Multiple Sclerosis and the Vestibular System:

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

Vestibular Medicine for Vestibular Physicians

Central Vestibular Pathology — Module 3.6

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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 central vestibular practice, where a working command of mechanism, localisation, 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 and Epidemiology

Multiple sclerosis (MS) is a chronic immune-mediated, inflammatory and neurodegenerative disorder of the central nervous system characterised by demyelination, axonal injury, and disseminated lesions separated in space and time [1,2,3]. For the vestibular physician it occupies a particular place: it is the most important central mimic of peripheral vestibular disease in the young adult, it produces the single most specific oculomotor sign in clinical neuro-otology — bilateral internuclear ophthalmoplegia — and it is one of the few central causes of vertigo in which early recognition meaningfully alters long-term outcome through disease-modifying therapy [2,8,21].

MS typically presents between the ages of 20 and 40 years, with a female-to-male ratio of approximately 3:1 in relapsing disease [2,3]. Global prevalence has risen to roughly 36 per 100,000, with a pronounced latitudinal gradient that places Australia — particularly Tasmania and the southern states — among the higher-prevalence regions worldwide [4,5]. The relapsing–remitting phenotype accounts for around 85% of incident cases; most untreated patients transition to a secondary progressive course over one to two decades, while approximately 10–15% follow a primary progressive course from onset [2,6].

Table 1. Multiple sclerosis and vestibular involvement — epidemiology at a glance.

Measure	Value	Notes
Typical age at onset	20–40 years	Vertigo in a young adult should raise MS [2,3]
Female : male ratio	~3 : 1	Relapsing disease; narrower in progressive forms [2]
Global prevalence	~36 / 100,000	Strong latitude gradient; Australia high [4,5]
Vertigo at some point in MS	~50%	True vertigo in roughly one-third to one-half [7,28]
Vertigo as presenting symptom	~5%	First clinical event in a minority [7,8]
Demyelination as cause of AVS	~4%	Of high-risk acute vestibular syndrome [8]

Vestibular symptoms are common across the disease course. Dizziness or vertigo is reported by around half of all patients at some stage, and true rotational vertigo by a substantial minority [7,28,39]. Vertigo is the presenting symptom in approximately 5% of cases and, when it is, the demyelinating lesion is usually infratentorial [7,8]. In the prospective Pula series of high-risk acute vestibular syndrome, demyelination accounted for 4% of presentations — uncommon, but with disproportionate consequences if missed, because three of those patients were experiencing a first clinically isolated syndrome [8].

The clinically important epidemiological point is that vestibular complaints in MS arise from at least three distinct mechanisms with very different management: a central demyelinating plaque in the brainstem or cerebellum; a coincident peripheral disorder such as benign paroxysmal positional vertigo, which is in fact the commonest single cause of new vertigo in established MS [7]; and the chronic imbalance of accumulated cerebellar and proprioceptive deficits [49]. Comorbidity is the rule rather than the exception in MS, and the vestibular physician is well placed to disentangle treatable peripheral contributions from the underlying central disease [27].

The burden of MS in Australia is substantial and growing. National prevalence has risen over successive surveys, consistent with the worldwide trend captured in the third edition of the Atlas of MS, and the steep latitudinal gradient means that southern states carry a disproportionate share of cases [4,5]. For a clinic such as a dedicated vestibular service, this translates into a steady stream of young adults in whom a central cause must be actively considered rather than assumed away. The economic and personal costs are concentrated in the progressive phase, when accumulated cerebellar and vestibular disability drives loss of independence — which is precisely the phase in which proportionate vestibular rehabilitation can preserve function [2,49].

It is worth being explicit about why vertigo in MS is so easily mishandled. The symptom is non-specific, the labyrinth and the brainstem generate overlapping bedside findings, and the default assumption in a young patient is a benign peripheral cause. Yet the consequences of the two errors are asymmetric. Treating a coincident BPPV as a relapse exposes the patient to needless corticosteroids and delays the

curative manoeuvre; missing a first demyelinating event delays a diagnosis that now carries real therapeutic weight [1,7,8]. The vestibular physician's discipline — examine the eye movements, localise the lesion, image when central features appear — resolves both errors at once [8,21].

□ **Key Point:** In a young adult with vertigo and any central oculomotor sign, MS is the leading central diagnosis. Conversely, the commonest single cause of new vertigo in a patient who already has MS is BPPV — a peripheral, curable disorder, not a relapse.

II. Pathophysiology — Demyelination of the Vestibular and Oculomotor Pathways

MS lesions form when autoreactive lymphocytes breach the blood–brain barrier and drive focal inflammatory demyelination, with relative early preservation and later loss of axons [2,3,36]. The myelin sheath exists to permit rapid, energetically efficient saltatory conduction; its loss produces conduction slowing, conduction block, and abnormal ephaptic and temperature-sensitive transmission. The vestibular and oculomotor systems are exquisitely sensitive to these changes because they depend on precise, high-frequency, bilaterally balanced signalling through heavily myelinated infratentorial tracts [9,21,23].

This is why the lesions that matter to the vestibular physician cluster in predictable, periventricular and infratentorial locations: the pontine medial longitudinal fasciculus (MLF), the vestibular nuclei and their commissural connections in the floor of the fourth ventricle, the cerebellar flocculus and nodulus, the brachium conjunctivum (superior cerebellar peduncle), and, less often, the intra-pontine fascicle and root-entry zone of the eighth nerve [7,8,9,19].

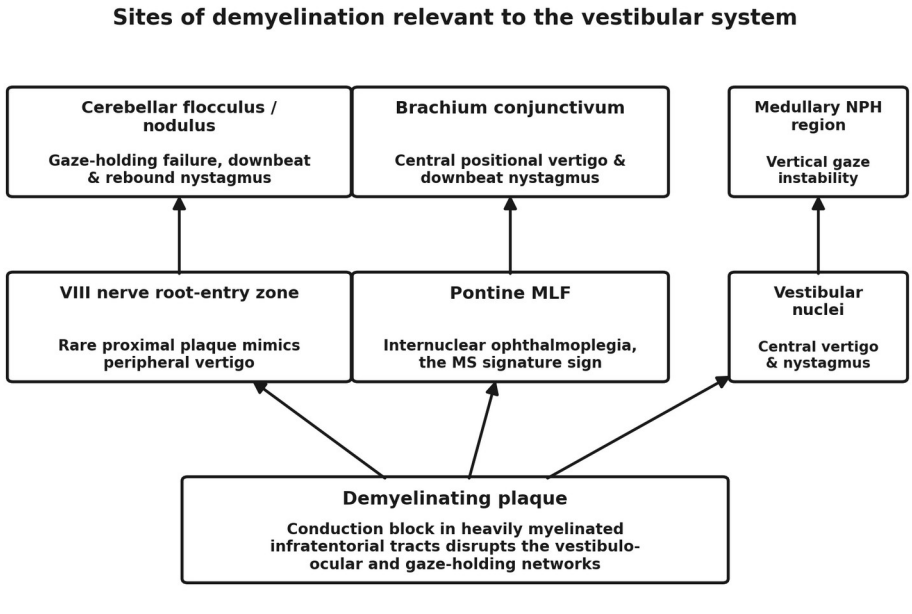

Figure 1. Sites of demyelination relevant to the vestibular system, and the syndrome each produces.
Source: Adapted from Prasad and Galetta [9] and Brandt and Dieterich [24].

Central vestibular imbalance

A plaque affecting the vestibular nuclei or their commissural fibres creates an asymmetry of central vestibular tone that the brain interprets as constant motion, producing vertigo and spontaneous nystagmus indistinguishable at first glance from a peripheral lesion [8,24,25]. The decisive difference is

the company it keeps: central lesions almost always disturb the adjacent gaze-holding and smooth-pursuit machinery, so fixation fails to suppress the nystagmus and additional oculomotor signs appear [10,11,25].

The medial longitudinal fasciculus and internuclear ophthalmoplegia

The MLF carries the internuclear signal from the abducens nucleus in the pons to the contralateral medial rectus subnucleus in the midbrain, yoking the two eyes during horizontal gaze [20,22]. A demyelinating plaque in the MLF slows or blocks this signal, producing internuclear ophthalmoplegia (INO): impaired adduction of the ipsilateral eye with dissociated abducting nystagmus of the contralateral eye, classically with preserved convergence [17,22]. Because the two MLFs lie close together in the dorsal pons, a single plaque can produce bilateral INO — a sign so characteristic that in a young adult it is regarded as MS until proven otherwise [16,17,22].

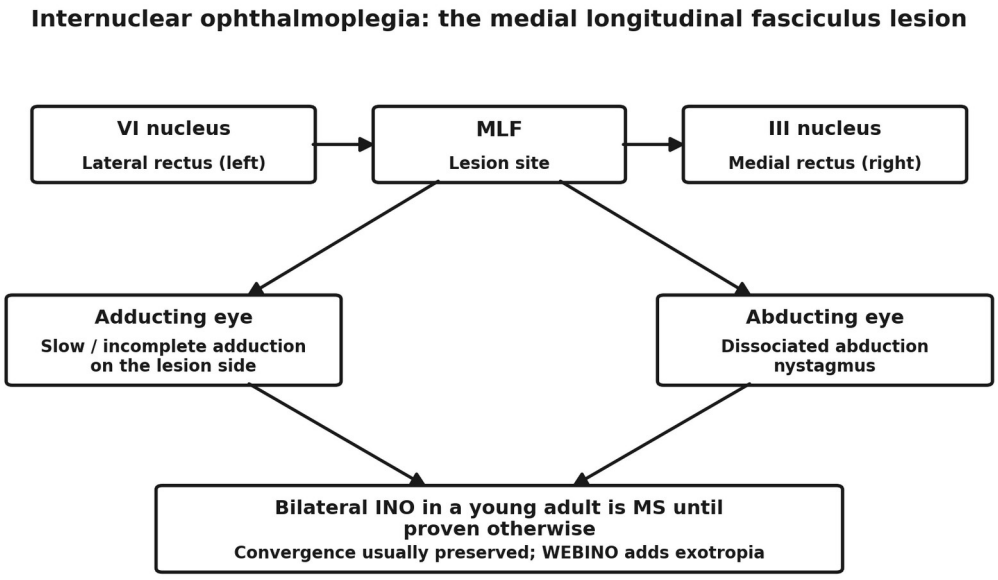

Figure 2. Internuclear ophthalmoplegia — the medial longitudinal fasciculus lesion and its ocular signature.
Source: Adapted from Frohman et al. [22] and Leigh and Zee [20].

The slowing of adducting saccades that defines INO is best understood against the normal pulse-and-step command the brainstem burst neurons generate to move and then hold the eye. A demyelinated MLF degrades the high-frequency pulse, so the adducting saccade is slow and hypometric while the step that holds eccentric gaze is relatively spared [9,22]. Figure 3 summarises this saccadic machinery.

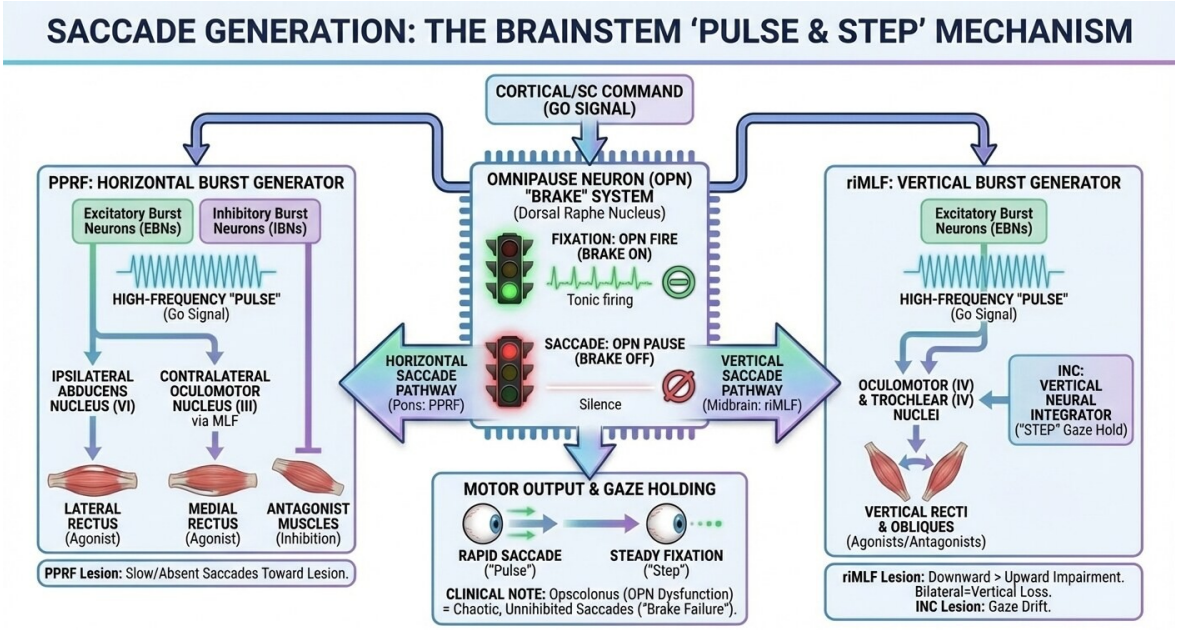

Figure 3. Saccade generation — the brainstem pulse-and-step mechanism that an internuclear lesion disrupts.
Source: Australian Dizziness Clinics education collection.

Cerebellar and brainstem gaze-holding lesions

Demyelination of the cerebellar flocculus, nodulus, or their brainstem projections degrades the neural integrator that holds the eyes steady in eccentric gaze and the velocity-storage mechanism that shapes the vestibulo-ocular reflex [18,20,23]. The clinical results are gaze-evoked and rebound nystagmus, downbeat nystagmus from cervicomedullary or flocculonodular involvement, impaired smooth pursuit, and — most useful at the bedside — failure of fixation to suppress the vestibulo-ocular reflex [10,18,25]. Acquired pendular nystagmus, a slow oscillation that severely degrades vision, reflects disruption of the cerebellar–brainstem network and is comparatively specific to MS and oculopalatal syndromes [13,23].

Central positional vertigo and the eighth nerve

A plaque in the brachium conjunctivum or nodulus can corrupt otolith-derived signals and generate central positional vertigo with downbeat nystagmus on positioning — a presentation that mimics posterior canal BPPV but lacks the latency, fatigability, and canal-specific torsional pattern of the true peripheral disorder [19,24]. Rarely, demyelination of the intra-pontine eighth-nerve fascicle or root-entry zone produces a genuinely peripheral-type vertigo ("pseudoneuritis"), in which the only clue to central localisation may be a paradoxically normal horizontal head-impulse test [8,9].

The temperature sensitivity of demyelinated axons explains a clinical phenomenon the vestibular physician will encounter: Uhthoff's phenomenon, in which a small rise in core temperature transiently worsens conduction and reproduces or amplifies oscillopsia, blurring, and imbalance after exercise or a hot shower [2,21]. This is a functional, reversible conduction failure rather than a new lesion, and recognising it prevents the misattribution of heat-provoked symptoms to a relapse. The same principle — that demyelinated pathways fail under physiological stress — underlies the fatigability of saccades and the worsening of nystagmus with sustained gaze that can be elicited at the bedside [9,23].

At the network level, the velocity-storage mechanism deserves particular attention. Normally the central vestibular system prolongs and integrates peripheral canal signals through commissural connections between the vestibular nuclei, under cerebellar (nodulus and uvula) control [24,25]. A demyelinating plaque that disrupts these connections distorts velocity storage, producing prolonged or perverted responses, periodic alternating nystagmus, and impaired tilt suppression — signs that are essentially never generated by a peripheral lesion and therefore localise with confidence to the central system [25]. This is the mechanistic reason that fixation suppression of the vestibulo-ocular reflex, an everyday bedside test, is so discriminating: suppression depends on an intact cerebellar–brainstem circuit that the plaque interrupts [10,25].

