episodic vestibular syndromes.

LOSS, COMPENSATION, OR MALADAPTIVE PREDICTION?

5. Chronic vestibular syndromes

Chronic dizziness is not a single syndrome. The resident should decide whether the dominant problem is persistent peripheral sensory loss, inadequate central compensation, multisystem degeneration, or an altered perceptual-control strategy. The examination should deliberately stress the systems that can compensate for one another: vision, somatosensation, vestibular input, and motor prediction.

Incomplete compensation after unilateral loss

After acute unilateral loss, static symptoms usually improve as the brain rebalances tonic activity. Dynamic deficits can persist: reduced VOR gain during rapid head turns, motion-provoked dizziness, visual blurring, and a tendency to avoid movement. A patient who remains on suppressants, minimizes head motion, sleeps poorly, or develops migraine or PPPD may plateau. The treatment is not to suppress the residual error signal indefinitely; it is to expose the nervous system to a tolerable error that drives adaptation and restores normal movement behavior.

Bilateral vestibulopathy

Bilateral vestibulopathy is a chronic syndrome of unsteadiness during standing or walking, worsened in darkness or on uneven ground, with oscillopsia or blurred vision during walking or rapid head motion in many patients. Symptoms are minimal while sitting or lying still. The physiologic requirement is bilaterally reduced angular VOR function, demonstrated by low horizontal vHIT gain, markedly reduced caloric responses, or low rotary-chair gain with abnormal phase. The pattern is a direct consequence of losing the fastest head-motion sensor while vision and somatosensation remain available only under favorable conditions.

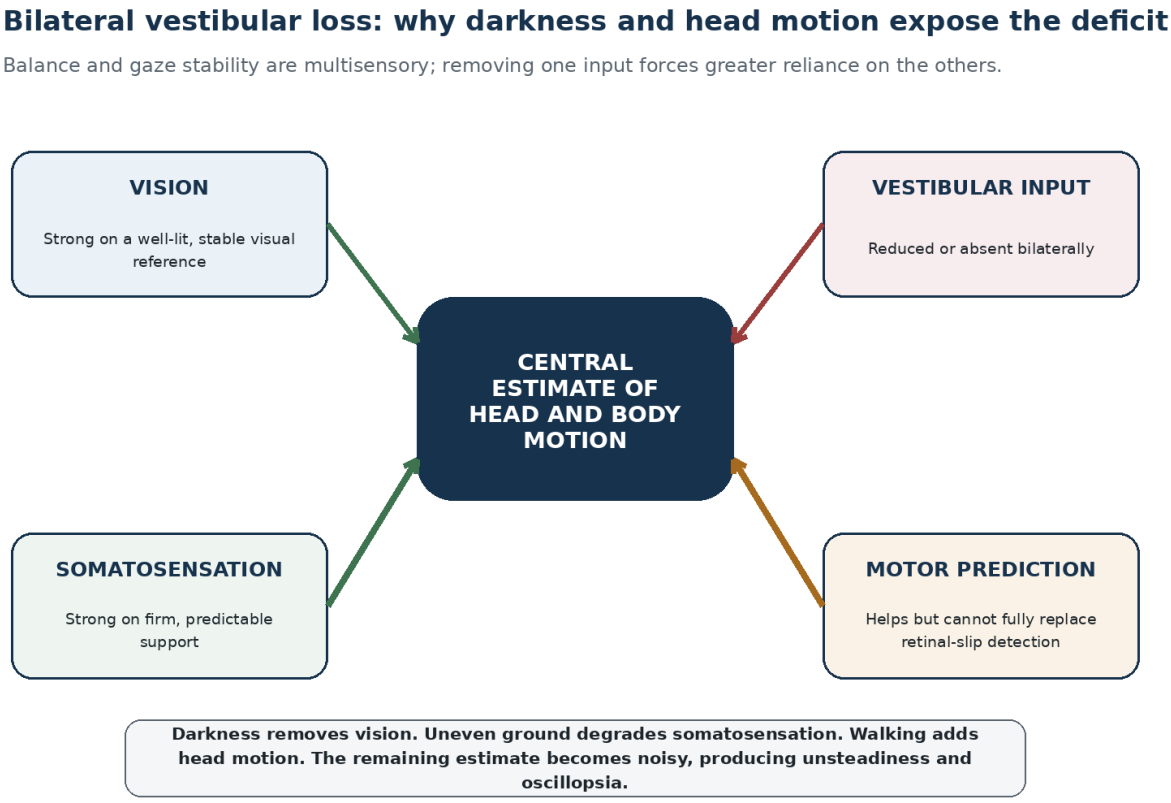


Figure 10. Multisensory compensation in bilateral vestibular loss and why darkness, uneven ground, and walking expose the deficit. Original synthesis.

Causes include aminoglycoside toxicity, bilateral Ménière disease, meningitis, autoimmune inner-ear disease, sequential vestibular neuritis, genetic disease, and neurodegeneration; many cases remain idiopathic. A careful medication and hospitalization history can be more useful than a long laboratory panel. Management emphasizes vestibular rehabilitation with substitution and gaze strategies, strength and balance training, lighting and environmental modification, fall prevention, and treatment of the underlying cause when possible. Chronic vestibular suppressants are particularly counterproductive.

