Australian Dizziness Clinics

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VESTIBULAR PHYSICIAN CHEAT SHEET

Multiple Sclerosis and the Vestibular System

Mechanism · Diagnosis · Management — a desk reference for vestibular physicians

SNAPSHOT

Onset 20–40 y, F:M ~3:1; prevalence ~36/100,000 with a strong latitude gradient (Australia high). Relapsing-remitting ~85%. Dizziness/vertigo affects ~50% at some stage; vertigo is the presenting symptom in ~5%; demyelination causes ~4% of high-risk AVS.

Three mechanisms of vertigo in MS: (1) central brainstem/cerebellar plaque; (2) coincident peripheral disorder — BPPV is the commonest cause of NEW vertigo in established MS; (3) chronic multisensory imbalance.

♦ *Bilateral INO in a young adult with vertigo is MS until proven otherwise.*

WHEN TO SUSPECT A CENTRAL / DEMYELINATING CAUSE

- Internuclear ophthalmoplegia (adduction lag + dissociated abducting nystagmus); gaze-evoked, rebound, downbeat or acquired pendular nystagmus; impaired VOR fixation suppression; skew deviation; central HINTS pattern; vertigo in a young adult with prior neurological symptoms.

BEDSIDE OCULOMOTOR EXAM — LOCALISE THE SIGN

Sign	Lesion site	Note
INO (adduction lag)	Medial longitudinal fasciculus	Bilateral = MS until proven otherwise; convergence preserved
Gaze-evoked / rebound	Cerebellar flocculus / integrator	Impaired gaze-holding
Downbeat nystagmus	Flocculus / cervicomedullary jn	May worsen on positioning
Acquired pendular	Cerebellar–brainstem network	Disabling oscillopsia; fairly specific to MS
Impaired VOR suppression	Cerebellum / brainstem	Peripheral lesions suppress normally
Normal h-HIT in acute vertigo	Central / pseudoneuritis	Central red flag — image

DIAGNOSIS & INVESTIGATIONS

- **2017 McDonald criteria:** dissemination in space + time, no better explanation. CSF oligoclonal bands can substitute for DIT; a symptomatic infratentorial/spinal lesion now counts for DIS.
- MRI brain + whole spine with gadolinium (thin infratentorial slices; early DWI can be falsely negative). Check AQP4 + MOG antibodies if atypical (exclude NMOSD/MOGAD — INO is uncommon in NMOSD).

♦ *Vestibular testing characterises but does not diagnose; normal calorics with florid vertigo points central.*

DIFFERENTIAL DIAGNOSIS

Condition	Discriminator
Vestibular neuritis	Abnormal h-HIT, unidirectional fixation-suppressed nystagmus, no central signs
BPPV	Latency, fatigability, canal-specific torsional nystagmus — cured by repositioning
Posterior circulation stroke	Vascular risk, abrupt onset — exclude FIRST (shares central HINTS)
NMOSD / MOGAD	Severe optic neuritis, LETM, AQP4/MOG positive; INO uncommon

MANAGEMENT

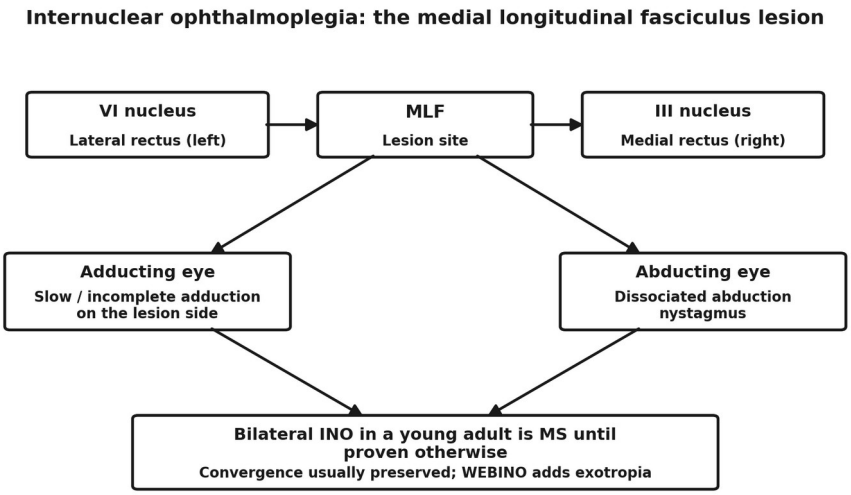
- **Relapse:** high-dose IV/oral methylprednisolone; plasma exchange if refractory. **Disease modification:** neurology-led DMT — refer early (narrow therapeutic window).
- **Symptomatic:** 4-aminopyridine for downbeat; gabapentin/memantine for pendular nystagmus; vestibular rehabilitation for chronic imbalance. Treat coincident BPPV by repositioning, not steroids.

SYMPTOMATIC PHARMACOTHERAPY FOR NYSTAGMUS / OSCILLOPSIA

Nystagmus	First-line agent	Note
Downbeat	4-aminopyridine (or 3,4-DAP)	Restores cerebellar inhibition; placebo-controlled benefit
Acquired pendular	Gabapentin or memantine	Controlled crossover trial; both reduce eye speed
Upbeat / see-saw	Baclofen / memantine	Lower-grade evidence; titrate to effect
Gait / cerebellar	Sustained-release fampridine	Improves walking speed in MS

TWO-MINUTE BEDSIDE OCULOMOTOR SEQUENCE

- Primary gaze — spontaneous nystagmus; does removing fixation increase it? (peripheral)
- Horizontal saccades — watch for adduction lag (INO); vertical saccades — slowing/dysmetria.
- Eccentric gaze — gaze-evoked / rebound; smooth pursuit; head-impulse test.
- VOR fixation suppression (fix on thumb, rotate en bloc) + alternate cover for skew; positional testing last.



Internuclear ophthalmoplegia — a medial longitudinal fasciculus plaque slows ipsilateral adduction with dissociated abducting nystagmus. Bilateral INO in a young adult is MS until proven otherwise.

⚑ Red flags — escalate / image

New central oculomotor sign, acute vestibular syndrome with vascular risk (exclude stroke), severe optic neuritis or longitudinally extensive myelitis (consider NMOSD/MOGAD), rapidly progressive deficit.

♦ *Avoid long-term vestibular suppressants — they blunt central compensation and prolong disability.*