Finally, the relationship between lesion and symptom is not always one-to-one. A single strategically placed dorsal-pontine plaque can produce a constellation of signs — an INO, a skew, and a gaze-evoked nystagmus — by simultaneously affecting the MLF, the interstitial nucleus pathways, and the adjacent gaze-holding circuitry [9,22]. Conversely, a heavy supratentorial lesion load may be clinically silent from a vestibular standpoint. For the vestibular physician this means the oculomotor examination, not the gross lesion count, is the better guide to the functional state of the brainstem and cerebellum [9,42].

□ **Clinical Insight:** Demyelination preferentially strikes heavily myelinated infratentorial tracts. That is precisely why the oculomotor examination localises the lesion: the pattern of nystagmus and the integrity of the head-impulse test, fixation suppression, and saccades tell you whether you are dealing with a central plaque or a peripheral labyrinth.

III. Clinical Features — Vestibular and Oculomotor Syndromes

MS-related vestibular symptoms fall into three broad clinical pictures: an acute vestibular syndrome from a fresh brainstem or cerebellar relapse; episodic positional or paroxysmal vertigo; and chronic imbalance and oscillopsia from accumulated central deficits [7,8,24]. The vestibular physician's task is to recognise the oculomotor fingerprints that betray a central origin and to localise the responsible lesion.

Acute demyelinating vestibular syndrome

A brainstem or cerebellar relapse can produce acute, continuous vertigo with nausea, gait unsteadiness, and spontaneous nystagmus lasting days — clinically an acute vestibular syndrome [8,11]. Most patients have overt central oculomotor signs that make the distinction from vestibular neuritis straightforward, but a minority show a deceptively peripheral pattern of unidirectional, fixation-suppressed nystagmus obeying Alexander's law [8]. In these the HINTS examination is decisive: a normal or atypical horizontal head-impulse test, direction-changing gaze-evoked nystagmus, or skew deviation indicates a central lesion and mandates imaging [11].

Acute vestibular syndrome: MS brainstem relapse vs vestibular neuritis

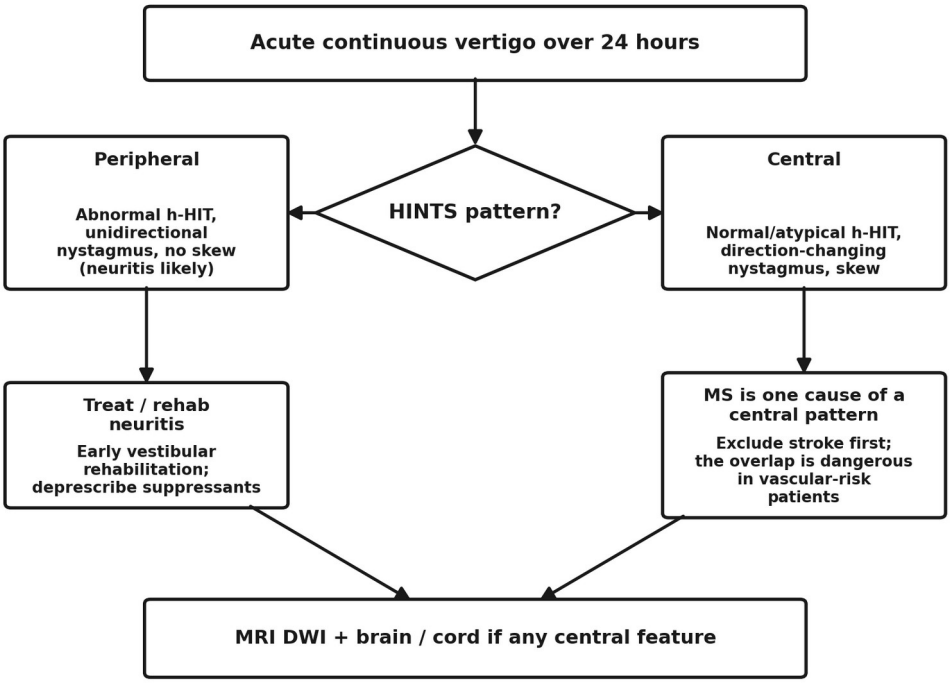

Figure 4. Acute vestibular syndrome — separating an MS brainstem relapse from vestibular neuritis at the bedside.

Source: Adapted from Kattah et al. [11] and Pula et al. [8].

Internuclear ophthalmoplegia and other oculomotor signs

INO is the most clinically useful sign in MS-related neuro-otology. It is detected by asking the patient to make horizontal saccades and watching for slowed adduction of one eye relative to abducting nystagmus of the other; subtle cases show only adduction lag on saccades with full range on pursuit [17,22]. Bilateral INO, with or without the wall-eyed exotropia of WEBINO, is highly suggestive of MS in a young adult [16,17]. INO discriminates MS from neuromyelitis optica spectrum disorder, in which it is far less common [15,17]. Other signs include gaze-evoked, rebound, downbeat, upbeat and acquired pendular nystagmus, saccadic dysmetria, impaired pursuit, and a positive head-impulse test only when the peripheral pathways are also involved [9,18,23].

Table 2. Localising the vestibular and oculomotor sign to the demyelinating lesion.

Sign	Likely lesion site	Comment
Internuclear ophthalmoplegia	Medial longitudinal fasciculus	Bilateral INO in a young adult = MS until proven otherwise [17,22]
Gaze-evoked / rebound nystagmus	Cerebellar flocculus, brainstem integrator	Impaired gaze-holding [18,20]
Downbeat nystagmus	Flocculus / cervicomedullary junction	May worsen on positioning [18,19]
Acquired pendular nystagmus	Cerebellar–brainstem (dentato-olivary) network	Disabling oscillopsia; fairly specific to MS [13,23]
Central positional nystagmus	Nodulus / brachium conjunctivum	No latency, persistent, not canal-specific [19,24]
Impaired VOR fixation suppression	Cerebellum / brainstem	Peripheral lesions suppress normally [10,25]
Normal h-HIT in acute vertigo	Intrapontine 8th nerve fascicle / central	"Pseudoneuritis" — a central red flag [8]

The localisation principle behind Table 2 is summarised graphically in Figure 5: the waveform of the nystagmus maps onto the site of the lesion, which is the conceptual core of the central oculomotor examination.

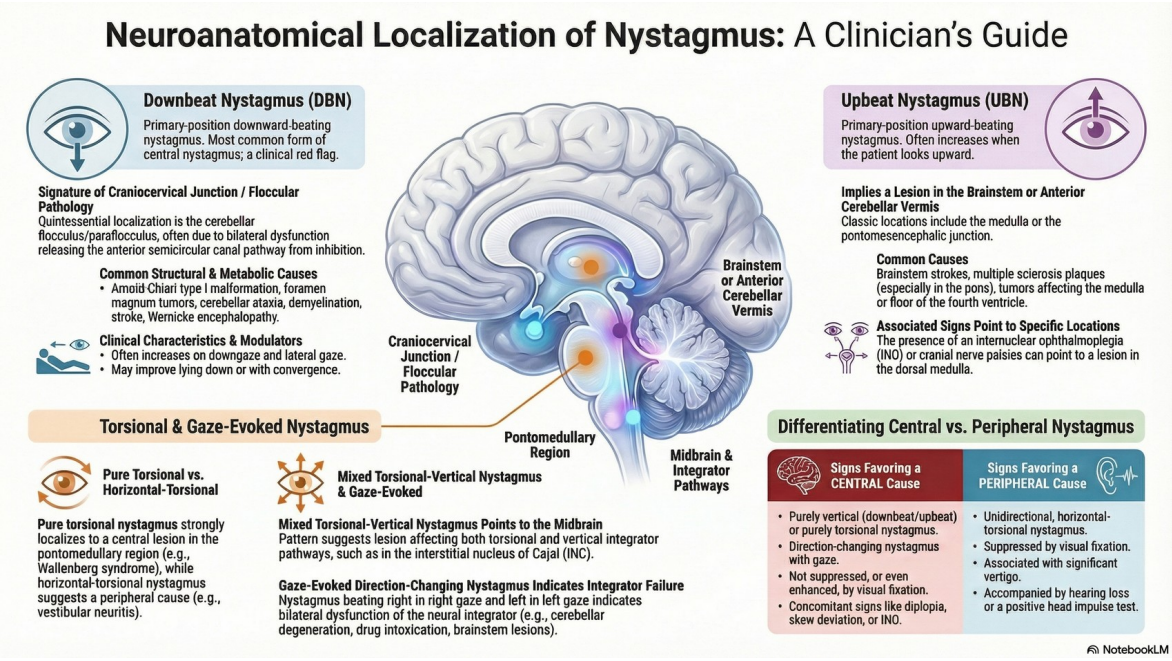

Figure 5. Neuroanatomical localisation of nystagmus — matching the waveform to the lesion site.