Objective criterion	Bilateral vestibulopathy	Presbyvestibulopathy
Horizontal vHIT gain	<0.6 on both sides	0.6 to <0.8 bilaterally
Caloric response	Sum of bithermal maximal peak slow-phase velocity <6°/s on each side	6 to <25°/s on each side
Rotary chair	Horizontal VOR gain <0.1 at 0.1 Hz with phase lead >68°	Gain 0.1 to <0.3 at 0.1 Hz
Clinical frame	Chronic unsteadiness/oscillopsia; worse in dark or uneven terrain; minimal static symptoms	Age 60 or older with chronic postural imbalance, gait disturbance, dizziness, or falls and mild bilateral hypofunction

Table 6. Bárány Society physiologic thresholds for bilateral vestibulopathy and presbyvestibulopathy.

Presbyvestibulopathy

Presbyvestibulopathy describes mild bilateral vestibular hypofunction in an adult 60 years or older with chronic postural imbalance, gait disturbance, dizziness, or recurrent falls that is not better

explained by another disorder. It is not permission to attribute all imbalance to age. Vision, proprioception, neuropathy, musculoskeletal limitation, medications, cognition, orthostasis, and central disease must still be assessed. The diagnosis is useful because mild vestibular loss can be a modifiable contributor to a multifactorial fall syndrome.

CANVAS and RFC1 spectrum disease

CANVAS combines cerebellar ataxia, sensory neuronopathy, and bilateral vestibular areflexia. Many patients have a chronic spasmodic cough years before the neurologic syndrome becomes obvious. Biallelic intronic AAGGG repeat expansions in RFC1 are a common cause and can produce a broader spectrum ranging from isolated sensory neuropathy to the full triad. Bedside clues include bilaterally abnormal head impulses, impaired visually enhanced VOR, sensory ataxia, absent sensory nerve action potentials, cerebellar ocular motor signs, and disproportionate worsening when visual cues are removed. The multisystem nature explains why standard vestibular rehabilitation helps but cannot normalize gait.

Persistent postural-perceptual dizziness

PPPD is a chronic functional vestibular disorder, not malingering and not simply “anxiety.” It is defined by nonspinning dizziness, unsteadiness, or both on most days for at least three months, lasting for prolonged periods and exacerbated by upright posture, active or passive motion, and moving or visually complex stimuli. It is precipitated by a vestibular, neurologic, medical, or psychological event, causes distress or functional impairment, and is not better explained by another disorder.

The precipitating event may have resolved. What persists is an altered control strategy: increased vigilance to motion, visual dependence, stiffened postural control, avoidance, and a prediction that movement is unsafe. That model explains why additional normal scans do not cure the disorder and why indefinite vestibular suppressants often worsen function. The diagnosis can coexist with residual vestibular loss or migraine; it should not erase objective pathology.

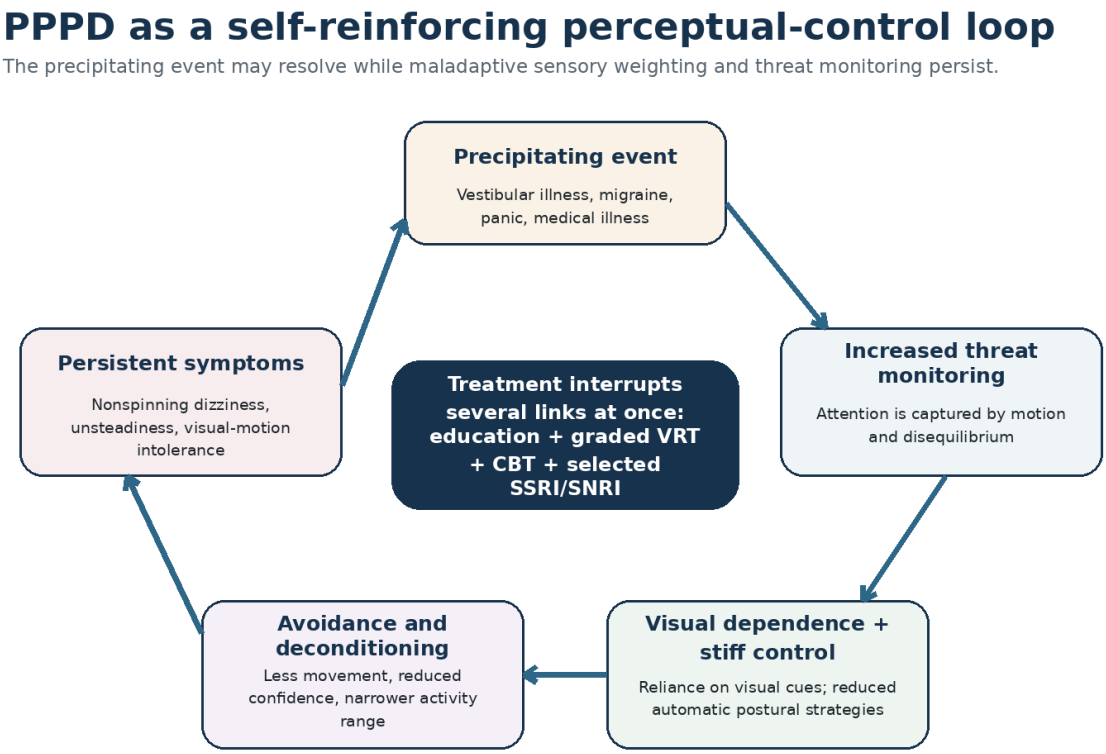


Figure 11. A self-reinforcing PPPD loop and the multiple treatment targets that interrupt it. Original synthesis.

