

# **Motion Sickness Susceptibility:** **A Vestibular Physician's Deep Review of Neural Mechanisms, Clinical Assessment, and Therapeutic Management**

## **Vestibular Medicine for Vestibular Physicians**

Vestibular Science and Special Topics — Module 8.1

Australian Dizziness Clinics | [www.AustralianDizzinessClinics.com](http://www.AustralianDizzinessClinics.com)

Version 1.0 | June 2026

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

Motion sickness susceptibility (MSS) occupies a uniquely cross-cutting position in vestibular medicine: it is a heritable biological trait, a prognostic variable, a treatment modifier, and a bridge between vestibular migraine, PPPD, MdDS, and post-concussive dizziness. This review synthesises the mechanistic, epidemiological, and therapeutic evidence base across ten structured sections. It is 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 patient information resources 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.

# Table of Contents

- I. Introduction and Epidemiology**
  - II. Pathophysiology — Sensory Conflict, Velocity Storage, and Brainstem-Cerebellar Integration**
  - III. Neural Mediators and Neurochemistry of Motion Sickness**
  - IV. Genetics, Heritability, and Biological Risk Factors**
  - V. Clinical Assessment — History, Questionnaires, and Provocation Testing**
  - VI. Motion Sickness Susceptibility in Vestibular Disorders**
  - VII. Non-Pharmacological Management**
  - VIII. Pharmacological Management**
  - IX. Special Populations, Occupational Contexts, and Cybersickness**
  - X. Controversies, Future Directions, and Research Frontiers**
- References**
- Disclaimer and Copyright**

# I. Introduction and Epidemiology

Motion sickness susceptibility (MSS) is a quantifiable psychophysical trait that captures the propensity of an individual's nervous system to generate nausea, emesis, and autonomic dysregulation in response to real or apparent motion [1,2]. Unlike most vestibular diagnoses, MSS is not a disease state but a biological parameter — normally distributed across the population, heritable, and modified by exposure history and clinical context [1,3]. It is precisely measured using validated questionnaires and is reproducible across sessions and assessors [2,15].

For the vestibular physician, MSS occupies a uniquely cross-cutting position. It is a premorbid trait that predisposes to vestibular migraine, amplifies symptom burden in persistent postural-perceptual dizziness (PPPD), predicts poor tolerance of provocative vestibular function tests, and governs treatment response across pharmacological and rehabilitative modalities [4,5,6,7,8]. Quantifying MSS is therefore not ancillary — it is a core clinical variable that should be documented at first assessment for every patient in a vestibular physician's practice.

## Epidemiology

The point prevalence of clinically relevant MSS — defined as moderate-to-severe symptoms on the MSSQ-Short in any of the nine transport modalities — in unselected adult populations is approximately 33–40% [1,2,4]. The condition is near-universal in childhood (peak incidence 3–12 years), declines steeply through adolescence, and stabilises in adulthood with a second gradual decline in later life [2,4,7]. Active nausea-and-vomiting motion sickness is less prevalent than historical MSS: approximately 10–15% of adults report severe symptoms on real-world transport exposure in any given year [1,4]. The clinical relevance of this epidemiology lies in recognising that high MSSQ-Short scores are common in vestibular clinic populations and should be expected rather than treated as incidental [8,11].

Sex modulates MSS throughout reproductive life: women consistently score 25–40% higher than men on the MSSQ-Short, with the disparity greatest between 20 and 45 years of age [2,8,13]. Hormonal cycles, oral contraceptive use, and pregnancy each independently elevate MSS scores, implicating the gonadal hormone axis as a susceptibility modulator [9,13]. After the sixth decade, the sex gap narrows, likely reflecting declining oestrogen levels and concurrent reduction in vestibular processing gain [2,10]. Age-related reduction in MSS has been partially attributed to physiological shortening of the velocity storage time constant with advancing age [14,38].

□ **Key Point:** MSS is a normally distributed, heritable trait measurable with validated questionnaires. Prevalence is 33–40% in adults; it is near-universal in children and declines with age. Women are 25–40% more susceptible than men across reproductive years. [1,2,4]

**Table 1. Epidemiology and established risk factors for motion sickness susceptibility.**

Factor	Magnitude of Effect	Clinical Notes
<b>Female sex (20–45 yrs)</b>	MSSQ 25–40% higher than males	Hormonal axis implicated; narrows after menopause [2,13]
<b>Childhood MSS</b>	70%+ VM patients vs 20–30% controls	Strongest historical predictor of vestibular migraine [12,49]
<b>Migraine family history</b>	OR 2.1–3.4 vs non-migraineurs	Shared brainstem sensitisation circuitry [7,42]
<b>Anxiety / panic disorder</b>	MSSQ significantly elevated	NTS–LC arousal loop amplifies susceptibility [25,41]
<b>OCP / pregnancy</b>	Acute MSS elevation	Oestrogen sensitises cholinergic pathways [9,13]
<b>PPPD diagnosis</b>	VIMSSQ very high	Single latent factor with PPPD severity score [45]
<b>Age &gt; 65 years</b>	Protective — MSS scores fall	Velocity storage time constant shortens [14,38]