Source: Australian Dizziness Clinics education collection.

Figure 6 catalogues the pathological nystagmus types most relevant to central vestibular disease, several of which — downbeat, gaze-evoked, and acquired pendular nystagmus — are characteristic of the brainstem and cerebellar plaques of MS [13,18,23].

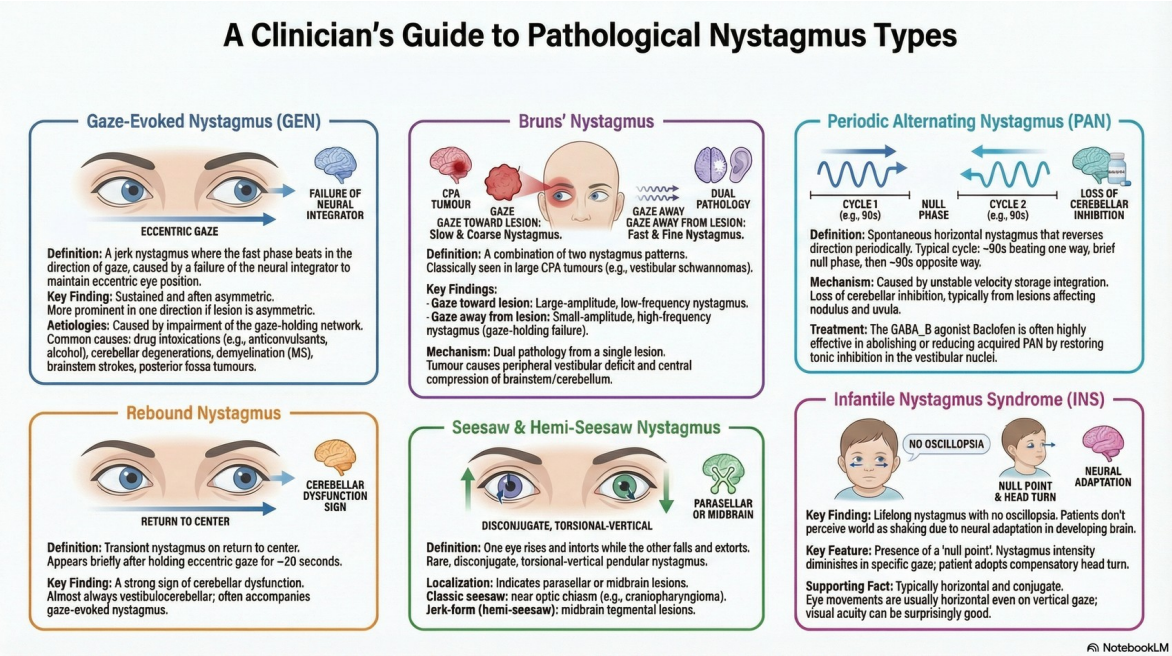

Figure 6. Pathological nystagmus types relevant to central vestibular disease.
Source: Australian Dizziness Clinics education collection.

Episodic and paroxysmal vertigo

Brief, stereotyped attacks of vertigo lasting seconds and triggered by movement or hyperventilation are a recognised paroxysmal symptom of MS, attributed to ephaptic transmission across a demyelinated brainstem plaque, and they respond well to low-dose carbamazepine [2,21]. These must be distinguished from BPPV, which remains the single commonest cause of positional vertigo even in established MS and is cured by particle-repositioning rather than corticosteroids [7].

Chronic imbalance and oscillopsia

Many patients are left with chronic disequilibrium from a combination of cerebellar ataxia, residual vestibular asymmetry, dorsal-column proprioceptive loss, and visual dysfunction [23,49]. Acquired nystagmus produces oscillopsia — the illusory movement of the visual world — which is among the most disabling and least appreciated visual symptoms in MS and a specific target for pharmacological and rehabilitative treatment [13,16,23].

Internuclear ophthalmoplegia exists on a spectrum of severity. In its complete form the adducting eye fails to cross the midline; in subtle disease the only abnormality is a measurable slowing of adducting saccades relative to the fellow abducting eye, best appreciated by asking for large, rapid horizontal refixations and watching the relative onset and velocity of the two eyes [9,22]. The dissociated abducting nystagmus is thought to reflect an adaptive increase in innervation intended to overcome the weak adduction, and it is the more conspicuous of the two components at the bedside [22]. A useful confirmatory sign is preserved convergence despite absent adduction on versional gaze, which localises the lesion to the internuclear pathway rather than the medial rectus or third nerve [17,22].

A structured bedside sequence makes these signs reliably detectable in a few minutes. Begin with fixation in primary position, looking for spontaneous nystagmus and whether removing fixation (with Frenzel lenses or a blank field) increases it — a feature of peripheral, not central, nystagmus [10]. Test the nine cardinal positions for gaze-evoked and rebound nystagmus, then horizontal and vertical saccades specifically for adduction lag and saccadic slowing or dysmetria [9,22]. Examine smooth pursuit, perform the horizontal head-impulse test, and assess fixation suppression of the vestibulo-ocular reflex by having

the patient fix on their own thumb while rotating en bloc [10,25]. Conclude with positional testing, interpreting any downbeat or non-canal-specific response as central until proven otherwise [18,19].

Skew deviation and the ocular tilt reaction warrant separate mention. A vertical misalignment of the eyes, detected on alternate cover testing, reflects disruption of graviceptive otolith pathways ascending from the vestibular nuclei through the brainstem, and in the acute vestibular syndrome it is a specific marker of central — including demyelinating — localisation [11,25]. When skew is accompanied by head tilt and ocular torsion, the full ocular tilt reaction localises to the pontomedullary or pontomesencephalic brainstem and is a valuable, often overlooked, central sign in MS relapse [25].

The chronic visual consequences of these oculomotor disorders are frequently under-reported by patients unless specifically asked. Oscillopsia from acquired pendular or downbeat nystagmus degrades reading and driving, and the resulting visual instability compounds the imbalance caused by cerebellar and proprioceptive deficits [13,16,23]. Eliciting a clear account of oscillopsia, and quantifying its functional impact, is the first step towards the targeted pharmacotherapy and rehabilitation described in Section VIII [13,14].

□ **Clinical Pearl:** Watch the eyes during horizontal saccades in every young patient with unexplained vertigo. An adduction lag — internuclear ophthalmoplegia — can be the only sign that points from the labyrinth to the brainstem and on to a diagnosis of MS.

IV. Diagnostic Assessment and the 2017 McDonald Criteria

The diagnosis of MS rests on demonstrating dissemination of CNS lesions in space (DIS) and in time (DIT) while excluding a better explanation [1]. The vestibular physician rarely makes the formal diagnosis but is frequently the clinician who first suspects it — from an oculomotor sign — and who initiates the imaging and onward referral that bring it about [8,21].

Assessment begins with the structured vestibular history and the TiTrATE framework — timing, triggers, and a targeted oculomotor examination — applied with a low threshold for central localisation in a young adult [31]. The examination should systematically cover spontaneous and gaze-evoked nystagmus, smooth pursuit, saccades (looking specifically for adduction lag), the head-impulse test, fixation suppression of the vestibulo-ocular reflex, and a search for skew deviation [10,11,17]. Any central feature triggers the diagnostic pathway in Figure 7.

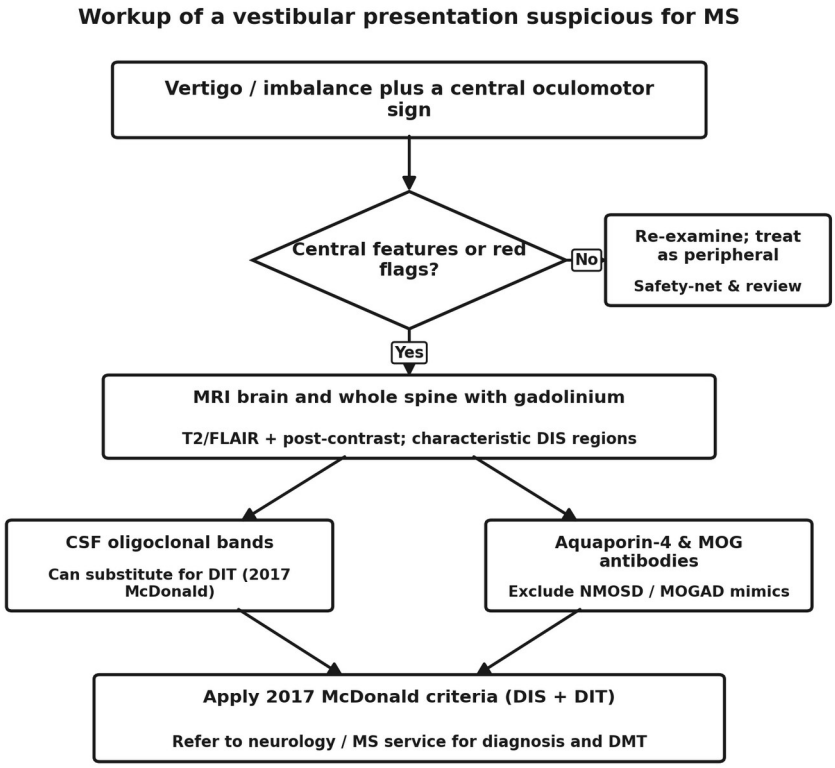

Figure 7. Workup of a vestibular presentation suspicious for multiple sclerosis.
Source: Adapted from Thompson et al. [1] and Pula et al. [8].

The 2017 revision of the McDonald criteria made the diagnosis achievable earlier and more often at the time of a first clinically isolated syndrome [1]. Two changes are particularly relevant to brainstem and vestibular presentations: the presence of CSF-specific oligoclonal bands can now substitute for dissemination in time, and a symptomatic infratentorial or spinal lesion may be counted towards dissemination in space — a reversal of the older rule that excluded the symptomatic lesion [1]. This matters because the brainstem plaque responsible for an MS vertigo is now usable evidence rather than discarded [1,8].

Table 3. Dissemination in space and time under the 2017 McDonald criteria (simplified).

Dissemination in space (DIS)	Dissemination in time (DIT)
One or more T2 lesions in at least two of four characteristic regions: periventricular, cortical/juxtacortical, infratentorial, spinal cord [1]	A new T2 or gadolinium-enhancing lesion on follow-up MRI compared with a baseline scan [1]
Symptomatic lesions now count towards DIS (2017 change), including the brainstem plaque causing the vertigo [1]	Simultaneous presence of gadolinium-enhancing and non-enhancing lesions on a single scan [1]
Optic nerve involvement is not yet formally included but is under active study [1]	CSF-specific oligoclonal bands can substitute for DIT in a typical clinically isolated syndrome (2017 change) [1]

Misdiagnosis remains a real hazard. Over-reliance on non-specific white-matter changes, migraine-related lesions, and small-vessel disease leads to incorrect MS labels in a substantial minority of referrals, with attendant exposure to disease-modifying therapy [48]. The discipline of requiring objective, characteristically located lesions and a clinical syndrome that fits — rather than treating MRI signal in isolation — protects the patient [1,37,48].

The conceptual core of the criteria — dissemination in space and time, with no better explanation — has been stable since the 2010 revision, but the 2017 changes lowered the threshold for an early, secure diagnosis [1,35]. The historical Barkhof criteria established which lesion locations carry diagnostic weight,

and that framework persists in the modern definition of dissemination in space across periventricular, cortical or juxtacortical, infratentorial, and spinal regions [37]. For a vestibular presentation, the infratentorial criterion is usually the one being satisfied by the symptomatic plaque itself, with a second region required to complete dissemination in space [1,37].

Prognostic stratification at first presentation is increasingly important because it informs the decision to start disease-modifying therapy. The presence of multiple T2 lesions, oligoclonal bands, and infratentorial or spinal involvement at the index event each raise the probability and the pace of conversion to definite MS [38]. A vestibular-onset clinically isolated syndrome with a brainstem plaque and positive CSF therefore sits towards the higher-risk end of the spectrum, which is one more reason that prompt and complete workup is not a neutral act [1,38].

□ **Important:** A symptomatic brainstem lesion now counts towards dissemination in space under the 2017 criteria. The vestibular plaque you identify is diagnostic evidence — but equally, do not over-call MS on non-specific white-matter change; misdiagnosis exposes patients to unnecessary immunotherapy.

V. Investigations and the Role of Imaging

MRI of the brain and whole spinal cord with gadolinium is the central investigation, and standardised protocols substantially improve diagnostic yield and reproducibility [1,47]. For vestibular presentations, thin infratentorial sections are essential: the small dorsal-pontine plaque that causes an INO or a central acute vestibular syndrome is easily overlooked on routine sequences, and 12% of diffusion-weighted scans are falsely negative within the first 48 hours of an acute event [8,11,47].

Cerebrospinal fluid analysis for CSF-specific oligoclonal bands supports the diagnosis and, under the 2017 criteria, can establish dissemination in time [1]. Serum aquaporin-4 and myelin oligodendrocyte glycoprotein antibodies should be checked when the presentation is atypical, to identify neuromyelitis optica spectrum disorder and MOG-associated disease, which are managed differently and in which INO is uncommon [15,17].

Vestibular function testing

Dedicated vestibular testing characterises and quantifies the deficit but does not diagnose MS. Caloric and rotational testing and video head-impulse testing are frequently normal or only mildly abnormal in central disease, which is itself informative when symptoms are prominent [9,28]. Impaired fixation suppression of the vestibulo-ocular reflex and abnormal smooth pursuit point to the cerebellum and brainstem [10,25]. Vestibular-evoked myogenic potentials and audiometric studies may reveal subclinical brainstem and eighth-nerve involvement, reflecting the historically recognised audiovestibular footprint of MS [28,30]. Optical coherence tomography of the retinal nerve fibre layer provides a sensitive, quantitative readout of the optic neuropathy that so often accompanies the vestibular syndrome [16].

Failure of fixation to suppress the vestibulo-ocular reflex is one of the most discriminating bedside and laboratory signs of central involvement in MS; Figure 8 summarises the cerebellar and brainstem basis of this finding and the clues that separate it from a peripheral pattern [10,25].

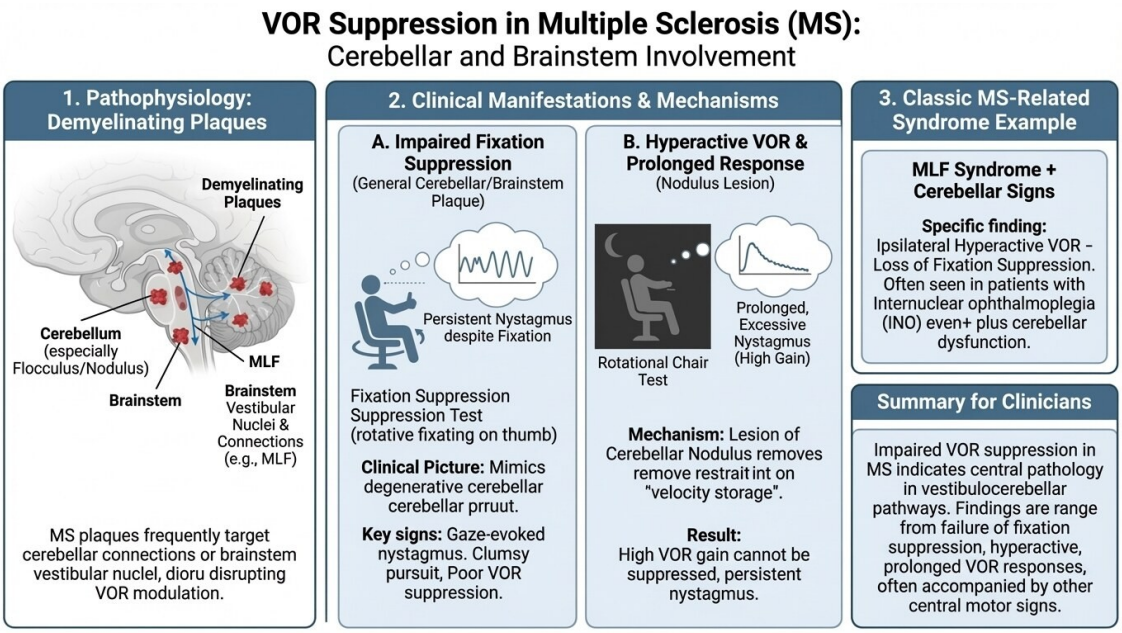

Figure 8. VOR fixation-suppression changes in multiple sclerosis — a cerebellar and brainstem sign, with clues that separate it from peripheral disease.

Source: Australian Dizziness Clinics education collection.

Protocol matters as much as access to MRI. Standardised acquisition with three-dimensional FLAIR, thin-section infratentorial imaging, and post-gadolinium sequences materially increases the detection of the small dorsal-brainstem and peduncular lesions responsible for vestibular and oculomotor syndromes [47]. Where the clinical suspicion is high and the initial scan is unrevealing, the lesion may be below the resolution of routine sequences or may sit in the cervical cord; whole-spine imaging is part of the vestibular workup, not an optional extra [1,47]. Repeat imaging after an interval can demonstrate dissemination in time when the first study is equivocal [1].

Electrophysiology retains a supporting role. Brainstem auditory evoked responses and, where available, vestibular-evoked myogenic potentials can demonstrate subclinical conduction abnormality along the audiovestibular pathways, echoing the otoneurological footprint of MS described in earlier systematic study of the audiovestibular system [28,30]. These add little when the MRI and clinical picture are already concordant, but they can be informative when symptoms outstrip imaging findings and a functional marker of brainstem involvement is wanted [28,30].

Quantitative oculography is emerging as a sensitive functional biomarker. Infrared video recording of saccades can reveal subclinical adduction slowing — a forme fruste of INO — when the bedside examination looks normal, and can track the response to symptomatic treatment of nystagmus over time [9,23]. Combined with optical coherence tomography of the retinal nerve fibre layer, these tools give the vestibular physician an objective, repeatable readout of brainstem and visual-pathway integrity that complements MRI and the structured clinical examination [16,23].

□ **Clinical Insight:** Normal caloric and head-impulse testing in a patient with florid vertigo is not reassurance — in the right context it is a pointer towards a central, possibly demyelinating, cause. Always pair quantitative vestibular testing with the oculomotor examination and infratentorial MRI.

VI. Differential Diagnosis

The differential runs in two directions: distinguishing MS-related vertigo from peripheral vestibular disorders, and distinguishing MS from other central and inflammatory causes [8,15,24]. The unifying principle is that a central oculomotor sign reorients the workup towards the brainstem and cerebellum, while its absence — together with a clear positional or canal-specific pattern — favours a peripheral, often curable, diagnosis [11,24].

Table 4. Differential diagnosis of vertigo in the young adult with possible MS.

Condition	Discriminating features	Pitfall
Vestibular neuritis	Abnormal h-HIT, unidirectional fixation-suppressed nystagmus, no central signs [26]	A normal h-HIT in 'neuritis' suggests central pseudoneuritis (MS or stroke) [8]
BPPV	Latency, fatigability, canal-specific torsional/up-beat nystagmus; cured by repositioning [7]	Still the commonest cause of new vertigo in established MS — do not treat as a relapse [7]
Posterior circulation stroke / TIA	Vascular risk, abrupt onset, central HINTS pattern [11,32]	MS and stroke can share a central HINTS pattern; exclude stroke first [11]
Vestibular migraine	Headache/photophobia, recurrent, normal interictal exam	Common comorbidity; can coexist with MS
NMOSD / MOGAD	Severe optic neuritis, longitudinally extensive myelitis, AQP4/MOG antibodies [15]	INO is uncommon — its presence favours MS over NMOSD [17]
Chiari malformation / posterior fossa lesion	Downbeat nystagmus, structural MRI abnormality [18]	Central positional downbeat can mimic posterior-canal BPPV [19]

Because the peripheral-versus-central distinction governs the entire workup, it is worth internalising the contrasting nystagmus patterns. Figure 9 sets the features side by side — direction-fixed, fixation-suppressed, head-impulse-positive peripheral nystagmus against the direction-changing, fixation-resistant, head-impulse-negative central pattern seen in MS and stroke [10,11,25].

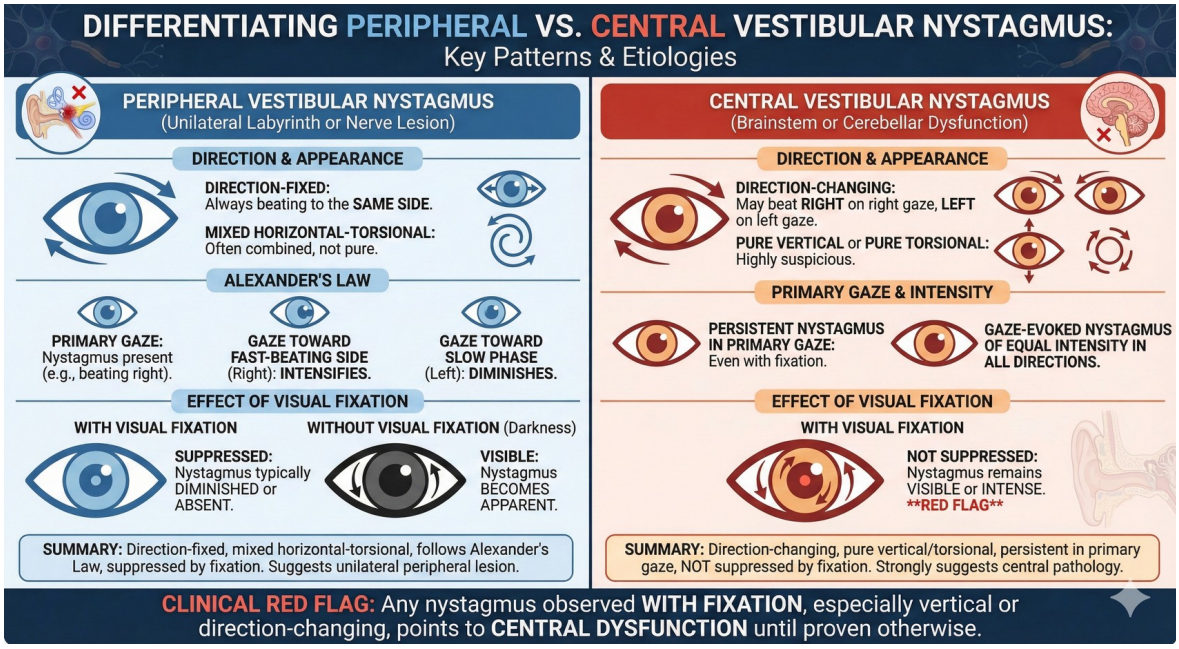

Figure 9. Differentiating peripheral from central vestibular nystagmus.
Source: Australian Dizziness Clinics education collection.

Two scenarios deserve emphasis. First, the patient with an apparent acute vestibular syndrome and a vascular risk profile: here stroke is the immediate priority and MS a secondary consideration, but both produce a central HINTS pattern, so neuroimaging governs disposition [11,32]. Second, the patient with established MS and new positional vertigo: the temptation to attribute every symptom to a relapse leads to inappropriate corticosteroids, when a Dix–Hallpike and a repositioning manoeuvre would cure a coincident BPPV [7].

Beyond the headline conditions, several quieter mimics recur in practice. Vestibular paroxysmia — brief attacks from neurovascular cross-compression of the eighth nerve — can imitate the paroxysmal vertigo of MS but responds to carbamazepine and lacks central oculomotor signs [24,40]. Functional and

persistent postural-perceptual dizziness frequently coexists with organic vestibular disease in MS, and recognising the functional overlay prevents both over-investigation and the false reassurance that nothing is wrong [24]. Migraine-related white-matter lesions are a notorious source of MS misdiagnosis, underscoring the need to anchor the diagnosis in a compatible clinical syndrome rather than imaging alone [29,48].

The antibody-mediated disorders deserve a final, practical word. Neuromyelitis optica spectrum disorder and MOG-associated disease can both present with brainstem syndromes including intractable nausea, vomiting, and vertigo from area postrema and dorsal-brainstem involvement [15]. The relative rarity of internuclear ophthalmoplegia in these conditions, contrasted with its frequency in MS, makes the oculomotor examination a useful early discriminator while antibody results are awaited — a distinction with direct treatment consequences, since some MS therapies aggravate NMOSD [15,17].

□ **Important:** In any acute vestibular syndrome with vascular risk factors, exclude posterior circulation stroke before attributing vertigo to demyelination — the bedside HINTS pattern overlaps, and the time-critical diagnosis is the stroke.

VII. Management of MS and its Vestibular Manifestations

Management has three arms: treating the acute relapse, modifying the disease to prevent future relapses and lesion accrual, and treating the vestibular and oculomotor symptoms themselves [33,44]. The first two are neurology-led; the third is where the vestibular physician adds the most value [13,14].

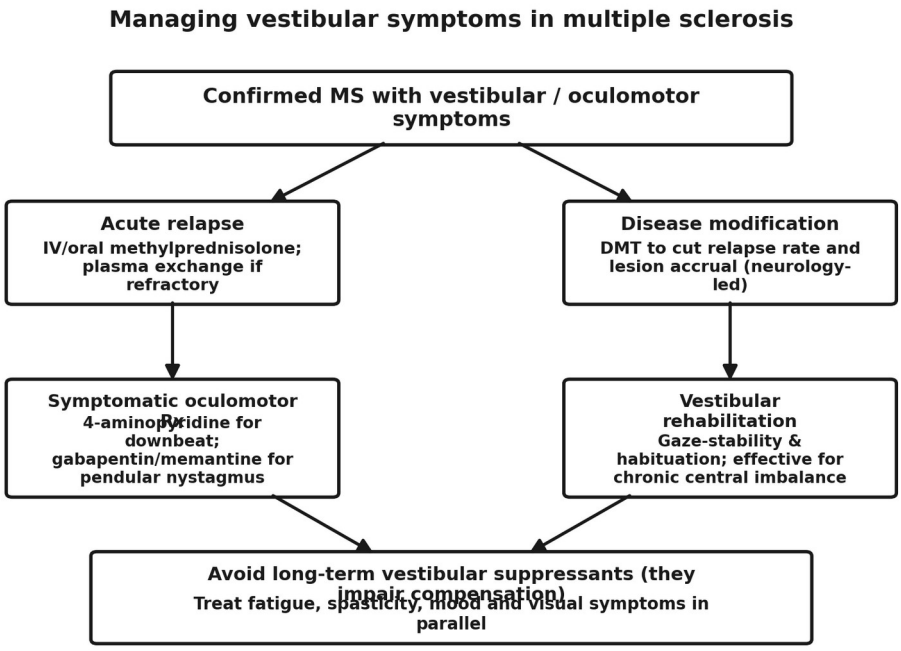

Figure 10. A management framework for vestibular symptoms in confirmed multiple sclerosis.
Source: Adapted from Montalban et al. [33] and Strupp et al. [40].

Acute relapse

A disabling brainstem or cerebellar relapse is treated with high-dose corticosteroids — typically intravenous or oral methylprednisolone — which hasten recovery without altering long-term disability [34,44]. In the Pula series, all demyelinating acute vestibular syndromes improved with steroids [8]. Plasma exchange is reserved for severe steroid-refractory relapses [44]. The decision to treat a vestibular

symptom as a relapse must follow confirmation that it is central and demyelinating rather than a coincident BPPV or a peripheral event [7,8].

Disease-modifying therapy

Disease-modifying therapies reduce relapse rate, MRI lesion accrual, and — for the higher-efficacy agents — disability progression, and current ECTRIMS/EAN guidance favours early effective treatment in active relapsing disease [33,44]. Early recognition of a vestibular-onset clinically isolated syndrome therefore has therapeutic consequence: the sooner the diagnosis is secured, the sooner effective treatment can begin [1,33]. Prescribing and monitoring sit with the treating neurologist, but the vestibular physician's prompt referral is part of the therapeutic chain.

Symptomatic principles

Two principles govern symptomatic care. First, treat coincident peripheral disease on its own terms — repositioning for BPPV, not steroids [7]. Second, avoid long-term vestibular suppressants: like any central or peripheral vestibular lesion, MS-related imbalance compensates better when the sedating antihistamines and benzodiazepines that blunt central adaptation are withdrawn [26,40]. Fatigue, spasticity, mood, heat sensitivity, and visual symptoms should be addressed in parallel because each amplifies perceived imbalance [27,44].

Short-term symptom control during an acute relapse is legitimate but must be time-limited. Anti-emetics and vestibular sedatives can be used for the first 24 to 72 hours of severe vertigo and vomiting, then withdrawn so that central adaptation is not blunted — the same discipline applied in peripheral acute vestibular syndromes [26,40]. Prolonged prescribing of benzodiazepines or sedating antihistamines is a common and avoidable error that converts a recoverable relapse into protracted disability by suppressing the very plasticity that drives recovery [26,40]. Where paroxysmal vertigo or ephaptic brainstem symptoms dominate, low-dose carbamazepine is a more rational and better-tolerated choice than chronic sedation [21,40].

The modern disease-modifying armamentarium ranges from injectable and oral agents of moderate efficacy to high-efficacy monoclonal antibodies, and the trend of evidence favours earlier use of more effective treatment in active relapsing disease [33,44]. The vestibular physician does not select these agents but should understand that the diagnostic work they initiate feeds directly into a treatment decision with a narrow window of maximal benefit — neuro-axonal loss accrues from the earliest stages, and relapses incompletely recovered leave fixed deficits [33,44]. This reframes the prompt recognition of a vestibular-onset clinically isolated syndrome as a time-critical contribution to long-term outcome.

Coordination of care is the other practical theme. A patient with MS and vertigo may need, in sequence, a repositioning manoeuvre, a course of steroids for a confirmed relapse, an adjustment of disease-modifying therapy, symptomatic treatment of nystagmus, and a tailored rehabilitation programme — drawing on the vestibular physician, the neurologist, and the vestibular physiotherapist [14,33,40]. Establishing who owns each decision, and communicating clearly between disciplines, prevents the fragmentation of care that leaves vestibular symptoms untreated while the underlying disease is managed in isolation [27].

□ **Clinical Pearl:** Confirm that an MS vestibular symptom is central and demyelinating before reaching for corticosteroids. A coincident BPPV is cured with an Epley manoeuvre in minutes; a relapse is treated with steroids and a review of disease-modifying therapy — getting this distinction right spares the patient both errors.

VIII. Symptomatic Treatment, Oscillopsia, and Vestibular Rehabilitation

Acquired nystagmus and chronic central imbalance often persist between relapses and are the symptoms most amenable to the vestibular physician's direct intervention. Treatment combines targeted pharmacotherapy for nystagmus and oscillopsia with structured vestibular and balance rehabilitation [13,40,43].

Pharmacotherapy for nystagmus and oscillopsia

Downbeat nystagmus responds to the potassium-channel blockers 3,4-diaminopyridine and 4-aminopyridine, which restore the inhibitory influence of the vestibulocerebellum on the vestibular nuclei; in placebo-controlled study, a single dose of 3,4-diaminopyridine more than halved slow-phase velocity in most patients and reduced oscillopsia [12,40]. Sustained-release fampridine (4-aminopyridine) is licensed to improve walking speed in MS and may additionally benefit cerebellar oculomotor symptoms [45]. Acquired pendular nystagmus — comparatively specific to MS — responds to gabapentin and memantine, each of which reduced eye-movement velocity and improved visual acuity in a controlled crossover trial [13,43]. These agents are titrated against benefit and sedation; baclofen has a role in upbeat and see-saw forms [40,43].

Table 5. Symptomatic pharmacotherapy for MS-related nystagmus and oscillopsia.

Nystagmus type	First-line agent	Evidence / notes
Downbeat nystagmus	3,4-diaminopyridine or 4-aminopyridine	Placebo-controlled benefit; restores cerebellar inhibition [12,40]
Acquired pendular nystagmus	Gabapentin or memantine	Controlled crossover trial; both reduce eye speed [13]
Upbeat / see-saw nystagmus	Baclofen / memantine	Lower-grade evidence; titrate to effect [40,43]
Gait/cerebellar symptoms	Sustained-release fampridine	Improves walking speed in MS [45]

Vestibular and balance rehabilitation

Vestibular rehabilitation is effective in MS-related imbalance and is under-used. In a randomised controlled trial, a six-week vestibular rehabilitation programme produced clinically meaningful improvements in fatigue, balance, and dizziness-related disability that exceeded both endurance exercise and usual care [14]. A subsequent randomised trial of combined balance and eye-movement exercises confirmed durable gains in postural control [50], and systematic reviews support exercise-based rehabilitation across the disability spectrum [41]. Programmes combine gaze-stability (vestibulo-ocular reflex) exercises, habituation to provocative movement, and progressive balance training, individualised to the patient's deficits and fatigue ceiling [14,50].

Outcome is best tracked with validated instruments such as the Dizziness Handicap Inventory alongside objective posturography and gait-speed measures, which capture the disability that matters to the patient and demonstrate response to treatment [14,46]. Heat sensitivity and fatigue should be accommodated in scheduling, since both transiently worsen conduction and perceived imbalance [27,44].

The rationale for rehabilitation in central disease differs from that in a fixed peripheral lesion. In unilateral peripheral loss, recovery depends on central compensation for a static deficit; in MS the deficit is multifocal, fluctuating, and superimposed on cerebellar and proprioceptive loss, so the programme targets sensory reweighting, gaze stability, and postural strategy rather than simple compensation [14,49]. The evidence base, though smaller than for peripheral disorders, is consistent: supervised, progressive, individualised programmes improve balance and dizziness-related disability and reduce fatigue, with gains that persist at follow-up [14,41,50].

Practical delivery has to respect the disease. Sessions are paced to avoid heat- and fatigue-induced deterioration, progressed sub-symptom-threshold, and adapted as relapses change the deficit; home programmes sustain the gains made in supervised sessions [14,50]. Where oscillopsia limits gaze-stability training, pharmacological reduction of nystagmus can be a necessary precursor to effective rehabilitation, illustrating how the drug and exercise arms of symptomatic care reinforce one another [13,40]. Driving fitness should be assessed where oscillopsia, diplopia, or imbalance is significant, with reference to the relevant national standards [16].

□ **Clinical Insight:** Vestibular rehabilitation is one of the most effective and least-prescribed treatments for chronic MS imbalance — it improves balance, dizziness-related disability, and even fatigue. Pair it with agent-specific pharmacotherapy for nystagmus, and avoid sedating suppressants that blunt adaptation.

IX. Prognosis, Disease Course, and Special Populations

The prognosis of an individual vestibular relapse is generally good: most acute demyelinating vestibular syndromes recover substantially with steroids and central compensation [8]. The prognosis of the underlying disease is more variable and depends on phenotype, lesion burden, and treatment [2,6,38].

Brainstem and cerebellar involvement at onset, incomplete recovery from early relapses, and a high infratentorial lesion load are associated with greater long-term disability [38,42]. Vertigo and imbalance recur across the disease course as new lesions accrue, and chronic disequilibrium becomes a dominant contributor to falls and reduced quality of life in progressive disease [29,39,49]. Falls in MS are multifactorial — combining vestibular, cerebellar, proprioceptive, visual, and motor deficits — so their assessment and prevention is intrinsically a vestibular-physician concern [49].

Special populations

Paediatric-onset MS is uncommon but recognised; children have a high relapse rate but typically recover well from individual events, and demyelinating vertigo in a child warrants the same central workup with particular care to exclude alternative inflammatory and structural causes [3]. In pregnancy, relapse rate falls in the third trimester and rises in the early post-partum period, which influences the timing of investigation and treatment of vestibular relapses [2,33]. Comorbidity — migraine, anxiety, depression, and coincident peripheral vestibular disease — is highly prevalent and consistently worsens symptom burden and outcome; its recognition and treatment is among the highest-yield interventions available [27].

The trajectory of vestibular symptoms tends to mirror the disease phenotype. In relapsing–remitting disease, vertigo arrives as discrete, largely recoverable events; in secondary and primary progressive disease, a more insidious, continuous disequilibrium predominates, reflecting accumulated cerebellar, brainstem, and spinal pathology rather than discrete plaques [2,6,39]. This distinction shapes expectations and treatment: episodic central vertigo justifies relapse-directed therapy and watchful rehabilitation, whereas chronic progressive imbalance is managed predominantly through sustained rehabilitation, falls prevention, and symptomatic measures [39,49].

Falls are the outcome that most directly concerns the vestibular physician. People with MS fall frequently, and the risk is driven by the same multisensory deficits — vestibular, cerebellar, proprioceptive, and visual — that the vestibular assessment is designed to characterise [49]. Structured falls-risk evaluation, gait-speed measurement, and targeted balance training therefore sit naturally within vestibular practice and offer one of the clearest opportunities to preserve independence in progressive disease [42,49].

□ **Key Point:** An individual vestibular relapse usually recovers well, but recurrent vertigo and accumulating cerebellar–vestibular deficits drive falls and disability in progressive disease. Treating comorbid migraine, mood disorder, and coincident BPPV materially improves the patient's overall vestibular burden.

X. Guidelines, Controversies and Future Directions

Diagnosis is governed by the 2017 McDonald criteria, which evolved from the 2010 revision to enable earlier, more sensitive diagnosis while preserving specificity [1,35]. Treatment is guided by the ECTRIMS/EAN recommendations on disease-modifying therapy and by standardised MRI protocols for diagnosis and monitoring [33,47]. For the vestibular physician, the practical message of these guidelines is consistency: a structured oculomotor examination, protocolised infratentorial imaging, and disciplined application of the criteria.

Several controversies remain live. The boundary between MS and the antibody-mediated disorders — neuromyelitis optica spectrum disorder and MOG-associated disease — continues to be refined, and accurate separation matters because their treatments diverge and some MS therapies worsen NMOSD [15]. Misdiagnosis driven by over-interpretation of non-specific white-matter change remains a documented harm and argues for diagnostic restraint [48]. The optimal symptomatic management of

acquired nystagmus is still being defined, and modelling of the underlying oculomotor instability is guiding rational drug choice [23].

Future directions of relevance to vestibular medicine include the formal incorporation of optic-nerve and advanced-imaging markers into the diagnostic criteria, quantitative video-oculography as a sensitive biomarker of brainstem and cerebellar involvement, and the understanding of how geography and environment shape MS risk in high-prevalence regions such as Australia [1,5,23]. As earlier diagnosis and higher-efficacy therapy become standard, the vestibular physician's role in recognising the oculomotor herald of MS — and in rehabilitating its enduring vestibular consequences — only grows.

A recurring tension in the field is the balance between sensitivity and specificity in diagnosis. Each successive revision of the criteria has made early diagnosis easier, but the same liberalisation increases the risk of mislabelling patients whose white-matter changes are vascular, migrainous, or incidental [1,35,48]. The corrective is clinical: a diagnosis of MS should rest on an objective syndrome in a characteristic location, supported by appropriately acquired imaging and, where needed, cerebrospinal fluid — not on radiological pattern-matching [37,47,48]. The vestibular physician contributes to specificity by confirming that an oculomotor or vestibular sign is genuinely central before the diagnostic machinery is set in motion.

Looking ahead, the integration of quantitative oculography, optical coherence tomography, and fluid biomarkers promises a more precise, earlier characterisation of brainstem and cerebellar involvement than lesion counting allows [16,23,47]. Coupled with the move towards earlier high-efficacy therapy, this raises the value of the clinician who can detect the subtle oculomotor herald of central disease. In a high-prevalence setting such as Australia, embedding structured vestibular assessment in the early evaluation of young adults with vertigo is both a diagnostic and a public-health opportunity [4,5,23].

□ **Clinical Pearl:** The vestibular physician is often the first to see the oculomotor herald of MS and the best placed to rehabilitate its lasting vestibular sequelae. Recognise the central sign, image the posterior fossa properly, refer early, and treat the symptoms that disable the patient between relapses.

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