Treatment is multimodal. Education provides a noncatastrophic explanation. Vestibular rehabilitation uses graded motion and visual exposure rather than aggressive symptom flooding. Cognitive behavioral therapy addresses threat interpretation and avoidance. An SSRI or SNRI is commonly used when symptoms are persistent or comorbid anxiety/depression is clinically important, although no medication is specifically approved for PPPD and the trial evidence remains heterogeneous. Improvement is usually measured in restored activity and reduced symptom dominance, not instantaneous absence of dizziness.

Mal de débarquement syndrome

MdDS is characterized by continuous or near-continuous rocking, bobbing, or swaying that begins within 48 hours after passive motion such as a cruise, flight, or prolonged vehicle travel. A striking clue is temporary improvement during re-exposure to passive motion, such as riding in a car. Symptoms lasting more than 48 hours meet the syndrome threshold; persistent MdDS lasts more than one month. Standard vestibular tests and imaging are often normal and are used to address alternatives rather than confirm the disorder. Treatment evidence is limited; education, sleep and migraine management, symptom-targeted rehabilitation, and specialized optokinetic protocols are used in selected centers.

Chronic pattern	Defining clue	Expected testing	Treatment emphasis
Incomplete unilateral compensation	Head motion/visual motion remains provocative after a prior acute loss	Residual unilateral VOR deficit may persist	Early movement, adaptation/habituation, stop chronic suppressants
Bilateral vestibulopathy	Worse in dark/uneven ground; oscillopsia with walking	Bilateral low VOR across one or more frequency tests	Substitution, gaze stability, fall prevention
Presbyvestibulopathy	Older adult with mild bilateral loss and multifactorial imbalance	Mild bilateral reduction	Multifactorial fall strategy plus vestibular rehab
CANVAS/RFC1 spectrum	Sensory neuronopathy + cerebellar signs + bilateral areflexia; chronic cough	Bilateral vestibular loss, abnormal sensory studies, cerebellar findings	Rehab and safety across all impaired systems; genetic evaluation
PPPD	Most-day symptoms worsened upright, moving, or in complex visual scenes	May be normal or show a coexisting precipitating lesion	Education, graded VRT, CBT, selected serotonergic medication
MdDS	Rocking/swaying after passive motion, improved by riding in a vehicle	Usually no diagnostic vestibular test	Education, trigger management, specialized therapy in selected patients

Table 7. Chronic vestibular syndrome patterns.

A THIRD WINDOW CHANGES THE PHYSICS

6. Sound-, pressure-, and exertion-induced syndromes

The cochlea and vestibular labyrinth normally behave as a fluid system with two compliant windows: the oval and round windows. A dehiscence or abnormal opening creates another pressure-release pathway. Acoustic and pressure energy can then be shunted through vestibular structures, producing both auditory and vestibular manifestations. The unifying concept is not merely a hole on CT; it is abnormal energy transmission demonstrated clinically and physiologically.

Superior canal dehiscence syndrome

Superior canal dehiscence syndrome requires three domains: at least one symptom consistent with a third mobile window, at least one physiologic sign or test demonstrating abnormal pressure transmission, and high-resolution CT showing dehiscence in the expected plane. Symptoms include bone-conduction hyperacusis or autophony, sound-induced vertigo or oscillopsia time-locked to the stimulus, pressure-induced vertigo or oscillopsia, and pulsatile tinnitus. Physiologic evidence includes eye movements in the superior canal plane during sound or pressure, low-frequency negative bone-conduction thresholds, or enhanced VEMP responses with low cVEMP thresholds or high oVEMP amplitudes.

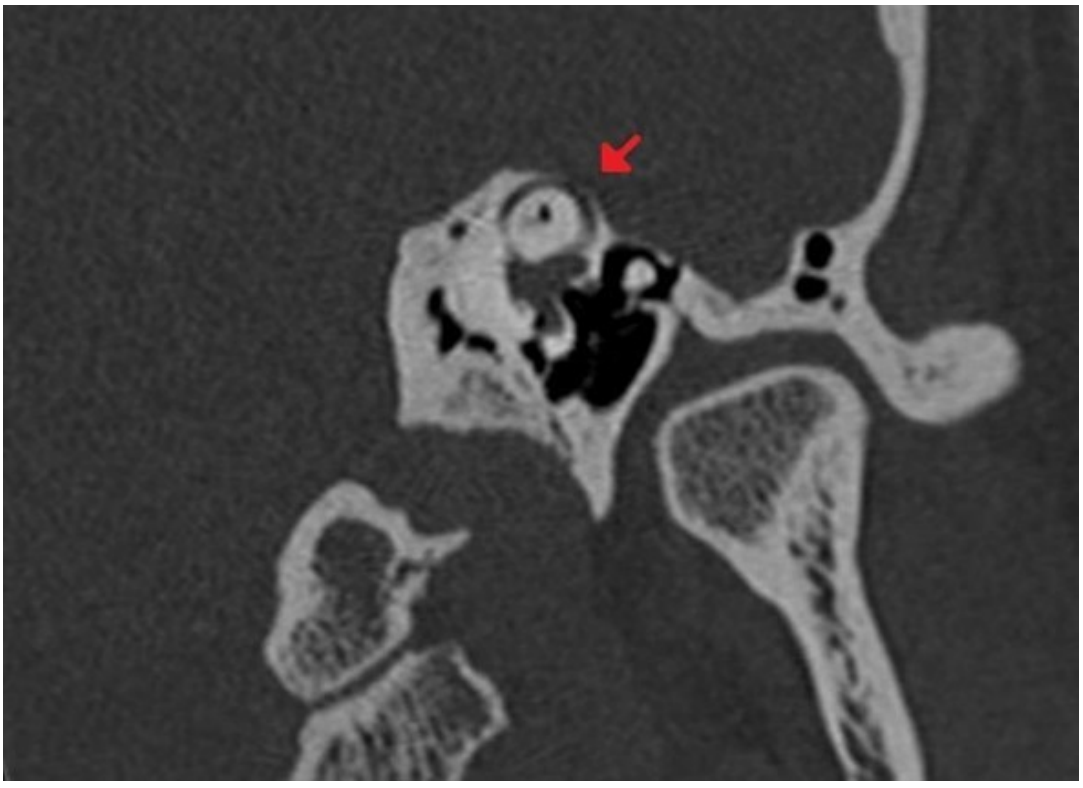


Figure 12. CT demonstration of superior semicircular canal dehiscence. Imaging is necessary but not sufficient for the syndrome. Han-Kuang Chen via Wikimedia Commons, CC BY 4.0.

Thin bone and radiographic dehiscence are common enough that CT alone overdiagnoses the syndrome. Scan technique matters: high-resolution temporal bone acquisition with reconstructions aligned to the superior canal reduces partial-volume artifact. Treatment is observation and counseling when symptoms are mild. Surgery is considered when disabling symptoms, physiologic

evidence, and imaging are concordant. Plugging, resurfacing, or combined techniques can be approached through the middle cranial fossa or transmastoid route depending on anatomy and surgeon judgment. The treatment target is the abnormal window, not the audiogram alone.

Perilymphatic fistula

Perilymphatic fistula remains a challenging and sometimes controversial diagnosis. A leak or abnormal communication is most plausible after penetrating or blunt trauma, barotrauma, temporal bone or stapes surgery, or a clear pressure event followed by acute cochleovestibular symptoms. Pressure- or sound-induced symptoms can occur, but bedside fistula tests are neither sensitive nor specific. Management ranges from activity modification and avoidance of pressure strain to blood patch or surgical exploration in selected, high-suspicion cases. The resident should avoid using "fistula" as a default explanation for nonspecific chronic dizziness.

Enlarged vestibular aqueduct and other third-window states

An enlarged vestibular aqueduct is most often encountered in children or young adults with congenital, fluctuating, or progressive sensorineural hearing loss. Vestibular symptoms may occur and can be precipitated by trauma or pressure change, but the dominant clinical problem is often hearing. Other structural third-window states include posterior canal dehiscence and defects adjacent to the cochlea. The same diagnostic discipline applies: symptom, physiology, and imaging must agree.

Disorder	Typical symptom clue	Audiologic/physiologic clue	Imaging clue	Management principle
SCDS	Autophony, sound/pressure-induced vertigo, pulsatile tinnitus	Low-frequency air-bone gap with normal middle ear; enhanced VEMP; canal-plane eye movement	High-resolution CT dehiscence in canal plane	Operate only for disabling, concordant syndrome
Perilymphatic fistula	Acute symptoms after trauma, barotrauma, or otologic surgery	Variable hearing/vestibular findings; fistula testing limited	May be normal; pneumolabyrinth is highly suggestive when present	Conservative or selective repair based on mechanism and severity
Enlarged vestibular aqueduct	Fluctuating/progressive hearing loss, sometimes after minor trauma	SNHL, sometimes mixed-appearing low-frequency component	Enlarged aqueduct/endolymphatic duct and sac	Hearing rehabilitation, counseling, genetics when appropriate

Table 8. Third-window and fistula syndromes.

ASYMMETRY AND BILATERAL LOSS HAVE CAUSES

7. Progressive, structural, and toxic vestibular disease

Vestibular schwannoma

Vestibular schwannoma is a benign nerve-sheath tumor, usually arising from the vestibular division of the eighth nerve within the internal auditory canal. The classic presentation is asymmetric sensorineural hearing loss, unilateral tinnitus, and chronic disequilibrium rather than dramatic recurrent vertigo. Slow growth allows central compensation, so vestibular symptoms can be mild despite substantial unilateral dysfunction. Larger tumors may produce facial or trigeminal symptoms, cerebellar signs, or hydrocephalus.

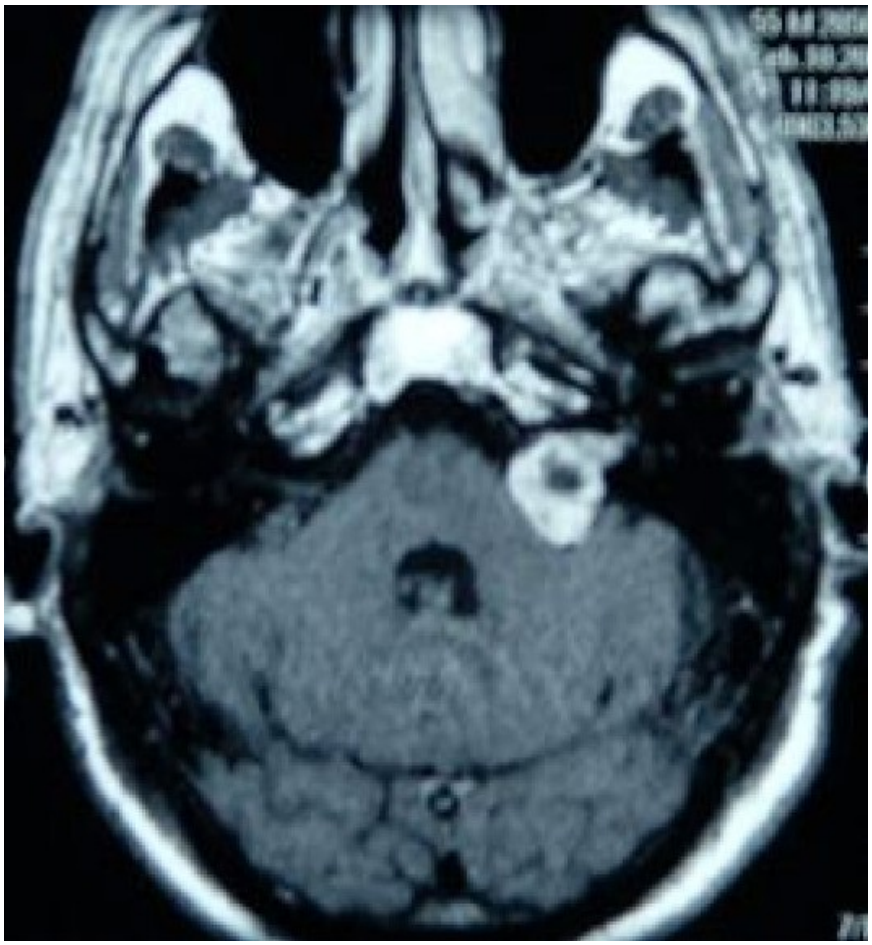


Figure 13. Contrast-enhanced MRI demonstrating a vestibular schwannoma in the internal auditory canal/cerebellopontine angle. Wikimedia Commons, CC BY 2.0.

MRI of the internal auditory canals is the definitive diagnostic study when asymmetric sensorineural hearing loss or other retrocochlear features warrant evaluation. Management is individualized among observation with serial imaging, stereotactic radiosurgery, and microsurgical resection. Age, tumor size and growth, hearing status, symptoms, patient goals, and institutional expertise matter. Surgical approach follows the functional goal: middle cranial fossa and retrosigmoid approaches may be used when hearing preservation is intended in selected tumors, whereas translabyrinthine surgery sacrifices residual hearing but offers direct IAC access without cerebellar retraction.

Ototoxic bilateral vestibular loss

Aminoglycosides are the prototypical vestibulotoxins. Gentamicin and streptomycin can injure vestibular hair cells bilaterally, producing oscillopsia and gait unsteadiness that become evident after the acute illness has resolved. Patients may not report spinning because symmetric bilateral loss creates little resting tone imbalance. Renal dysfunction, cumulative exposure, critical illness, and concurrent ototoxins increase risk. The diagnosis is often delayed because the symptoms are attributed to deconditioning.

Management begins with prevention: use the least toxic effective agent, monitor renal function and drug levels when relevant, and ask specifically about visual blurring while walking and instability in darkness. Once established, loss is often permanent. Stop further exposure when medically feasible and begin vestibular rehabilitation. Cochlear toxicity and vestibular toxicity do not perfectly track; a patient can have severe bilateral vestibular loss with relatively preserved hearing.

Agent/class	Dominant concern	Resident clue
Gentamicin / streptomycin	Vestibular toxicity; cochlear toxicity also possible	No vertigo required - look for oscillopsia and dark/uneven-ground imbalance
Amikacin / kanamycin / neomycin	More prominent cochlear toxicity, though vestibular injury can occur	Audiometric monitoring does not fully replace vestibular symptom screening
Cisplatin	Dose-related bilateral high-frequency cochlear loss	Vertigo is not the dominant expected toxicity
Loop diuretics	Often reversible cochlear toxicity, potentiated by other agents	Consider critical illness and combination exposure
Intratympanic gentamicin	Intentional vestibular ablation for refractory Ménière disease	Titrate to vertigo control while counseling hearing and imbalance risk

Table 9. Selected ototoxic patterns.

Autoimmune inner-ear disease

Autoimmune inner-ear disease is an uncommon clinical syndrome of rapidly progressive, fluctuating, often bilateral sensorineural hearing loss over weeks to months, sometimes with vestibular symptoms. There is no single diagnostic blood test. Systemic autoimmune features may be present, but many cases are isolated. The differential includes bilateral Ménière disease, ototoxicity, genetic disease, infection, and retrocochlear pathology. Treatment is typically a time-limited corticosteroid trial with objective audiometric follow-up, followed by rheumatologic or immunologic collaboration when steroid-responsive or systemic disease is suspected. Avoid treating a vague dizziness syndrome with prolonged immunosuppression in the absence of a convincing hearing phenotype.

> **Structural disease pearl**
>
> Progressive asymmetric hearing loss and chronic disequilibrium localize differently from recurrent spinning attacks. A slowly growing unilateral lesion may be well compensated vestibularly; the audiogram, speech discrimination pattern, and MRI question often lead the diagnosis.