<b>Bilateral vestibulopathy (complete)</b>	Paradoxical immunity	Velocity storage signal abolished [14,50]
--	----------------------	---

## II. Pathophysiology — Sensory Conflict, Velocity Storage, and Brainstem-Cerebellar Integration

The sensory conflict (neural mismatch) model, formalised by Reason and Brand in 1975 [1], remains the most clinically and experimentally supported explanatory framework for motion sickness. The model proposes that the brainstem–cerebellar comparator continuously computes a predicted sensory state based on prior experience — an internal model or efference copy of expected vestibular, visual, and somatosensory inputs during movement. Motion sickness arises when the actual pattern of incoming signals diverges from this prediction [1,11]. The severity of sickness is proportional to the degree and duration of the mismatch, and susceptibility reflects the individual's threshold and tolerance for unresolved conflict [2,5,11].

### Visual-vestibular conflict and the dominant clinical phenotypes

Two canonical conflict configurations account for most clinical presentations. In active motion with visual suppression — reading in a moving vehicle, operating in a vibrated environment — the vestibular system signals motion that the stabilised visual scene does not confirm [1,11]. In passive visual motion without physical movement — scrolling interfaces, cinema, VR headsets — the optic flow signals movement that the immobile labyrinth contradicts [12,39]. The former characterises classical MSS; the latter underpins visually induced motion sickness (VIMS), which has emerged as the dominant phenotype in vestibular clinic practice over the past decade [15,27]. Both phenotypes can coexist in the same patient and are captured by different subscales of the MSSQ-Short and VIMSSQ-Short respectively [15,45].

### Velocity storage and the nodulo-uvular gate

The velocity storage mechanism — a brainstem integrator extending the duration of the vestibular low-pass signal beyond the mechanical decay time of the semicircular canal cupula — is the principal neurophysiological substrate linking canal stimulation to prolonged motion sickness [14,22]. The time constant of velocity storage (TV) in susceptible individuals characteristically exceeds 15–20 seconds; pharmacological or habituation-induced shortening of TV reliably reduces motion sickness severity [14,22]. The longer the TV, the greater the duration over which the mismatch signal persists at the NTS level, and the more severe the cumulative autonomic response [14].

The nodulus and uvula of the vestibulocerebellum provide the primary inhibitory control over velocity storage [14,22]. Nodular lesions — infarction, demyelinating plaques, tumour — disrupt this gate, producing pathological prolongation of velocity storage and exaggerating motion sickness responses. This is a clinically important observation: the patient with unexpected worsening of MSS in middle age should prompt cerebellar imaging to exclude structural nodular pathology. Single-unit studies in non-human primates confirm that vestibular-only neurons in the vestibular nuclei, under tonic nodular inhibition, encode the velocity storage signal; stimulation of the nodulus shortens TV and attenuates nausea-equivalent behaviours [14,22].

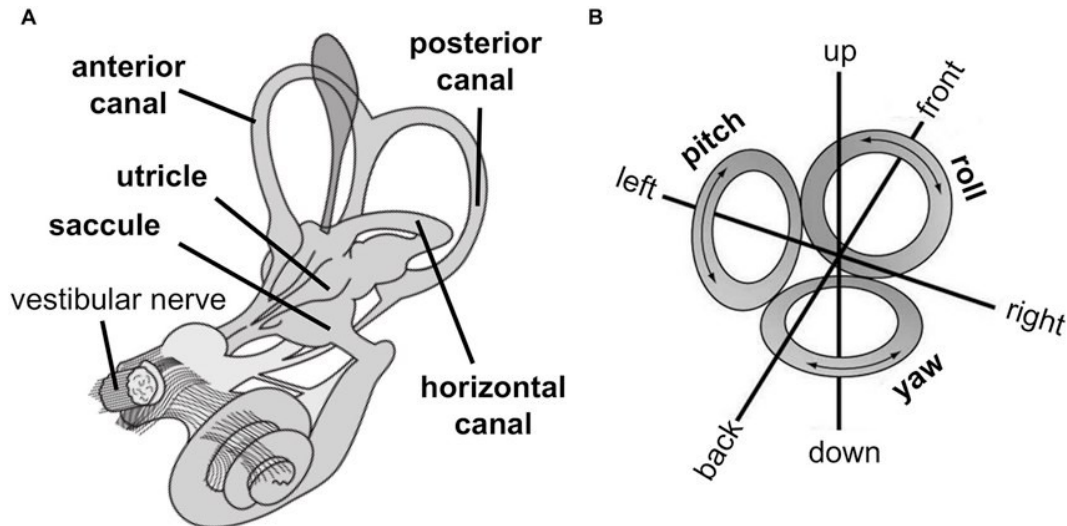


Figure 1. The peripheral vestibular apparatus — the three semicircular canals detect rotational acceleration in the pitch, roll and yaw planes, while the otolith organs (utricle and saccule) detect linear acceleration and gravity. These afferents form the vestibular limb of the sensory-conflict model.

Source: Pfeiffer C, Serino A, Blanke O. *Front Integr Neurosci* 2014;8:31. CC BY 3.0, via Wikimedia Commons.

## Sensory Conflict Cascade and Velocity Storage Circuit

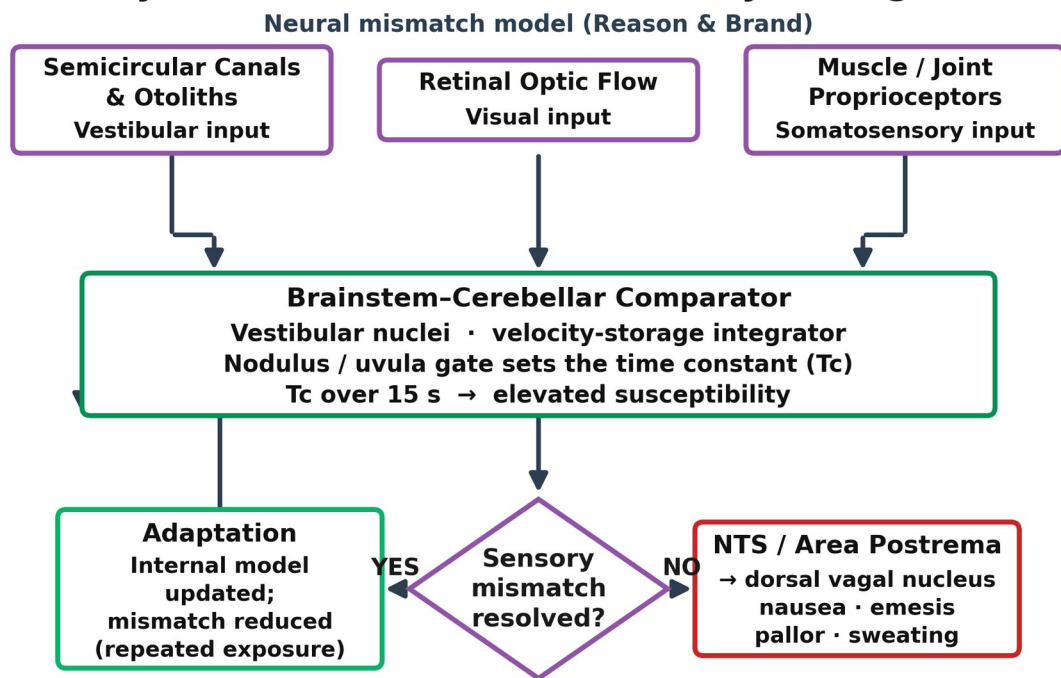


Figure 2. Sensory conflict cascade and velocity storage circuit — the three afferent streams converge on a brainstem–cerebellar comparator; unresolved mismatch triggers the autonomic-emetic efferent cascade.  $T_c$  = velocity storage time constant; NTS = nucleus tractus solitarius.

Source: Adapted from Reason and Brand [1], Schmä [6], and Maruta [14].

### Autonomic efferents and the emetic pathway

The transition from sensory mismatch to clinical sickness follows a predictable cascade through defined brainstem circuits. The nucleus tractus solitarius (NTS) and the area postrema — the classical chemoreceptor trigger zone, accessible to blood-borne emetic agents — receive afferent vestibular input via the direct vestibulo-NTS pathway [23,24]. The NTS projects to the dorsal motor nucleus of the vagus, producing the parasympathetic efferents responsible for increased salivation, gastric hypomotility, and nausea [23]. Central projections to the hypothalamus mediate the thermoregulatory and cardiovascular autonomic components (pallor, sweating, hypotension) [6,23]. The vomiting reflex itself is coordinated by

the lateral tegmental field pattern generator — the classical "vomiting centre" — which receives convergent input from the NTS, area postrema, and higher cortical structures [6,21].

### The migraine brainstem overlap

Motion sickness and migraine share central circuitry in ways that are directly clinically relevant. Trigeminal-vestibular projections, serotonergic modulation through 5-HT<sub>3</sub> and 5-HT<sub>4</sub> receptors, and histaminergic H<sub>1</sub> pathways are common to both conditions [6,24,42]. The dopaminergic D<sub>2</sub> receptor in the area postrema modulates nausea threshold and may represent the mechanistic link through which D<sub>2</sub> antagonists (domperidone, prochlorperazine) attenuate motion sickness in migraineurs [24]. Calcitonin gene-related peptide (CGRP) release during migraine attacks sensitises central vestibular processing, directly explaining why MSSQ-Short scores worsen during and immediately after migraine episodes [42,49].

□ **Clinical Insight:** Unexpected new-onset or worsening MSS in a patient over 50 should prompt cerebellar MRI. Structural nodular pathology (infarction, demyelination) disrupts velocity storage gating and produces disproportionate motion sickness. [14,22]

## III. Neural Mediators and Neurochemistry of Motion Sickness

Understanding the neuropharmacology of motion sickness is prerequisite to rational pharmacotherapy. Five receptor systems are clinically actionable, each with distinct anatomical substrates and pharmacological leverage points [5,6,23,24].

### Muscarinic (M1/M3) receptors

Muscarinic acetylcholine receptors mediate nausea and vomiting through projections from the vestibular nuclei to the NTS and from the NTS to the dorsal vagal nucleus [23,29]. Anticholinergic agents — most potently scopolamine (hyoscine) — suppress these projections and remain the most effective single pharmacological intervention for motion sickness prophylaxis [16,29]. The muscarinic pathway is also modulated by the acetylcholine interneurons within the cerebellum, providing a cerebellar–cholinergic interface that may explain why anticholinergics also partially attenuate velocity storage prolongation [22,23].

### Histaminergic H1 receptors

Histamine H<sub>1</sub> receptors in the medial and superior vestibular nuclei, the NTS, and the periaqueductal grey gate the transmission of labyrinthine signals toward the emetic centre [6,32]. First-generation H<sub>1</sub> antihistamines — promethazine, cinnarizine, dimenhydrinate, cyclizine — cross the blood-brain barrier and reduce this gating, accounting for their well-documented motion sickness efficacy [32]. Second-generation, non-sedating antihistamines (cetirizine, fexofenadine) have negligible CNS penetration and are ineffective for motion sickness, an important prescribing distinction [4,32].

### Dopaminergic D2 receptors

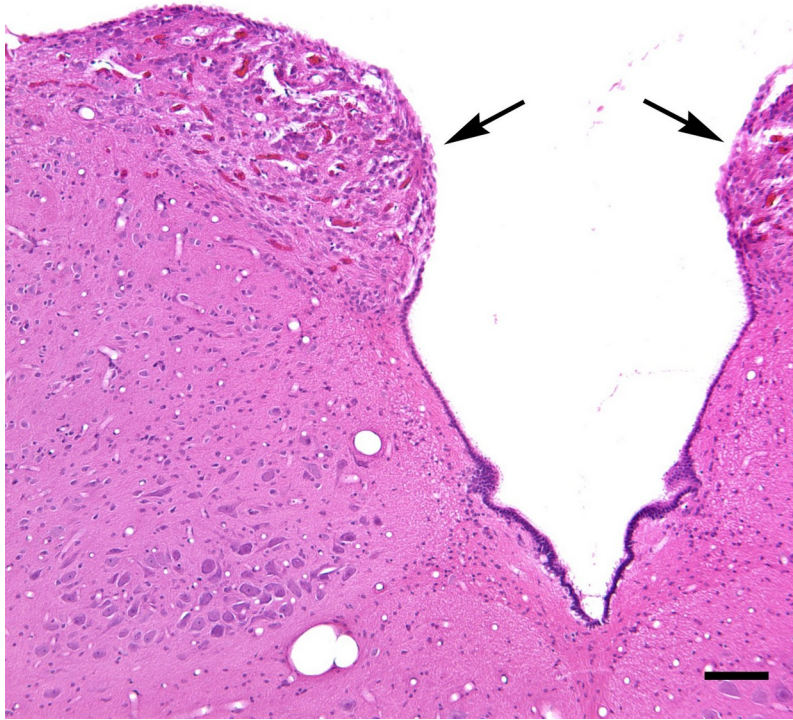
Dopaminergic D<sub>2</sub> receptors in the area postrema modulate the emetic trigger threshold. D<sub>2</sub> antagonists (metoclopramide, prochlorperazine, domperidone) are useful adjuncts, particularly in subjects with migraine comorbidity where central dopamine dysregulation amplifies nausea [24]. The clinical corollary is important: in a vestibular migraine patient whose motion sickness episodes are nausea-dominant and incompletely controlled by antihistamines, adding a D<sub>2</sub> antagonist targets a parallel emetic pathway and may provide superior symptom control [24,42].

### Serotonergic and noradrenergic pathways

Serotonergic 5-HT<sub>3</sub> receptors in the NTS and vagal nerve efferents modulate emesis broadly; 5-HT<sub>3</sub> antagonists (ondansetron) are highly efficacious for postoperative and chemotherapy-induced nausea but demonstrate only weak efficacy for motion sickness, suggesting that the vestibular-emetic pathway relies more heavily on muscarinic and histaminergic transmission than on serotonin at the NTS level [6,24]. Noradrenergic projections from the locus coeruleus (LC) to the cerebellum and vestibular nuclei; central alpha-2 agonists (clonidine) have shown modest anti-nausea properties in experimental motion

sickness models by attenuating LC-mediated arousal of the NTS [6,23]. The practical relevance is that SSRIs and SNRIs — used in PPPD and VM — partially modulate the serotonergic component of NTS activity, which may contribute to their incidental improvement of MSS in these conditions [26,46].

□ **Clinical Pearl:** Second-generation antihistamines (cetirizine, loratadine, fexofenadine) do not cross the blood-brain barrier sufficiently to block H1 vestibular-NTS transmission and are clinically ineffective for motion sickness. Always prescribe a first-generation agent. [4,32]



*Figure 3. The area postrema (arrows), a paired swelling flanking the floor of the fourth ventricle in the dorsal medulla (primate specimen). As a circumventricular chemoreceptor trigger zone it samples blood-borne emetic stimuli and, with the adjacent nucleus tractus solitarius, forms the final common emetic pathway.*

*Source: Micrograph by Tulemo, Wikimedia Commons. CC BY-SA 4.0.*

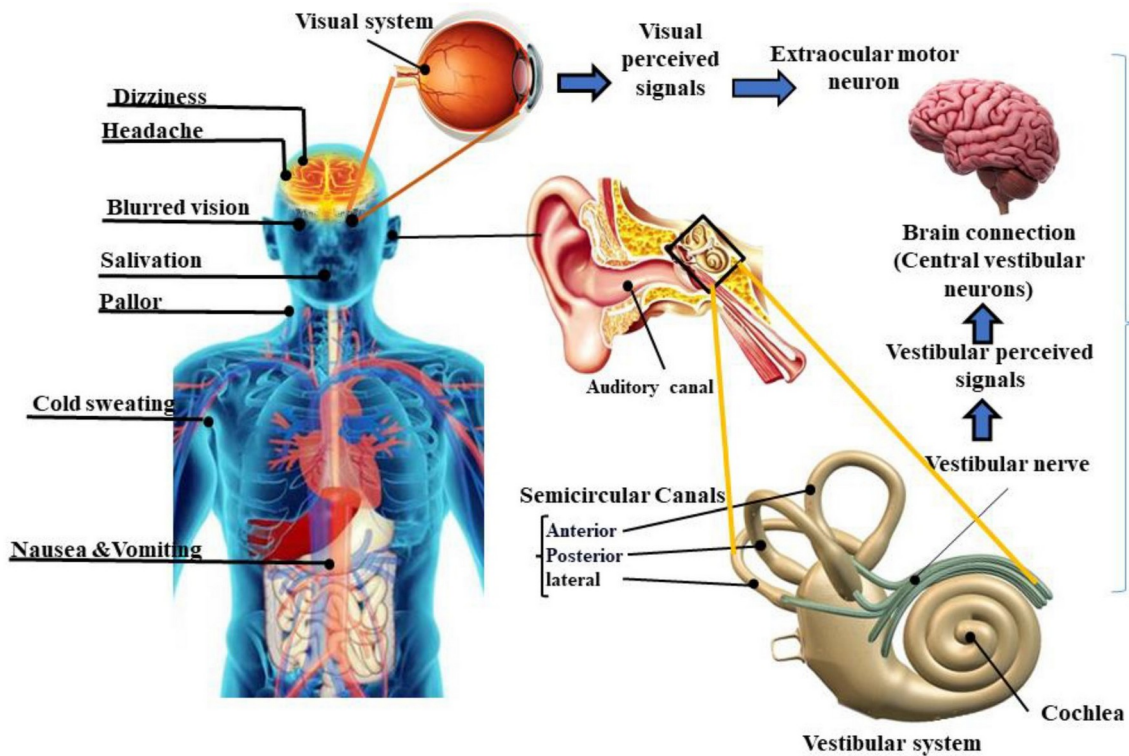


Figure 4. Integrated motion-sickness pathway — visual and vestibular signals converge on central vestibular neurons; unresolved conflict drives the autonomic-emetic cascade of pallor, salivation, cold sweating, nausea and emesis.

Source: Rahimzadeh G, et al. *Nutrients* 2023;15(6):1320. CC BY 4.0, via Wikimedia Commons.

## IV. Genetics, Heritability, and Biological Risk Factors

The twin study by Reavley et al. [3] established the heritability of adult MSS at 57% (95% CI 51–63%) in a registry of 3,652 female pairs from the TwinsUK cohort. Heritability was highest for childhood MSS recall (70%; 95% CI 59–80%), declining through adolescence and adult years — consistent with the clinical observation that paediatric and adult MSS are phenotypically related but partially independent traits [3,7]. Non-shared environmental factors account for the residual variance, implicating exposure history, habituation, and disease-state changes as meaningful modulators of trait expression across the lifespan [3,18].

### Genome-wide association findings

A genome-wide association study (GWAS) by Hromatka et al. [44] analysed data from 80,494 23andMe research participants and identified 35 independent genomic loci reaching genome-wide significance for MSS susceptibility. Implicated genes cluster in four functional categories: inner ear development (PVRL3, TSHZ1), eye movement control (HOXB3, HOXD3), glucose homeostasis (ACCS), and broader neurological signalling. Sex-specific effect sizes for several loci were up to threefold larger in women than in men — providing a genetic basis for the observed sex differences in susceptibility [3,44]. The distribution of risk loci across inner ear, cerebellar, and metabolic pathways confirms that MSS is a polygenic trait rather than a single-system vulnerability [44].

The overlap between MSS risk loci and migraine susceptibility genes is emerging as a productive research frontier. Several loci are shared between MSS and migraine GWAS data, including regions implicated in trigeminal sensitisation and ion channel function [42,44]. This genetic co-aggregation provides the biological basis for the strong clinical association between migraine family history and elevated MSSQ-Short scores [7,12,44].

### Biological risk modulators

Beyond inherited genetics, several biological factors independently and reversibly elevate MSS. Hormonal state is the most clinically accessible: oestrogen sensitises cholinergic projections in the vestibular nuclei and NTS, explaining the cycle-dependent fluctuation in MSS scores in women of

reproductive age [9,13]. Oral contraceptive pills containing synthetic oestrogen produce a sustained elevation of MSS that normalises on cessation [9]. Pregnancy — particularly the first trimester — dramatically amplifies MSS through both hormonal sensitisation and the direct emetic effects of human chorionic gonadotrophin (hCG) on the area postrema [9]. Anxiety and panic disorder independently elevate MSS through locus coeruleus – NTS arousal loops; this is not simply a coincidence of high-neuroticism individuals — pharmacological and CBT-based anxiety reduction produces measurable reductions in MSSQ-Short scores [25,41,43].

□ **Important:** Complete bilateral vestibulopathy confers paradoxical immunity to most forms of physical motion sickness by eliminating the velocity storage signal. However, partial bilateral loss with a dysregulated residual velocity storage mechanism can worsen VIMS — a clinically underappreciated pattern. [14,50]

## V. Clinical Assessment — History, Questionnaires, and Provocation Testing

### History

The clinical history for MSS should systematically characterise: symptom type (nausea-dominant, dizziness-dominant, or mixed autonomic); trigger modalities (vehicular, visual, VR/screen, oscillatory); age of onset and childhood history; family history of migraine or motion sickness; relevant comorbidities (migraine, anxiety, PPPD, MdDS); and medication history including oral contraceptive use and any prior motion sickness treatments [2,4,9]. Two validated structured instruments are available for routine clinic use and should be administered to every new patient where vestibular migraine, PPPD, or visually triggered dizziness is in the differential [2,15].

### The MSSQ-Short (Golding 1998)

The MSSQ-Short [2,47] measures historical susceptibility across nine transport and environmental modes: car, bus, train, aircraft, small boat, large ship, swings and rides, films and videos, and virtual reality. Subscale A captures childhood experience; subscale B captures the past ten years. The total MSSQ score is computed from raw subscale scores normalised to the number of applicable modes, yielding a 0–18 scale. Population normative data (Golding 1998, n = 400+) show median scores of 6.4 in women and 4.8 in men; scores above 9 classify as "high susceptibility" and scores above 12 as "very high" [2,47]. The MSSQ-Short has been validated in otolaryngological populations including patients with tinnitus and vestibular disorders [19]. The subscale A/B ratio has diagnostic value: a high A with low B (childhood-dominant pattern) is characteristic of migraine-associated MSS, where spontaneous improvement through adolescence is common; a high B with low or moderate A (adult-onset pattern) should raise suspicion for a secondary cause — new vestibular pathology or cerebellar lesion [2,11].

### The VIMSSQ-Short (Golding and Keshavarz 2023)

The VIMSSQ-Short [15] was developed specifically to capture susceptibility to visually induced triggers — an important gap not fully addressed by the vehicular-heavy MSSQ-Short. It comprises nine items targeting cinema with wide optic flow, video gaming, VR head-mounted displays, reading in moving vehicles, scrolling smartphone feeds, occupational simulator use, passenger car travel at speed, and two additional visual exposure scenarios. It is normed on 1,123 subjects (median 4.1 women, 2.8 men) [15]. High VIMSSQ-Short scores (>10) are strongly associated with PPPD diagnosis and predict occupational VIMS interference [15,45]. The correlation with the MSSQ-Short is  $r = 0.50$ , and with migraine  $r = 0.44$ , confirming partial but not complete phenotypic overlap [15]. Critically, a 2023 structural equation model [45] demonstrated that PPPD severity, VIMSSQ-Short, MSSQ-Short, and migraine load on a single latent dimension of central sensitivity — suggesting shared neurobiological substrate rather than independent conditions.

### Single-item screen

The single question "Can you read in a moving car without becoming sick?" offers sensitivity of approximately 74% and specificity of 69% for the high MSSQ-Short category in a general vestibular clinic population [9]. Its brevity makes it a practical first-line triage tool for time-limited consultations; positive

respondents should proceed to full MSSQ-Short and VIMSSQ-Short assessment [9]. A single positive response also independently increases the pre-test probability of vestibular migraine by an OR of 2.8–3.4 in clinic-based studies [9,12].

## Clinical Assessment Pathway for MSS

Vestibular physician consultation

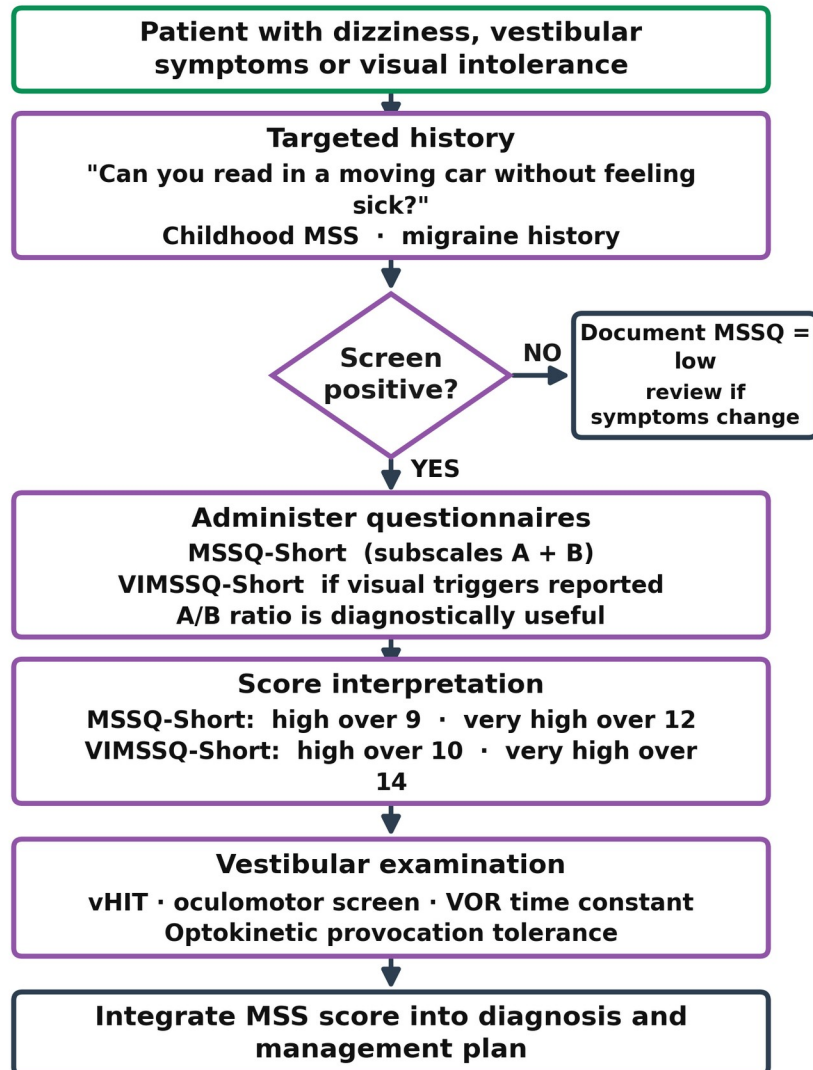


Figure 5. Clinical assessment pathway for motion sickness susceptibility in a vestibular physician consultation — from initial screen to examination and integration into diagnostic management.

Source: Adapted from Golding [2] and Akdal et al. [9].

### Examination findings and provocation

Standard vestibular examination for MSS assessment should specifically include: (i) vHIT — VOR gain and corrective saccade pattern across all six canals; (ii) rotatory chair testing where available — the velocity storage time constant (TV) is directly measurable and is the most objective physiological correlate of MSS [14,20]; (iii) ocular motor screen — smooth pursuit gain, VOR suppression, optokinetic nystagmus; (iv) brief optokinetic provocation using a moving-stripe display — nausea onset latency and symptom severity are semi-quantitative MSS measures [18]; and (v) galvanic vestibular stimulation (GVS) — where available — as elevated perception of head tilt to GVS correlates with high MSSQ-Short scores [20]. The Dix-Hallpike and supine roll tests may precipitate nausea disproportionate to the positional

nystagmus amplitude in high-MSS patients — a useful clinical sign distinguishing central sensitisation from peripheral mechanical BPPV alone [20].

□ **Clinical Insight:** An elevated MSSQ-Short subscale B (recent 10 years) that is substantially higher than subscale A (childhood) warrants investigation for a secondary cause: new vestibular pathology, cerebellar lesion, or medication effect. The typical migraine pattern is high A with partial improvement in B. [2,11]

**Table 2. Validated assessment instruments for motion sickness susceptibility.**

Instrument	Items	Subscales	Score Range	Clinical Cut-off	Key Strength
<b>MSSQ-Short (Golding 1998)</b>	9 items	A (childhood) B (recent 10 yr)	0–18	>9 = high >12 = very high	Transport MSS; subscale A/B ratio diagnostically useful
<b>VIMSSQ-Short (Golding &amp; Keshavarz 2023)</b>	9 items	Single composite	0–18	>10 = high >14 = very high	Visual/screen triggers; PPPD severity predictor
<b>Single-item screen (Akdal et al.)</b>	1 question	N/A	Yes/No	Positive = proceed to full MSSQ	Sensitivity 74%; rapid triage tool
<b>Rotatory chair TV (laboratory)</b>	VOR decay	TV (seconds)	Continuous	TV >15 s = elevated	Physiological correlate; objective measure

## VI. Motion Sickness Susceptibility in Vestibular Disorders

MSS does not present uniformly across vestibular diagnoses. Five distinct phenotypic patterns are clinically recognisable, each with different onset timing relative to disease, different questionnaire profiles, and different management implications [7,8,11,13,28,45]. Understanding these patterns is essential for the vestibular physician — both for diagnostic reasoning and for sequencing the management approach correctly.

### Vestibular migraine

MSS in vestibular migraine (VM) predates disease onset in most patients: trait MSS is a premorbid vulnerability rather than a secondary effect of vestibular dysfunction [7,8,49]. MSSQ-Short scores in VM are 40–60% higher than age- and sex-matched controls [7,8]. Childhood motion sickness is the single strongest historical predictor of eventual VM diagnosis, present in over 70% of VM patients versus 20–30% in controls [12,49]. The Abouzari et al. [8] 2021 cohort confirmed that VM patients showed the highest MSSQ-Short scores of any vestibular diagnostic category in a tertiary dizziness clinic population. The mechanism is shared sensitisation of brainstem and cerebellar circuits — the same velocity storage instability and trigeminal-vestibular sensitisation that underlies migraine also lowers the threshold for sensory conflict nausea [7,42,49]. The practical corollary: in a patient with chronic dizziness and elevated MSSQ-Short, VM must be at the top of the differential. Anti-migraine prophylaxis (topiramate, propranolol, amitriptyline, venlafaxine) improves MSSQ-Short scores alongside headache outcomes [7,49].

### PPPD and visually induced dizziness

PPPD is defined by persistent (>3 months) non-spinning dizziness with marked visual dependence and upright posture exacerbation [26]. The VIMSSQ-Short consistently loads on the same latent dimension as PPPD severity, suggesting that PPPD and VIMS susceptibility share visual-dependence circuitry rather than being coincident independent conditions [45]. A 2024 comparative study [45] confirmed that PPPD patients report more visual vertigo than VM patients, but VM patients carry a higher childhood MSS burden — a clinically useful distinction when differentiating the two. The management implication is direct: PPPD-associated VIMS specifically responds to graded optokinetic desensitisation (see Section VII), whereas VM-associated MSS responds to anti-migraine prophylaxis as the priority intervention [26,45].

## Ménière's disease

MSS is elevated in Ménière's disease but — critically — specifically in established disease rather than in at-risk or pre-clinical individuals, strongly suggesting that the elevated MSS reflects secondary vestibular dysfunction rather than a trait vulnerability [11]. Golding and Patel's prospective 751-patient registry analysis [11] confirmed that MSS onset coincides with disease onset in Ménière's, in clear contrast to VM where it precedes it. This temporal distinction is the key diagnostic differentiator: a patient presenting with both episodic dizziness and current motion sickness, but no childhood MSS history, favours Ménière's over VM in the differential [11].

## Post-concussive vestibular dysfunction

Post-concussive dizziness is associated with elevated MSS scores, particularly on the VIMSSQ-Short, with a proposed mechanism involving diffuse axonal injury to vestibulo-cerebellar pathways and optic flow processing cortex (superior temporal sulcus, MT/V5) [5]. MSS quantification using the VIMSSQ-Short at 4–8 weeks post-concussion is clinically prognostic: patients with high scores are at elevated risk for prolonged symptoms and benefit from early graded habituation and active optokinetic stimulation rather than vestibular rest [5,17]. The traditional advice of prolonged visual rest in post-concussive dizziness conflicts with vestibular rehabilitation principles and should be reframed as "graded, controlled reintroduction" rather than complete avoidance [5,17].

## Mal de débarquement syndrome

Mal de débarquement syndrome (MdDS) shares motion sickness neurobiology at the level of velocity storage persistence, but the pharmacological management profile diverges completely from classical MSS. Anticholinergic and antihistaminergic agents that attenuate motion sickness do not ameliorate MdDS and may worsen the oscillatory perception [28]. The vestibulocerebellar optokinetic re-adaptation approach — using earth-vertical axis rotation paired with matched optic flow — is the specific intervention for MdDS [28]. This is a critical management distinction: prescribing cinnarizine or scopolamine to a patient with MdDS (a common error when the history emphasises boat travel and motion sickness history) is not only ineffective but potentially counterproductive [28].

**Table 3. MSS phenotypic profiles across vestibular diagnoses.**

Diagnosis	MSS Onset	MSSQ-Short	VIMSSQ-Short	Key Mechanism	Management Priority
<b>Vestibular Migraine</b>	PREMORBID (trait)	HIGH (40–60% > ctrl)	Moderate–High	Brainstem sensitisation + shared genetic loci	Anti-migraine prophylaxis first [7,42,49]
<b>PPPD / VIMS</b>	Variable (often premorbid)	Moderate	VERY HIGH (primary tool)	Visual dependence + optic flow gain	Graded OKN desensitisation; SSRIs/SNRIs [26,45]
<b>Ménière's Disease</b>	SECONDARY (after onset)	Moderate	Moderate	Vestibular hypofunction + overshoot	Vestibular rehab; treat hydrops [11]
<b>Post-Concussive</b>	Post-injury secondary	HIGH	HIGH (dominant)	VC pathway disruption; VS dysregulation	Early graded habituation; avoid rest [5,17]

<b>MdDS</b>	Exposure-triggered	Moderate	Moderate	Velocity storage persistence	Re-adaptation ONLY; NOT antihistamines [28]
-------------	--------------------	----------	----------	------------------------------	---

□ **Clinical Pearl:** The temporal relationship between MSS onset and vestibular disease onset is the key differential marker: premonitory MSS strongly favours vestibular migraine; onset coinciding with disease onset favours Ménière's disease. