Mal de Débarquement Syndrome (MdDS):

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

Vestibular Medicine for Vestibular Physicians

Functional and Chronic Vestibular Disorders — Module 4.4

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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 functional and chronic vestibular practice, where a working command of mechanism, criteria, and atypical presentations is expected rather than optional.

The review is dense by design — intended as a 30–40 minute deep read or a desktop reference. It is supported by an A4 clinician cheat sheet, short-form clinician videos, audio episodes, and a patient information leaflet within the same Education Hub module.

Callout Box Guide

□ **Key Point:** Foundational concepts and summary statements that anchor the core clinical content of each section.

□ **Clinical Insight:** Clinically relevant observations for direct application in assessment and management.

□ **Clinical Pearl:** High-yield memorable clinical points — the take-home messages most likely to change practice.

□ **Important:** Red flags, atypical presentations, and critical safety points requiring escalation or imaging.

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I. Introduction and Epidemiology

Mal de Débarquement Syndrome (MdDS) — French for 'sickness of disembarkation' — is a chronic vestibular disorder characterised by a persistent internal perception of rocking, swaying or bobbing that persists after the cessation of prolonged passive motion. Unlike most vestibular disorders, the pathology in MdDS lies not in the peripheral labyrinth but in the central vestibular adaptation networks of the brainstem and cerebellum [1,2]. The syndrome has been recognised in various forms for centuries; Hippocrates observed that 'sailing on the sea shows that motion disorders the body,' and J.A. Irwin described 'unsteady gait of sailors on land' in 1881 [1]. The modern clinical definition was crystallised by Brown and Baloh in 1987, who reported six patients with chronic motion-induced dizziness following travel — a publication that brought the syndrome into the consciousness of neuro-otology [1,13,21,42].

□ **Key Point:** MdDS is a disorder of central vestibular neuroplasticity — not a peripheral labyrinthine lesion. Peripheral vestibular function tests are expected to be normal.

Prevalence and Demographics

MdDS is considered a rare disorder in the general population, and precise incidence figures have not yet been established through large-scale epidemiological studies [2,4]. Community prevalence remains unknown. However, clinic-based data suggest it is not negligible within specialist dizziness practice: one tertiary neuro-otology centre in the United States reported that 1.3% of all dizziness patients seen over a five-year period received a diagnosis of MdDS [3]. Other specialised referral centres report figures of 1–3%, suggesting that while rare at a population level, MdDS constitutes a small but clinically significant proportion of the chronic dizziness cohort [3,32].

Demographic data consistently demonstrate a striking female predominance. Approximately 75–80% of patients with persistent MdDS are women [3,6,7]. The peak age of onset is the fourth to fifth decade — a pattern that has led to hypotheses regarding hormonal and oestrogen-related contributions to the neuroplasticity underpinning the syndrome [3,17]. Paediatric cases are extremely rare, with virtually all documented cases occurring in adults [3,17]. The oldest reported patients have been in their eighth decade, though the condition is uncommon outside the mid-adult demographic [3].

Table 1. Epidemiological Characteristics of MdDS.

Parameter	Finding	Source / Comment
Sex ratio	Female 75–80%	Consistent across case series and surveys [3,6,7]
Peak age of onset	40–50 years (mean ~44 years)	Hormonal influence hypothesised [3,17]
Clinic prevalence	1–3% of specialist dizziness patients	Tertiary neuro-otology centres [3,4]
Community prevalence	Unknown — no population studies	Likely under-estimated [2,4]
Paediatric cases	Extremely rare — isolated reports only	Adolescent cases documented [3]
Recurrence risk	Elevated after first episode	Subsequent episodes often longer [2,3]

II. Pathophysiology — Central Vestibular Maladaptation and Velocity Storage

The pathophysiology of MdDS is not fully characterised but is widely understood to involve a maladaptation of the central vestibular system's normal plasticity — specifically the velocity storage mechanism of the brainstem and cerebellum — rather than a peripheral labyrinthine lesion [5,8,16]. During prolonged passive motion (for example, a multi-day sea voyage), the vestibulo-ocular reflex (VOR)

and its velocity storage circuitry undergo normal adaptive recalibration to the oscillatory frequency of the environment, typically around 0.16–0.2 Hz for ocean swell [5,11,18]. In susceptible individuals, this adaptive state fails to extinguish upon cessation of motion — the brain remains 'locked' to the adapted oscillatory frequency, generating a persistent internal motion signal in the absence of actual environmental movement [5,8,16].

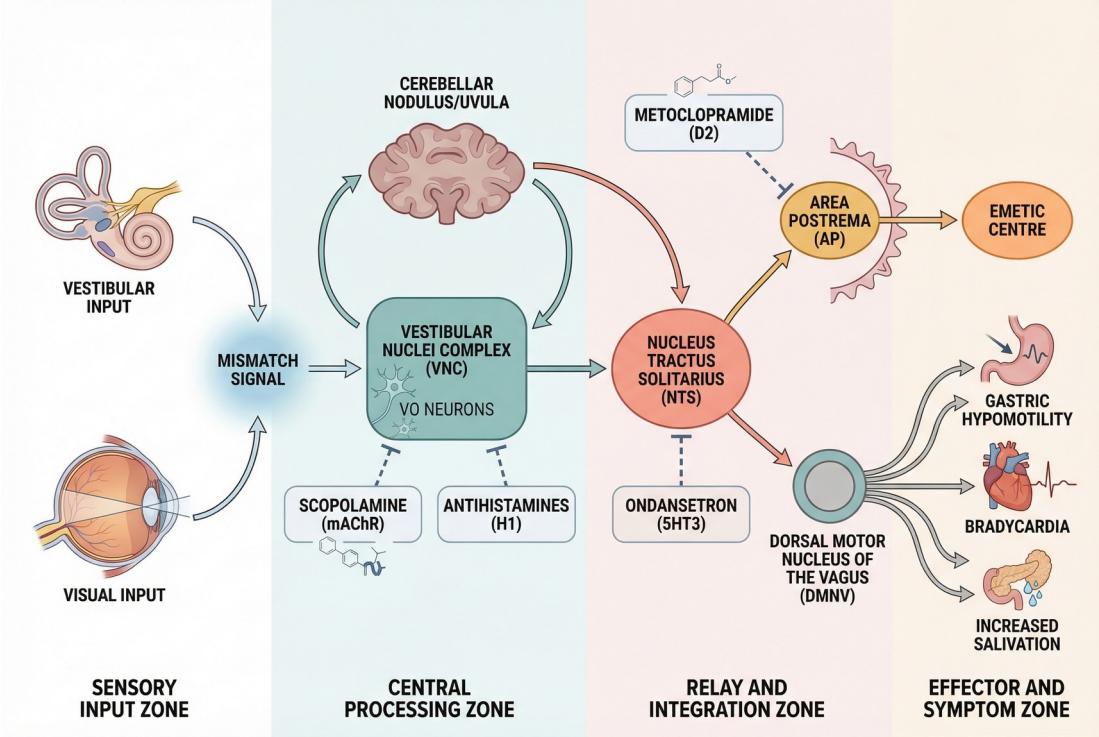

Figure 1. Central vestibular processing and the velocity-storage network implicated in MdDS.
Source: Australian Dizziness Clinics.

Experimental support for this maladaptation model was provided by Dai et al., who induced MdDS-like states in non-human primates by subjecting them to prolonged rotation — animals subsequently exhibited persistent nystagmus and postural instability consistent with entrainment of the velocity storage mechanism [11,18]. Guedry and Graybiel's classic 1962 study of compensatory nystagmus in subjects adapting to prolonged rotation in a rotating room provided early human evidence of the same principle: the visual-vestibular system adapts to sustained oscillatory input in a manner that outlasts the stimulus [15]. Maruta's 2023 work extended this to a rabbit model, demonstrating lasting alterations in spatial orientation following passive motion — further supporting a conserved mechanism across mammalian species [11,14,18,25].