TREAT THE PHYSIOLOGY AND THE BEHAVIOR

8. Treatment principles across vestibular diagnoses

Vestibular suppressants: useful briefly, harmful indefinitely

Antihistamines, anticholinergics, benzodiazepines, and selected antiemetics can make an acute vestibular crisis tolerable. They reduce nausea, motion sensitivity, or central vestibular responsiveness. Their value is greatest during a short, severe attack or the first phase of continuous vertigo. The same mechanism becomes a liability when used every day: sedation increases falls, vestibular error signals are blunted, and the patient learns that movement requires medication. A prescription should therefore have a purpose, an expected duration, and a stop plan.

Clinical situation	Reasonable role	Common mistake
Severe acute AVS with vomiting	Short course to permit hydration, sleep, and mobilization	Continuing for weeks after vomiting stops
Ménière attack	Limited use during the attack	Using daily suppressants as maintenance prevention
BPPV	Occasional rescue only when vomiting prevents maneuver	Treating instead of repositioning
Bilateral vestibulopathy	Generally avoid	Worsening already limited vestibular function and falls
PPPD	Generally avoid chronic use	Reinforcing avoidance and preventing graded adaptation

Table 10. Mechanism-based use of vestibular suppressants.

Vestibular rehabilitation

Vestibular rehabilitation is an active recalibration program, not generic balance therapy. Adaptation exercises use retinal slip during head movement to adjust VOR gain. Substitution teaches predictive saccades and greater use of vision and somatosensation when vestibular input cannot recover. Habituation and graded exposure reduce motion-provoked symptoms and visual dependence. Balance and gait training integrate these mechanisms into real-world movement. The exercise must be difficult enough to create a training signal but not so provocative that the patient cannot repeat it consistently.

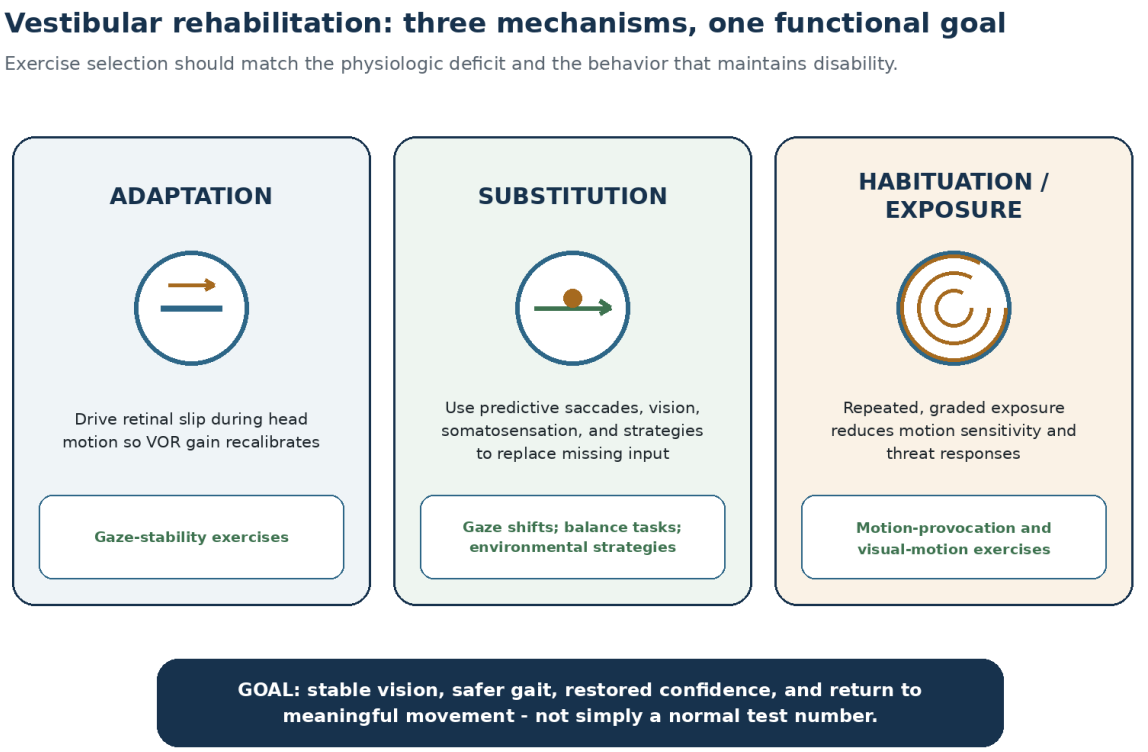


Figure 14. Adaptation, substitution, and habituation/exposure are distinct rehabilitation mechanisms that converge on functional recovery. Original synthesis.

Peripheral unilateral and bilateral hypofunction have strong evidence for supervised vestibular physical therapy. Rehabilitation is also useful for chronic imbalance after Ménière treatment, persistent symptoms after neuritis, and selected patients with vestibular migraine or PPPD. It is not a substitute for canalith repositioning in active BPPV or for treating an unstable central lesion. Improvement should be tracked with functional outcomes: dynamic visual acuity, gait, falls, confidence, activity tolerance, and return to work or sport.

Fall prevention and return to activity

Falls are not an accessory outcome. Ask about near-falls, nighttime bathroom trips, stairs, uneven ground, vision, footwear, sedatives, neuropathy, and orthostasis. Recommend lighting, handrails, removal of trip hazards, an assistive device when needed, and strength training. Driving and occupational decisions should be individualized to attack unpredictability, oscillopsia, sedating medications, and ability to make rapid head movements safely. Patients should not be told simply to “avoid motion”; avoidance preserves disability.

When procedures are appropriate

Procedural treatment is strongest when anatomy and symptoms are concordant and conservative therapy has a defined failure. Repositioning treats BPPV because particles are in the wrong compartment. Intratympanic therapy or labyrinthectomy treats refractory Ménière disease by reducing unstable vestibular output. Canal plugging treats a symptomatic third window. Tumor surgery or radiosurgery treats a structural lesion. The more destructive the procedure, the more

explicit the discussion should be about residual hearing, expected vestibular loss, central compensation, contralateral function, and rehabilitation.

Treatment hierarchy

1) Correct the mechanical problem when one exists. 2) Stop or treat the injuring process. 3) Suppress symptoms only briefly. 4) Drive compensation and restore movement. 5) Escalate to ablation or surgery only when the phenotype, anatomy, and goals agree.

WORK THE SYNDROME, THEN COMMIT

9. Integrated clinical encounters

Case 1. “Every time I roll right, the room spins”

A 67-year-old patient reports 15-second attacks when rolling right in bed and is normal while sitting still. Right Dix-Hallpike produces a brief latency followed by upbeat right-torsional nystagmus that crescendos and resolves. This is triggered episodic vestibular syndrome with a right posterior canal pattern. The mechanism is canalithiasis; the treatment is a right posterior canal repositioning maneuver. MRI and chronic meclizine add no value unless another feature contradicts the pattern.

Case 2. “I woke up spinning and cannot walk”

A 54-year-old patient has 18 hours of continuous vertigo, vomiting, spontaneous left-beating nystagmus, and marked gait instability. Hearing is unchanged. A rightward head impulse produces a corrective saccade, there is no skew, and nystagmus remains left-beating in right and left gaze. In a trained examiner, the pattern supports right acute unilateral vestibulopathy. The resident still documents neurologic findings and gait, considers imaging based on the full context, limits suppressants, and mobilizes early. If the head impulse were normal, nystagmus changed direction, skew were present, or the patient could not sit unsupported, the central pathway would dominate.

Case 3. “My ear fills, then I spin for two hours”

A 42-year-old patient has recurrent 1- to 3-hour attacks with left aural fullness, roaring tinnitus, and fluctuating hearing. Audiometry documents left low-frequency sensorineural hearing loss. This is a spontaneous episodic cochleovestibular syndrome meeting definite Ménière criteria. The plan separates rescue medication for attacks, education and maintenance prevention, interval audiometry, and escalation only if disabling active vertigo persists. A migraine history is assessed because coexistence is common, but it does not negate the objective cochlear pattern.

Case 4. “The grocery store makes me feel like I am floating”

A 35-year-old patient had vestibular neuritis six months ago. The spontaneous nystagmus resolved and vHIT improved, but the patient now has most-day nonspinning dizziness that worsens when standing, walking, scrolling, and shopping in patterned aisles. The patient avoids exercise and uses meclizine daily. The current syndrome fits PPPD precipitated by a peripheral event, possibly with residual motion sensitivity. Treatment is explanation, withdrawal of chronic suppressants, graded vestibular and visual-motion exposure, and consideration of CBT and serotonergic medication. Repeating MRI for reassurance does not target the maintaining mechanism.

Case 5. “I hear my eyes move”

A 46-year-old patient reports autophony, audible eye movements, pulsatile tinnitus, and brief vertigo with loud sounds. Audiometry shows a low-frequency air-bone gap despite normal tympanometry and acoustic reflexes. oVEMP amplitudes are enhanced, and high-resolution CT shows left superior

canal dehiscence in the Pöschl plane. The symptom, physiology, and anatomy are concordant. The CT alone would not establish SCDS; the triad does.

Case 6. “I cannot see signs while I walk”

A 70-year-old patient treated with prolonged gentamicin during sepsis describes bouncing vision while walking and severe instability in darkness without spinning attacks. Bedside head impulses are abnormal bilaterally and dynamic visual acuity drops substantially. vHIT demonstrates bilateral low gains with corrective saccades. This is bilateral vestibulopathy, likely aminoglycoside-related. The priorities are no further vestibulotoxic exposure, vestibular rehabilitation emphasizing substitution and gaze strategies, and aggressive fall prevention.

Case	Syndrome	Localizing feature	Diagnosis	First treatment move
1	Triggered episodic	Upbeat-torsional positional nystagmus	Posterior canal BPPV	Canalith repositioning
2	Acute continuous	Peripheral HINTS pattern in valid AVS	AUVP after central risk addressed	Short symptom control + early rehab
3	Spontaneous episodic	Fluctuating unilateral cochlear findings	Ménière disease	Attack plan + preventive ladder
4	Chronic perceptual	Upright/motion/visual exacerbation after precipitant	PPPD	Education + graded VRT/CBT
5	Stimulus linked	Third-window symptom + physiology + CT	SCDS	Counsel; surgery if disabling
6	Chronic bilateral loss	Oscillopsia, dark dependence, bilateral low VOR	Bilateral vestibulopathy	Substitution rehab + falls strategy

Table 11. Integrated case summary.

RAPID RETRIEVAL

Appendix A. Diagnostic criteria cards

Definite Ménière disease

- 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, documented before, during, or after an episode.
- Fluctuating aural symptoms - hearing, tinnitus, or fullness - in the affected ear.
- Not better accounted for by another vestibular diagnosis.

Vestibular migraine

- At least five episodes of vestibular symptoms of moderate or severe intensity lasting 5 minutes to 72 hours.
- Current or previous migraine with or without aura.
- Migraine features during at least half of vestibular episodes: migraine-type headache, photophobia and phonophobia, or visual aura.
- Not better accounted for by another vestibular or headache diagnosis.

PPPD

- One or more symptoms of dizziness, unsteadiness, or nonspinning vertigo on most days for at least three months; symptoms last for prolonged periods but may wax and wane.
- Exacerbated by upright posture, active or passive motion, and exposure to moving or complex visual stimuli.
- Precipitated by a condition causing vertigo, unsteadiness, dizziness, or balance disruption.
- Causes significant distress or functional impairment and is not better explained by another disorder.

Bilateral vestibulopathy

- Chronic unsteadiness when standing or walking plus oscillopsia during walking/quick head movement and/or worsening in darkness or uneven terrain.
- No symptoms while sitting or lying under static conditions.
- Bilaterally reduced angular VOR documented by vHIT, calorics, and/or rotary chair using accepted thresholds.
- Not better accounted for by another disease.

Superior canal dehiscence syndrome

- At least one third-window symptom: bone-conduction hyperacusis, sound-induced vertigo/oscillopsia, pressure-induced vertigo/oscillopsia, or pulsatile tinnitus.
- At least one physiologic sign: canal-plane eye movement with sound/pressure, low-frequency negative bone-conduction thresholds, or enhanced VEMP response.
- High-resolution CT demonstrating superior canal dehiscence.

- Not better explained by another disorder.

Vestibular paroxysmia

- At least ten spontaneous attacks of spinning or nonspinning vertigo.
- Duration less than one minute, stereotyped in the individual patient.
- Response to carbamazepine or oxcarbazepine.
- Not better accounted for by another diagnosis.

MATCH THE INTERVENTION TO THE MECHANISM

Appendix B. Maneuver and treatment matrix

Problem	Mechanism	Primary intervention	Avoid
Posterior canal BPPV	Free-floating posterior canal particles	Epley or Semont	Routine imaging, daily meclizine, postural restrictions
Horizontal geotropic BPPV	Horizontal canal canalithiasis	Lempert/barbecue or geotropic Gufoni	Using posterior canal maneuver repeatedly
Horizontal apogeotropic BPPV	Cupular/anterior-arm debris	Subtype-specific Gufoni/Appiani or conversion	Lateralizing from symptom side alone
AUVP	Acute unilateral loss	Brief antiemesis/suppression; early mobility/VRT; selective steroid discussion	Prolonged suppression and bed rest
Ménière disease	Episodic unstable cochleovestibular function	Attack plan, prevention, IT therapy or ablation when refractory	Using VRT for an active attack
Vestibular migraine	Migraine network disorder	Lifestyle regularity, individualized migraine prevention, graded VRT for persistent sensitivity	Diagnosing solely from a migraine history
Bilateral vestibulopathy	Bilateral VOR loss	Substitution/gaze strategies and falls prevention	Vestibular suppressants
PPPD	Maladaptive perceptual/postural strategy	Education, graded VRT, CBT, selected SSRI/SNRI	Endless testing and avoidance
SCDS	Third mobile window	Observation or canal repair for disabling concordant syndrome	Operating on CT alone

Table 12. Mechanism-to-treatment matrix.

HIGH-YIELD DISTINCTIONS

Appendix C. Boards and call-room pearls

- BPPV is a nystagmus diagnosis. “Positional dizziness” is a symptom category.
- In geotropic horizontal canal BPPV, the stronger side is usually affected; in apogeotropic disease, the weaker side is usually affected.
- Pure positional downbeat nystagmus is central until a coherent anterior canal pattern is demonstrated.
- Acute hearing loss is not part of vestibular neuritis. In AVS, it broadens the differential to cochleovestibular injury and AICA ischemia.
- HINTS is valid only in the correct acute vestibular syndrome and when performed by a trained examiner.
- Ménière disease requires a time-limited episodic syndrome and an ipsilateral cochlear phenotype; tinnitus alone is not diagnostic.
- Vestibular migraine can occur without headache during the dominant attacks, but migraine linkage must satisfy criteria.
- Bilateral vestibular loss causes oscillopsia and dark/uneven-ground dependence, often without vertigo.
- PPPD can coexist with organic vestibular disease. It describes the current chronic pattern, not the absence of prior injury.
- MdDS improves temporarily during passive motion; PPPD usually worsens with motion and visually complex environments.
- SCDS requires symptom + physiology + CT. Radiographic dehiscence alone is not the syndrome.
- Slowly progressive vestibular schwannoma commonly causes disequilibrium and hearing asymmetry rather than recurrent spinning attacks.
- Chronic vestibular suppressants increase sedation and can impede compensation. Every prescription needs a stop plan.
- Vestibular rehabilitation is diagnosis-specific: adaptation, substitution, and habituation are different tools.
- After a failed BPPV maneuver, re-observe and re-localize before repeating the same sequence.

EVIDENCE BASE

Appendix D. Key references

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Figures 1, 9, 10, 11, and 14 are original educational syntheses created for this volume.

Educational use

This resident study guide summarizes general diagnostic and management principles. It does not replace local protocols, direct supervision, specialist consultation, or patient-specific clinical judgment. Medication selection, procedural candidacy, stroke evaluation, and operative approach require individualized assessment.