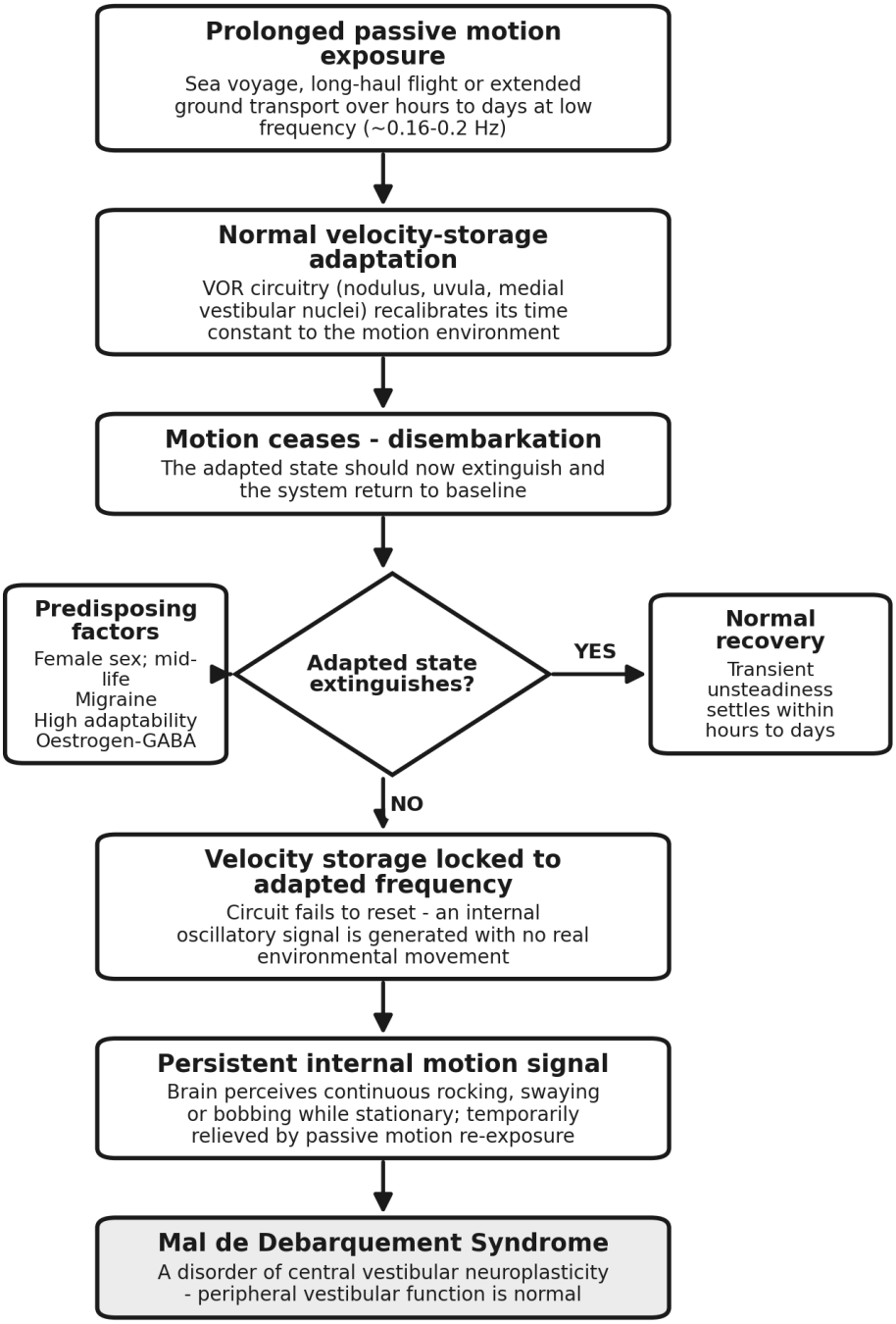

Figure 2. Pathophysiology of MdDS — Velocity Storage Maladaptation Model.
Source: Adapted from Dai et al. [11], Yakushin et al. [16], and Mucci et al. [8].

The Velocity Storage Mechanism

The velocity storage integrator — a neural circuit centred on the nodulus and uvula of the vestibulocerebellum and the medial vestibular nuclei — normally functions to extend the time constant of the VOR beyond the mechanical time constant of the semicircular canals, improving the vestibulo-ocular response to low-frequency rotational stimuli [16,18]. In MdDS, this circuit appears to retain an abnormally prolonged time constant tuned to the frequency of the preceding motion environment. Yakushin et al. (2024) demonstrated that therapeutic interventions targeting attenuation of the velocity storage mechanism — specifically through optokinetic suppression — produced measurable symptom reduction, providing mechanistic validation for the VOR readaptation therapeutic approach [16,18,22,38,41].

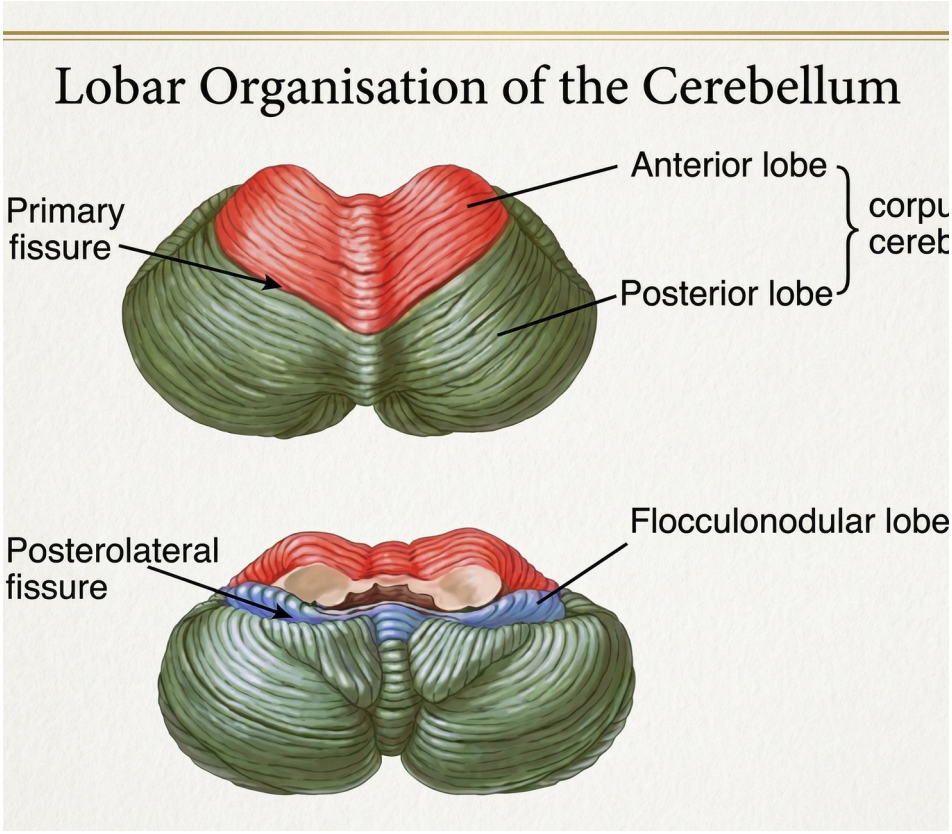

Figure 3. Lobar organisation of the cerebellum — the flocculonodular lobe (nodulus and uvula) houses the velocity-storage mechanism.

Source: OpenStax, Anatomy & Physiology (Rice University) — Creative Commons Attribution (CC BY 4.0).

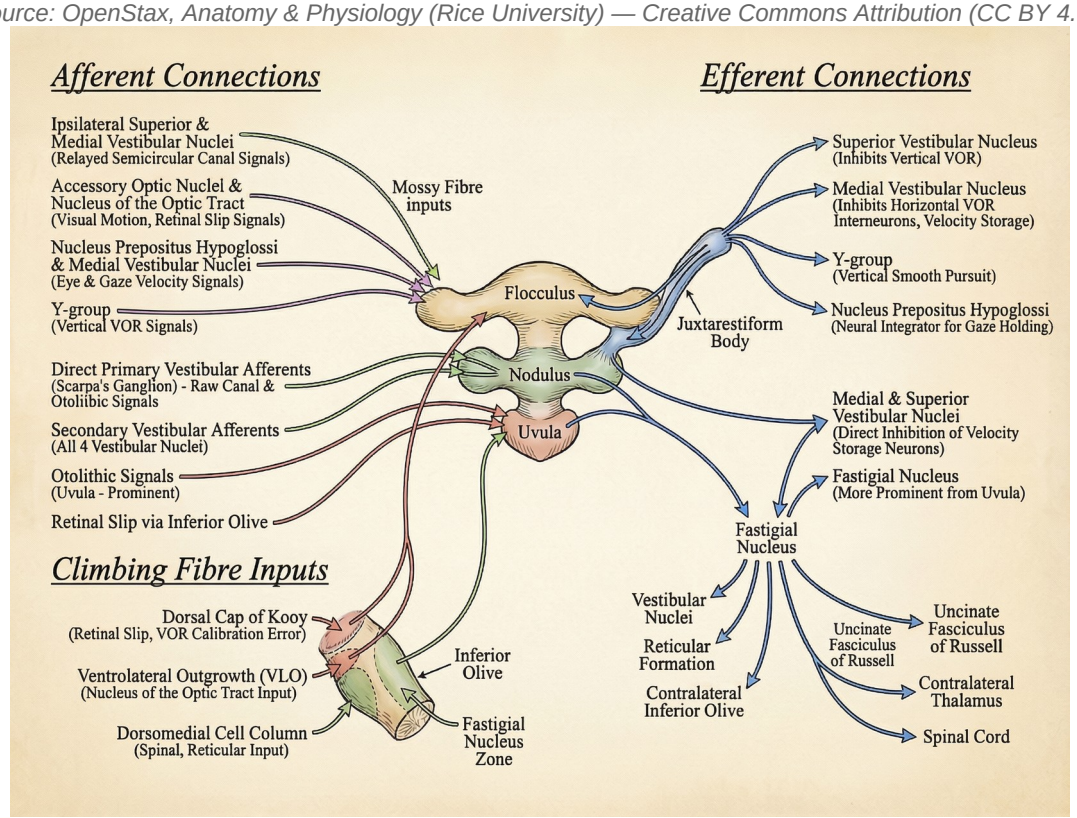

Figure 4. Afferent and efferent connections of the vestibulocerebellum that modulate velocity storage.

Source: Australian Dizziness Clinics.

□ **Clinical Insight:** The characteristic paradoxical relief of symptoms during passive motion re-exposure (driving, flying) supports the velocity storage maladaptation model: returning the patient to the frequency environment to which the system is adapted temporarily 'matches' the internal signal,

reducing the perceived mismatch and thus the symptom [5,11].

Neuroimaging Correlates

Advanced neuroimaging has begun to provide objective evidence of the central nature of MdDS. Functional MRI and PET studies demonstrate abnormal resting-state activation of the vestibulo-insular cortex and motion-sensitive visual areas in MdDS patients compared with healthy controls — a pattern consistent with the brain 'perceiving' motion even in its absence [9,19]. Resting-state connectivity analyses reveal aberrant coupling between the default mode network and vestibular processing regions, suggesting a failure of normal multisensory integration at rest [9]. Cha et al.'s metabolic and functional connectivity study (2012) identified altered glucose metabolism in regions implicated in motion processing and sensory gating, including the entorhinal cortex and parahippocampal gyrus [19]. More recently, Mucci et al.'s scoping review of neuroimaging evidence (2021) summarised that consistent findings across modalities include abnormal activity in the parieto-insular vestibular cortex (PIVC), cerebellum and visual cortex during resting states in MdDS [9,36,30].

EEG and magnetoencephalography studies have identified preliminary evidence for abnormal low-frequency oscillatory network activity in the vestibular circuit of MdDS patients, potentially corresponding to the internal oscillatory signal perceived as rocking [5]. While no single neuroimaging finding yet constitutes a diagnostic biomarker, the totality of evidence strongly positions MdDS as a disorder of central vestibular processing and neuroplasticity, not peripheral pathology [5,8,9].

Hormonal and Predisposing Factors

The striking female predominance and mid-life age distribution have prompted investigation of hormonal influences on vestibular neuroplasticity. Oestrogen receptors are present in vestibular and cerebellar circuits, and oestrogen is known to modulate GABA-ergic inhibition within the brainstem [3,17]. It is hypothesised that fluctuating or declining oestrogen levels in perimenopausal women may alter the threshold for maladaptive velocity storage entrainment. Additionally, the high comorbidity of migraine in MdDS patients (present in 20–50% in various series) suggests shared neuroplastic vulnerability — cortical spreading depression and trigemino-vestibular sensitisation may lower the threshold for vestibular maladaptation [7,17]. Cha et al.'s clinical features analysis confirmed that MdDS patients have a significantly higher prevalence of migraine than the general population [17].

□ **Clinical Pearl:** Migraine is not merely a comorbidity in MdDS — its presence may represent shared neuroplastic vulnerability. All MdDS patients should be screened for vestibular migraine, and optimal migraine management should be part of the treatment plan.

III. Clinical Features — Symptomatology, Subtypes and Associated Features

Core Symptomatology

The hallmark of MdDS is a persistent internal perception of rocking, swaying or bobbing — a non-spinning, oscillatory form of vertigo — that the patient experiences when stationary but which is temporarily relieved or abolished by passive motion [1,3,6]. Unlike acute vestibular neuritis or Ménière's disease attacks, nausea and vomiting are typically absent or mild in MdDS, a fact that can both confound the diagnosis (patients may not present it as 'vertigo') and distinguish it from peripheral disorders [1,6]. The rocking is usually perceived as a rhythmic motion in the roll plane (side-to-side), the pitch plane (forward-backward) or as a generalised 'floating on waves' sensation [3,7]. Patients may find it difficult to articulate the symptom precisely, often describing themselves as feeling like 'still on a boat' or 'walking on a trampoline' [1,3].

A defining clinical feature is the motion-contingency of symptom modulation: passive motion (riding in a car, aircraft or train) typically reduces or abolishes the rocking, which then returns when motion ceases [1,3]. This paradoxical relief is diagnostically important and pathophysiologically meaningful — it reflects

the temporary re-matching of the external motion environment to the internally 'adapted' state of the velocity storage mechanism [5,11]. Active self-generated motion (walking, exercise) does not reliably relieve symptoms and may exacerbate them, distinguishing MdDS from PPPD where voluntary movement is often a key trigger [1,3].

Subtypes: Motion-Triggered vs. Spontaneous-Onset

MdDS is clinically divided into two principal subtypes based on the presence or absence of an identifiable motion trigger [5,6,7]. Motion-triggered MdDS (MT-MdDS) — the classic and most common form — develops within hours to approximately 48 hours of the cessation of prolonged passive motion exposure, most commonly following sea travel (cruise voyages), long-haul flights or extended ground transport. The duration of the triggering motion exposure is typically many hours to days; very brief motion exposures (minutes) are insufficient to trigger the syndrome [2,3]. Sea travel represents the most frequently reported trigger, reflecting the low-frequency, multi-axis oscillatory environment of ocean swell [1,6].

Spontaneous-onset MdDS (SO-MdDS) describes clinically identical presentations in which no passive motion trigger is identifiable [7,8]. These cases are more controversial nosologically: the 2020 Bárány Society consensus classified them separately from MT-MdDS, acknowledging that they may represent a different underlying precipitant (including psychological stress, illness, surgery or other events) acting on the same neuroplastic substrate [2,5]. Cha et al.'s comprehensive clinical profile (2018) found that SO-MdDS patients tended to have longer illness durations and a somewhat higher prevalence of anxiety and autonomic symptoms compared with MT-MdDS, though core symptom features were identical [7]. Some investigators argue that SO-MdDS shares significant overlap with PPPD and that the distinction may be partly semantic — an area of active debate [5,7,8].

Table 2. MT-MdDS versus SO-MdDS — Key Comparative Features.

Feature	MT-MdDS	SO-MdDS
Motion trigger	Present (sea, air, land)	Absent — no identifiable trigger
Onset timing	Within 48h of disembarkation	Spontaneous — often insidious
Core symptoms	Rocking, swaying, bobbing	Identical to MT-MdDS
Motion-relief	Present in ~85–90%	Present but less consistent (~70%)
Migraine comorbidity	Elevated (~20–40%)	Higher (~40–50%)
Typical illness duration	Variable — often months	Often longer — years in some series
Bárány 2020 status	Formally classified	Probable SO-MdDS category
Treatment response	Similar — VOR readaptation effective	Somewhat less predictable

Associated Features

Beyond the core oscillatory vertigo, MdDS patients frequently report a constellation of associated symptoms that significantly impact quality of life [3,6,7]. Gait instability is common, particularly in visually complex environments or on compliant surfaces; patients may walk with a wider base and reach for support, though frank falls are uncommon in the absence of additional vestibular lesions [3]. Cognitive impairment — colloquially described as 'brain fog' — affects concentration and working memory, likely reflecting the continuous attentional load imposed by the unresolved motion signal and its interference with other cognitive processes [3,6]. Visual motion sensitivity is prevalent: busy visual environments (supermarkets, crowds, escalators) exacerbate symptoms, and some patients develop frank visual vertigo analogous to PPPD [3,6,7,28].

Secondary anxiety and depression are extremely common, arising as psychological responses to chronic unexplained dizziness rather than as primary causal factors [3,6,37]. Macke et al. (2012) documented the profound social, occupational and economic burden of MdDS, with many patients unable to work, drive, or engage in normal activities of daily living — contributing to significant rates of anxiety disorder and major depression [5]. Headache, fatigue, photophobia and autonomic symptoms (palpitations, postural

lightheadedness) are also frequently reported [3,7]. Hearing is not affected in MdDS; the presence of tinnitus or hearing loss should prompt investigation for concurrent pathology [1,3].

□ **Important:** Hearing loss or tinnitus is NOT part of the MdDS syndrome. If present, concurrent Ménière's disease, labyrinthitis or other inner ear pathology must be excluded. Similarly, spontaneous direction-changing nystagmus, focal neurological signs or gait ataxia warrant urgent neuroimaging to exclude a central structural lesion.

IV. Diagnostic Criteria — The 2020 Bárány Society Consensus

MdDS is a clinical diagnosis. There is no confirmatory laboratory test or imaging finding that establishes the diagnosis; rather, the diagnosis rests on a characteristic symptom profile, a compatible temporal relationship with passive motion exposure, and the exclusion of alternative explanatory conditions [1,2,3]. The Bárány Society Classification Committee published formal consensus diagnostic criteria for MdDS in 2020, providing the first internationally endorsed framework for the condition [2]. These criteria markedly improved consistency across research and clinical practice and resolved longstanding disagreements about the duration threshold required for diagnosis [1,2,3,29].

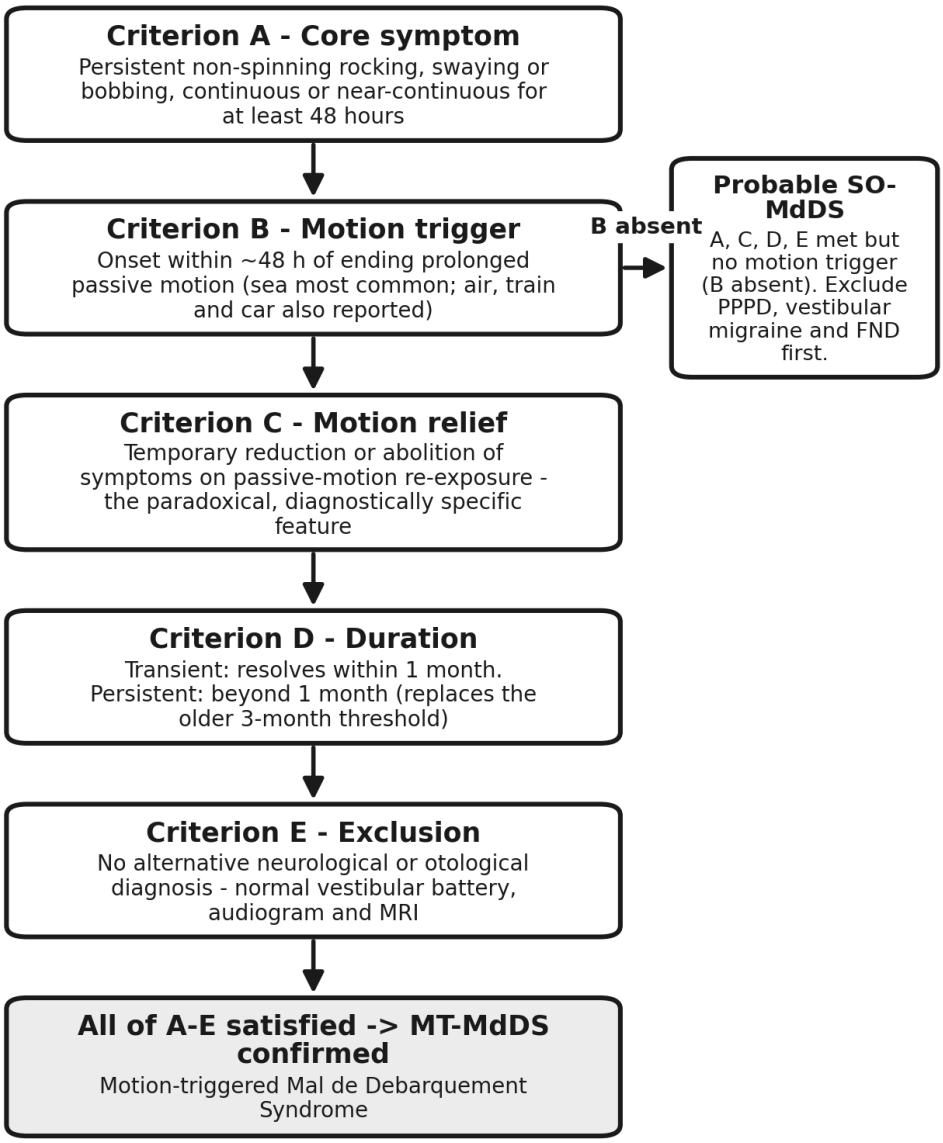

Figure 5. Bárány Society 2020 Diagnostic Criteria for MdDS.
Source: Adapted from Cha et al. [2] — Bárány Society Classification Committee consensus document.

The Five Diagnostic Criteria for MT-MdDS

Criterion A requires a persistent non-spinning oscillatory vertigo — specifically described as rocking, swaying and/or bobbing — present continuously or nearly continuously for at least 48 hours [2,3]. This duration threshold distinguishes MdDS from the normal, transient after-sensations of motion (mal de mer residual) that most individuals experience for minutes to hours following travel [1,2]. Criterion B requires that symptom onset occurs within approximately 48 hours of the cessation of a prolonged passive motion

exposure — the triggering motion having lasted hours to days, not merely minutes [2]. Criterion C requires that symptoms are temporarily reduced or abolished by re-exposure to passive motion, the hallmark paradoxical relief that is both diagnostically specific and pathophysiologically meaningful [1,2,3].

Criterion D provides a duration classification: if symptoms resolve within one month, the episode is termed 'transient MdDS'; persistence beyond one month qualifies as 'persistent MdDS' [2]. This replaces earlier three-month thresholds used in some case series and provides clearer guidance for early clinical recognition [1,13]. Criterion E requires that no other neurological, otological or medical condition better explains the symptom profile [2]. In practice, this means that a complete vestibular battery, audiogram and neuroimaging (MRI) have been obtained at initial presentation and are unremarkable [1,3].

Table 3. Bárány Society 2020 MdDS Diagnostic Criteria Summary.

Criterion	Description	Clinical Note
A — Core symptom	Persistent rocking/swaying/bobbing ≥48h, continuous or near-continuous	Non-spinning — distinguishes from episodic rotatory vertigo
B — Motion trigger	Onset within ~48h of ending prolonged passive motion exposure	Sea most common; air, train and car also reported
C — Motion relief	Temporary reduction or abolition of symptoms with passive motion	Paradoxical feature — pathophysiologically diagnostic
D — Duration	Transient: resolves <1 month; Persistent: >1 month	Replaces older 3-month threshold
E — Exclusion	No alternative neurological or otological diagnosis	Normal vestibular battery + audiogram + MRI required

Spontaneous-Onset MdDS — Diagnostic Approach

For patients who meet criteria A, C, D and E but in whom no passive motion trigger is identifiable (criterion B absent), the 2020 consensus designates a category of 'probable SO-MdDS' [2]. These patients require particularly thorough exclusion of alternative diagnoses — especially PPPD, vestibular migraine and functional neurological disorder — before SO-MdDS is applied [5,7,8]. Co-existing conditions do not preclude MdDS: a patient with established vestibular migraine may develop MdDS after a cruise, and both diagnoses may co-exist, provided neither fully explains the other's features [2,3].

□ **Clinical Pearl:** In practice, the diagnostic triad of: (1) recent prolonged travel, (2) continuous rocking dizziness, and (3) paradoxical relief with motion — in the context of normal vestibular examination — is essentially pathognomonic for MT-MdDS. Clinicians should explicitly ask about any motion exposure in the preceding two weeks when taking the dizziness history.

V. Investigations and Neuroimaging

There is no diagnostic laboratory or imaging test for MdDS; investigations are conducted primarily to exclude alternative diagnoses and to document the baseline vestibular profile of the patient [1,3]. A structured tiered approach to investigation is recommended, with the depth of work-up calibrated to the atypicality of the presentation [10,12].

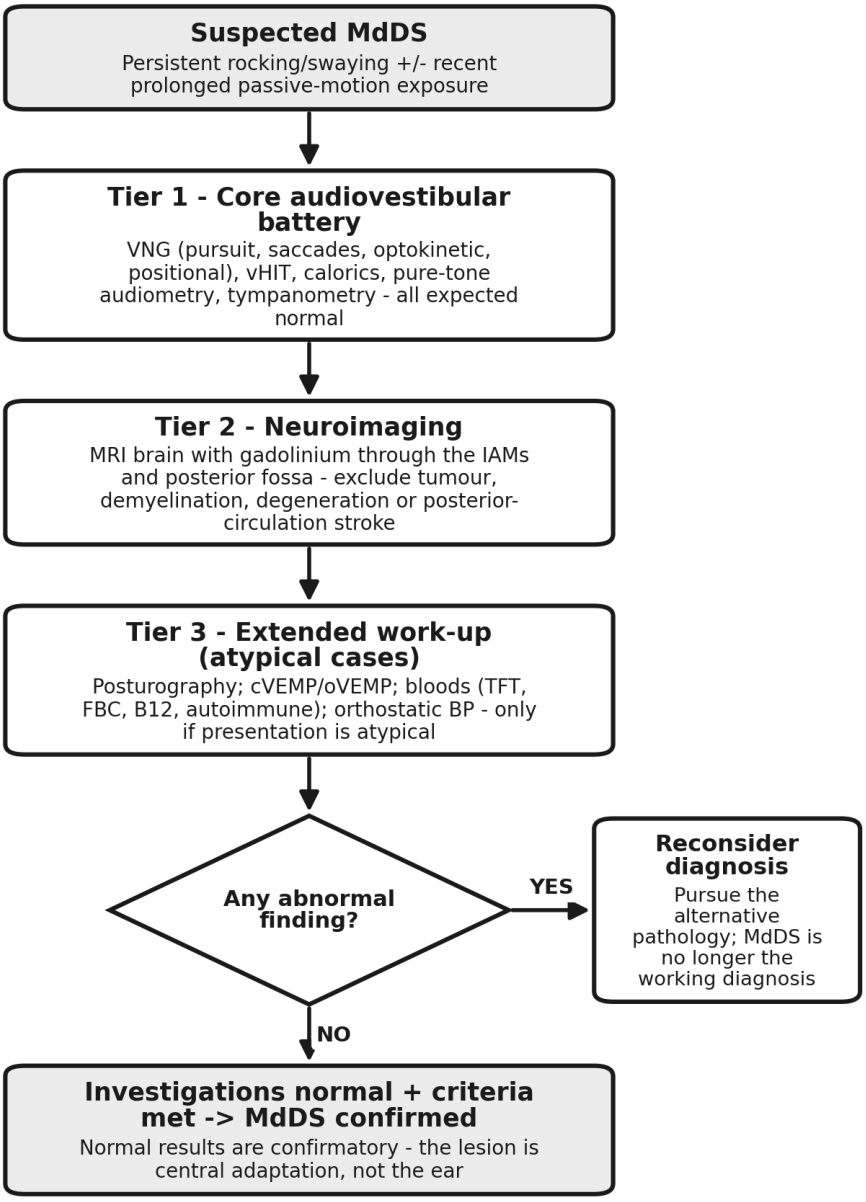

Figure 6. Investigation Algorithm in Suspected MdDS.
Source: Adapted from Saha and Fife [1], Cha et al. [2], and Schoenmaekers et al. [10].

Vestibular Function Testing

Videonystagmography (VNG) including smooth pursuit, saccades, optokinetic and positional testing is expected to be normal in MdDS [1,3]. Specifically, no spontaneous nystagmus should be present in room light, though subtle non-direction-fixed nystagmus in total darkness has been reported in a minority of cases and is not considered diagnostic [3]. Caloric testing typically demonstrates symmetric canal paresis, as the issue in MdDS is maladaptation of velocity storage rather than loss of peripheral canal function [1,16]. The video head impulse test (vHIT) is expected to show normal VOR gain bilaterally across all six canal pairs, with no corrective saccades — any abnormality should prompt reconsideration of the diagnosis [1,12]. Rotational chair testing may occasionally demonstrate a prolonged post-rotatory nystagmus time constant, reflecting heightened velocity storage activity, though this is not consistently reported [1,5].

Pure-tone audiometry and tympanometry are expected to be normal in MdDS, distinguishing it from Ménière's disease (low-frequency sensorineural hearing loss, aural fullness) and labyrinthitis (asymmetric sensorineural hearing loss in affected ear) [1,3]. Cervical and ocular vestibular evoked myogenic

potentials (cVEMP and oVEMP) are typically within normal limits and are not part of the routine diagnostic work-up unless otolith pathology is clinically suspected [12].

Neuroimaging

MRI of the brain with gadolinium contrast, including dedicated sequences through the internal auditory meati and posterior fossa, is recommended at first presentation to exclude structural causes of chronic dizziness — particularly posterior fossa tumours, demyelinating lesions, cerebellar degeneration or posterior circulation stroke [1,3,10]. In MdDS, standard structural MRI is expected to be unremarkable; the functional and metabolic changes documented by research-grade fMRI and PET are not visible on clinical sequences [9,19]. CT brain is reserved for situations where MRI is contraindicated or where bony structures require assessment.

Additional Investigations

Posturography (computerised dynamic posturography) is not mandatory but may demonstrate increased postural sway, particularly under conditions of reduced or conflicting sensory input, reflecting the internal oscillatory signal [5,10]. Some centres use posturography to document treatment response in a semi-objective manner — measuring sway frequency before and after VOR readaptation therapy. Blood tests (thyroid function, full blood count, vitamin B12, autoimmune screen) should be considered when metabolic or autoimmune causes of dizziness are clinically possible, though results are expected to be normal in true MdDS [1,3]. Orthostatic blood pressure measurement excludes orthostatic hypotension, which can coexist and should be identified and managed independently [3].

□ **Clinical Insight:** Normal results across the entire vestibular battery in a patient with classic MdDS symptoms are not frustrating — they are confirmatory. Clinicians should explicitly frame this for patients: 'the normal test results tell us that your ears are working correctly — the problem is in how the brain processed the adaptation to motion, not in the ears themselves.'

VI. Differential Diagnosis

The differential diagnosis of persistent rocking or swaying dizziness is broad but can be substantially narrowed by careful attention to trigger history, temporal pattern, response to motion, and vestibular test results [1,3,10]. The most clinically important differentials are PPPD, vestibular migraine, bilateral vestibulopathy, anxiety-mediated dizziness and posterior fossa structural pathology [2,3,24].

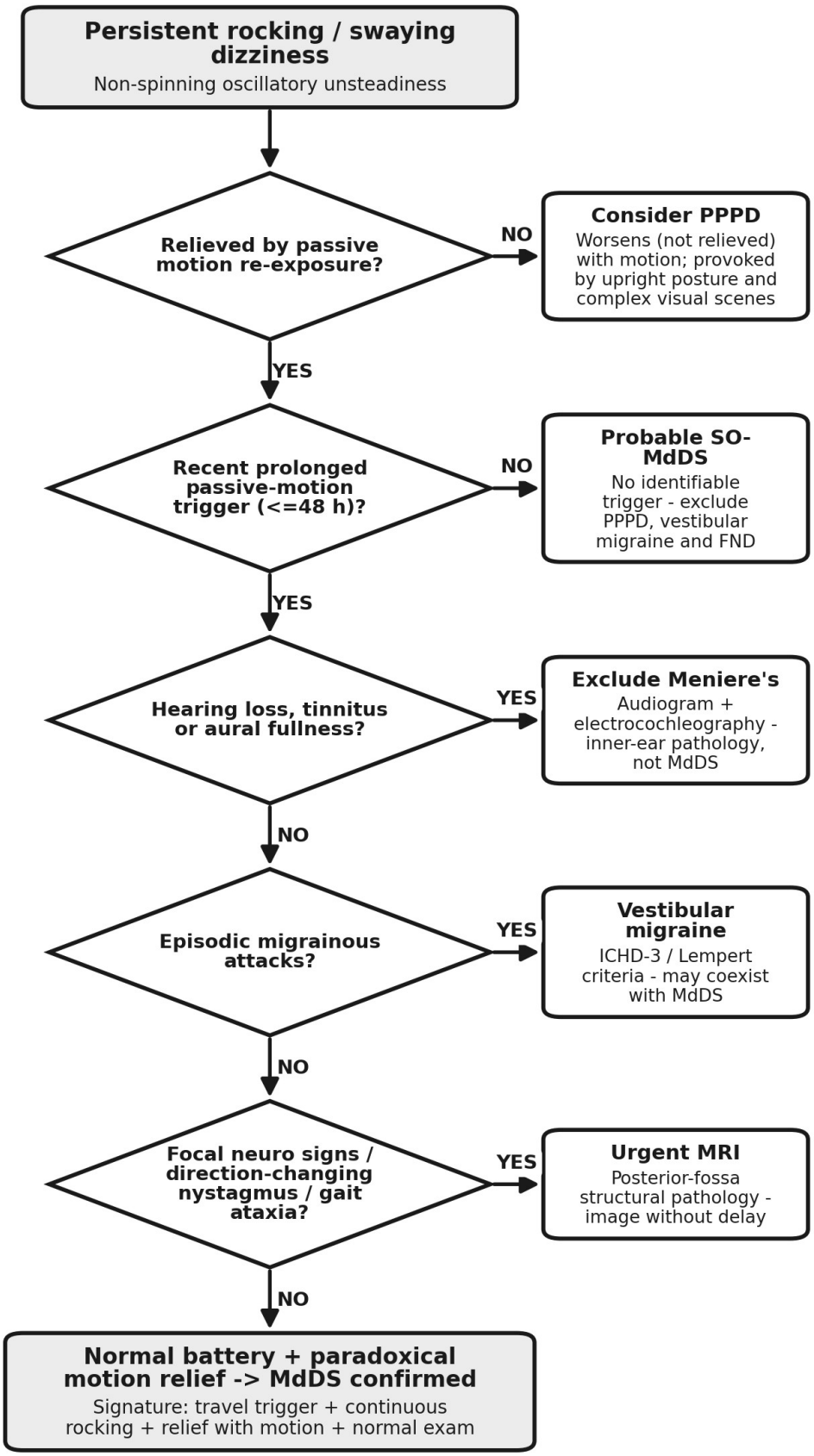

Figure 7. Differential Diagnosis Algorithm — Persistent Rocking/Swaying Dizziness.
Source: Adapted from Saha and Fife [1], Cha et al. [2], and Staab et al. [24].

Table 4. Differential Diagnosis of Persistent Rocking/Swaying Dizziness.

Condition	Key Distinguishing Features	Distinguishing Tests
PPPD	Exacerbated by upright posture, active motion and complex visual environments. NOT relieved by passive motion. Usually follows a precipitating vestibular event. No travel trigger required.	Clinical history. Staab PPPD criteria [24]. Normal VFT [24,40].
Vestibular Migraine	Episodic attacks (minutes to days) with migrainous features. May have rocking quality between attacks. Headache or photophobia/phonophobia during attacks.	ICHD-3 / Lempert criteria [23]. Clinical history.
Bilateral Vestibulopathy	Oscillopsia with head movement. Imbalance worsening in dark or on uneven terrain. Gait ataxia more prominent. No travel trigger for onset.	Bilateral caloric weakness or reduced vHIT gain bilaterally [33,34].
Ménière's Disease	Episodic spinning vertigo with sensorineural hearing loss, tinnitus and aural fullness. No travel trigger. Hearing changes are diagnostic.	Audiogram: low-frequency SNHL. Electrocochleography [43].
Anxiety / PPPD overlap	Fluctuating with emotional state. Not consistently triggered by motion. May have comorbid panic attacks. No specific trigger history.	Psychiatric assessment. Normal VFT. Response to CBT/SSRIs.
Posterior fossa pathology	Central neurological signs (dysmetria, dysdiadochokinesis, dysarthria, diplopia). Direction-changing nystagmus. Gait ataxia > dizziness perception.	MRI brain with posterior fossa sequences. Urgent if red flags present.
Functional Neurological Disorder	Variable and inconsistent presentation. Tremorogenic Romberg. May resolve with distraction. No consistent motion trigger.	Clinical assessment. FAHN criteria. Normal VFT.

□ **Important:** PPPD is the most important differential to distinguish from MdDS. The critical distinguishing feature is the motion-relief characteristic of MdDS: patients with PPPD do NOT improve with passive motion re-exposure — in fact, they often worsen with motion. This single clinical feature carries the greatest discriminatory power.

VII. Medical Management — Pharmacological Approaches

No pharmacological agent is specifically approved for MdDS, and all medication use is off-label. Pharmacotherapy in MdDS is primarily symptomatic — aimed at reducing perceived symptom severity and managing comorbidities, rather than addressing the underlying maladaptation mechanism [1,3,12]. For the majority of patients, pharmacotherapy is a bridging strategy used alongside the disease-modifying intervention of VOR readaptation therapy [10,12]. Setting appropriate expectations is paramount: medications may reduce the perceived intensity of rocking but rarely abolish it entirely [1,12].

Benzodiazepines

Benzodiazepines — particularly clonazepam and diazepam — represent the most consistently useful pharmacological class in clinical MdDS management and are the agents with the greatest anecdotal evidence base [1,3,12]. Clonazepam at a dose of 0.25–0.5 mg twice daily is most commonly employed; its longer half-life and minimal rebound effect make it preferable to shorter-acting agents [1]. The proposed mechanism of action involves enhancement of GABA-ergic inhibition within the vestibular nuclei and brainstem, dampening the aberrant oscillatory velocity storage signal [1,12]. Many patients report partial symptom relief — reduced awareness of rocking, improved functional capacity and reduced

anxiety — though the maladaptive state is not 'reset' by this mechanism [1,3]. Clinicians must balance the symptomatic benefit against risks of cognitive side effects, sedation and long-term benzodiazepine dependence, particularly in a predominantly middle-aged female population [1,12,44].

SSRIs and SNRIs

Selective serotonin reuptake inhibitors (SSRIs) and serotonin-norepinephrine reuptake inhibitors (SNRIs) are employed primarily for MdDS patients with significant concurrent anxiety or depression, or in spontaneous-onset cases where PPPD-like central sensitisation mechanisms may be operative [1,3,37]. Sertraline and venlafaxine are most commonly prescribed. There is limited evidence that these agents directly reduce the core rocking sensation in classic MT-MdDS; their principal benefit is in stabilising the psychological overlay and potentially modulating central sensitisation over time [1,3]. The overlap between SO-MdDS and PPPD means that SSRI/SNRI therapy may be more effective in the spontaneous-onset subgroup — consistent with the evidence base for these agents in PPPD [24,45].

Migraine Prophylaxis

In MdDS patients with comorbid vestibular migraine — which may affect 20–50% of the MdDS population in some series — standard migraine prophylaxis is recommended as part of the overall management plan [7,17,23]. Topiramate, amitriptyline, propranolol and nortriptyline have all been used; agent selection should be guided by the patient's headache phenotype, comorbidities and tolerability profile. There is no specific evidence that migraine prophylaxis reduces the MdDS rocking symptom per se, but optimal control of migraine burden reduces an important aggravating comorbidity and may improve overall vestibular stability [7,12].

Agents with Limited or No Evidence

Antihistamine vestibular suppressants (meclizine, dimenhydrinate) and anticholinergic agents (scopolamine) have minimal efficacy for the core chronic rocking of MdDS and are not recommended as ongoing therapy [1,3]. They may provide modest symptomatic relief acutely but do not address the maladaptation mechanism and may impair central compensation with prolonged use [1]. Calcium channel blockers (verapamil), anti-epileptic agents (topiramate, lamotrigine) and beta-blockers are occasionally used in specific comorbidity contexts but lack specific MdDS evidence [1,12]. Gabapentin and memantine have been trialled anecdotally without convincing evidence [12].

Table 5. Pharmacological Options in MdDS — Evidence Summary.

Agent/Class	Typical Dose	Target	Evidence Level	Key Caution
Clonazepam	0.25–0.5 mg BD	Symptomatic relief of rocking	Best available (anecdotal/case series)	Dependence, sedation, cognitive effects
Diazepam	2–5 mg PRN or daily	Symptomatic relief	Case reports [1]	Short-term use preferred
Sertraline / Venlafaxine	Variable (standard doses)	Comorbid anxiety / depression / PPPD overlap	Limited direct MdDS evidence [1,3,37]	Not for core rocking — adjunct only
Amitriptyline / Topiramate	Standard migraine doses	Migraine comorbidity	Migraine evidence; MdDS indirect [7,23]	Tolerability; titrate slowly
Meclizine / Antihistamines	Not recommended long-term	Acute nausea (minimal in MdDS)	Minimal benefit [1,3]	Risk of compensation suppression

VIII. Non-Pharmacological, Rehabilitation and Neuromodulation Strategies

Non-pharmacological interventions represent the principal disease-modifying treatment approach in MdDS, with the VOR readaptation protocol of Dai et al. constituting the most evidence-supported strategy

currently available [10,11,12]. Traditional vestibular rehabilitation exercises (habituation-based, Epley-derived gaze stabilisation protocols) have generally proven ineffective in MdDS and may worsen symptoms in some patients — a critical point of difference from standard peripheral vestibular rehabilitation [1,3,12].

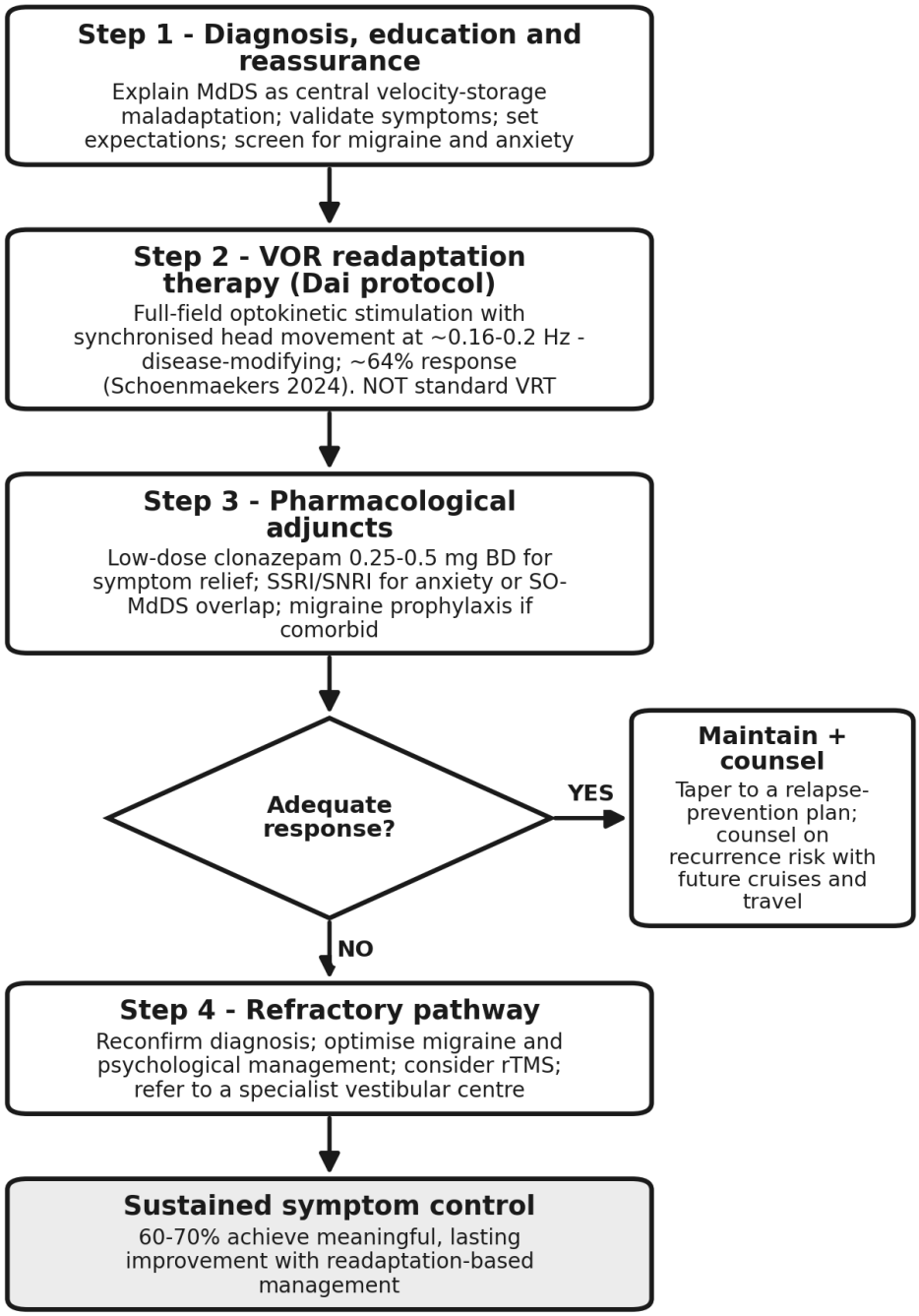

Figure 8. Management Algorithm — Mal de Débarquement Syndrome.
Source: Adapted from Schoenmaekers et al. [10], Dai et al. [11], and Kinkhabwala et al. [12].

VOR Readaptation Therapy — The Dai Protocol

The VOR readaptation protocol, developed by Dai, Cohen, Smouha and Cho, exploits the same neuroplastic mechanism responsible for generating MdDS to drive therapeutic readaptation [11]. The patient is seated before a full-field optokinetic drum or screen displaying moving visual patterns, while the clinician or a mechanical device induces synchronised rhythmic head movements in the yaw or roll plane at a specific frequency (typically ~0.16–0.2 Hz, matching the frequency of the adapted velocity storage

mechanism) [10,11]. This combination of visual optokinetic stimulation and head movement at the target frequency is designed to extinguish the maladaptive velocity storage state — essentially re-exposing the vestibular system to the responsible motion pattern in a controlled environment that facilitates re-adaptation [11,16].

Sessions typically last 45–90 minutes and are conducted either intensively over three to five consecutive days or as weekly outpatient sessions over several weeks [10]. Schoenmaekers et al.'s 2024 guideline paper, based on 131 MdDS patients, reported a 64% meaningful treatment response rate — defined as substantial, sustained symptom reduction — making this the largest evidence base for any MdDS therapeutic intervention [10]. Responders typically note a reduction in the perceived intensity and persistence of the rocking within the first one to three sessions, and maintained improvement on follow-up [10,11]. The protocol requires specialised equipment and trained clinicians, limiting its availability outside tertiary vestibular centres [12].

□ **Clinical Pearl:** Standard vestibular rehabilitation exercises (gaze stabilisation, habituation) are NOT appropriate first-line therapy for MdDS — they are designed for peripheral vestibular hypofunction, not central maladaptation. Referring an MdDS patient for standard VRT without the Dai protocol may be ineffective or counterproductive.

Cognitive-Behavioural and Psychological Interventions

Cognitive-behavioural therapy (CBT) adapted for chronic vestibular disorders has been applied to MdDS, particularly for patients with significant anxiety, avoidance behaviour or functional overlay [37]. CBT addresses maladaptive illness cognitions, reduces avoidance of vestibular triggers and treats comorbid anxiety and depression [37]. While evidence specific to MdDS is limited, the substantial psychological burden of the condition — and the frequent diagnostic delay (mean delay of four or more years in some series) — means that psychological support is a clinically important adjunct [6,37]. Mindfulness-based approaches and interdisciplinary vestibular rehabilitation combining psychological and physical therapy components have shown utility in related chronic dizziness conditions [37,31,26].

Neuromodulation — Repetitive Transcranial Magnetic Stimulation

Repetitive transcranial magnetic stimulation (rTMS) has been investigated as an intervention for refractory MdDS, based on the hypothesis that non-invasive cortical stimulation may modulate the aberrant vestibular network activity responsible for persistent symptoms [20,39]. Özgen et al.'s 2016 pilot study demonstrated symptom improvement in a small cohort of MdDS patients treated with rTMS directed at the posterior parietal cortex [20]. Cha and colleagues subsequently conducted a double-blind sham-controlled crossover trial, finding meaningful symptom improvement in the active rTMS arm compared with sham stimulation [39]. However, sample sizes in existing trials are small, protocols vary considerably across centres, and rTMS is not yet a routinely available option outside specialist research settings [39]. It remains a promising emerging therapy for refractory or severe cases where VOR readaptation therapy has been insufficient.

□ **Clinical Insight:** For patients who do not have access to a specialist VOR readaptation programme, a reasonable interim approach combines: (1) low-dose clonazepam for symptomatic relief; (2) optimisation of migraine management; (3) CBT for anxiety and avoidance; and (4) patient education about the expected natural history and the availability of specialist readaptation therapy.

IX. Prognosis, Recurrence and Predisposing Factors

Natural History and Prognosis

The natural history of MdDS is highly variable and remains incompletely characterised due to the absence of large prospective cohort studies [2,3]. A proportion of MT-MdDS episodes — particularly shorter-duration cases — will resolve spontaneously, often within weeks to a few months of onset [1,2]. However,

the probability of spontaneous remission decreases substantially with increasing illness duration; persistent MdDS lasting beyond six months to one year becomes progressively less likely to remit without intervention [2,3]. The 2020 Bárány consensus distinguishes 'transient MdDS' (resolution within one month) from 'persistent MdDS' (duration exceeding one month), with the latter carrying a more guarded prognosis for spontaneous resolution [2].

With appropriate therapy — specifically VOR readaptation protocols — prognosis improves significantly, with 60–70% of patients achieving meaningful and sustained symptom reduction [10,12]. Kinkhabwala et al.'s (2023) scoping review of treatment options confirmed that the readaptation approach consistently outperforms pharmacotherapy alone or standard VRT in terms of response rate and sustained benefit [12]. However, complete elimination of symptoms remains achievable in a minority: many patients achieve partial improvement with residual low-grade symptoms that are manageable with adaptive strategies [10,12].

Recurrence

A clinically important feature of MdDS is the elevated risk of recurrence following initial remission, particularly with repeated motion exposures [2,3,6]. Patients who have experienced one MdDS episode are at significantly higher risk of developing the syndrome again after subsequent travel [2,3]. Critically, recurrent episodes have been observed to be of longer duration than the first, a pattern with important implications for counselling [2,3]. Clinicians should advise patients in remission that further cruise voyages or extended travel carries meaningful recurrence risk, and that early treatment initiation — if symptoms do recur — is likely to yield better outcomes than delayed intervention [2,10].

□ **Clinical Insight:** Counsel patients in remission that subsequent prolonged passive motion exposures (particularly cruises) carry a significant recurrence risk. Some patients choose to avoid cruise travel indefinitely after an MdDS episode; others elect to travel with a pre-arranged action plan for early readaptation therapy should symptoms recur.

Predisposing Factors and Special Populations

Several factors appear to predispose individuals to developing MdDS following motion exposure or to having a more refractory course [3,6,7]. Female sex and midlife age are the most consistently identified demographic risk factors, implicating hormonal modulation of vestibular neuroplasticity [3,17]. A personal or family history of migraine is a significant risk factor, appearing in 20–50% of MdDS patients across series and suggesting shared neuroplastic vulnerability [7,17]. Individuals with a history of motion sickness tolerance (paradoxically, good adaptors who experienced little or no seasickness during the triggering voyage) appear to be over-represented in MdDS cohorts, consistent with the hypothesis that high vestibular adaptability predisposes to the syndrome of maladaptation [3,27].

Anxiety and depression — whether pre-existing or arising secondary to MdDS — are associated with greater illness duration and more refractory symptoms [3,6,37]. They should be actively identified and managed as part of a comprehensive treatment plan. SO-MdDS patients in some series have longer mean illness durations than MT-MdDS patients, which may in part reflect delayed or missed diagnosis — an important quality-of-care consideration [7].

Table 6. Prognostic Factors in MdDS — Summary.

Factor	Impact on Prognosis	Clinical Implication
Illness duration at presentation	Longer duration → less likely spontaneous resolution	Treat early — delay worsens prognosis
Migraine comorbidity	Associated with more refractory course	Optimise migraine management in all MdDS patients
Spontaneous onset (SO-MdDS)	Typically longer illness duration in series	Consider earlier referral to specialist centre
Significant anxiety / depression	Worsens functional impairment and illness course	Integrated psychological management essential
Prior MdDS episode	Elevated recurrence risk —	Counsel re: travel risk; plan early

	subsequent episodes longer	intervention
Access to readaptation therapy	Significantly improves prognosis (60–70% response)	Refer to specialist VOR readaptation programme

X. Controversies, Guidelines and Future Directions

Nosological Controversies

The classification of MdDS — particularly the boundary between SO-MdDS and PPPD — remains one of the most actively debated issues in chronic dizziness nosology [5,7,8]. The 2020 Bárány consensus defined MdDS strictly around the motion trigger criterion, relegating spontaneous cases to a 'probable' category; some researchers argue that this definition is unnecessarily restrictive and that SO-MdDS shares the same neurobiological substrate and responds to identical therapies, warranting inclusion within the core diagnostic category [5,7]. Others maintain that the motion trigger is a crucial pathophysiological distinguishing feature — not merely a clinical detail — and that its absence should prompt more serious consideration of PPPD, functional neurological disorder or psychiatric dizziness [5,8,24].

The appropriate duration threshold for diagnosis has historically been debated: some earlier case series required three months of symptoms, while the Bárány consensus lowered this to 48 hours (minimum) and one month (transient vs. persistent delineation) [1,2]. This shift enables earlier diagnosis and intervention — clinically important given that delayed diagnosis has historically been associated with poorer prognosis [2,10]. The under- and over-diagnosis dilemma is also acknowledged: increasing awareness risks non-specific application of the label to any post-travel dizziness, while historical under-recognition has contributed to diagnostic delays of four or more years in many patients [10,46].

Lack of Objective Diagnostic Biomarkers

A major ongoing challenge is the absence of validated objective biomarkers for MdDS [2,10]. Unlike BPPV (Dix-Hallpike) or Ménière's disease (audiogram, electrocochleography), MdDS has no confirmatory clinical test. Proposed biomarker candidates include characteristic sway frequency profiles on posturography (~0.2 Hz sway), resting-state fMRI connectivity patterns, and EEG oscillatory signatures — but none has achieved sufficient sensitivity or specificity for routine diagnostic use [5,9,36]. The development of a validated objective diagnostic marker remains a high-priority research target, as it would enable earlier diagnosis, more rigorous study enrolment, and objective monitoring of treatment response [10].

□ **Clinical Pearl:** The absence of a diagnostic biomarker for MdDS is not a diagnostic dead end — it is a clinical opportunity. The diagnostic 'test' is a meticulous history: the temporal relationship with travel, the paradoxical relief with passive motion, and the normal vestibular battery constitute a highly specific clinical signature that experienced vestibular physicians can recognise reliably.

Therapeutic Controversies and Future Directions

While the Dai VOR readaptation protocol represents the current therapeutic standard for MdDS, it requires specialised equipment, trained personnel and is not yet widely available. The development of home-based or telehealth-adapted optokinetic readaptation protocols is an area of active investigation [10,12]. The role of rTMS as a viable clinical alternative or adjunct to readaptation therapy requires larger randomised controlled trials; current evidence is preliminary but promising [20,39]. Pharmacological development is limited by the absence of validated animal models that replicate the human MdDS phenotype reliably — a significant barrier to drug discovery [10,12,47].

Relapse prevention represents a significant unmet need: current management does not include validated preventive strategies for patients planning future motion exposure [2,10]. Whether pre-exposure benzodiazepine administration, pre-emptive optokinetic priming or selective travel avoidance can reduce relapse rates is unknown and warrants prospective study [10,12]. The intersection of MdDS with

vestibular migraine — both in terms of shared neurobiology and the question of whether migraine prophylaxis reduces MdDS incidence or severity — is a promising but understudied area [7,17]. International registries of MdDS patients, coordinated through the Bárány Society and patient advocacy organisations, are being established to address the longstanding limitations imposed by small sample sizes in individual centre studies [10,35].

□ **Clinical Insight:** The 2024 Schoenmaekers et al. standardised treatment guideline represents the most current evidence-based framework for MdDS management. Vestibular physicians treating MdDS should familiarise themselves with this guideline, as it provides the most comprehensive and up-to-date treatment algorithm currently available.

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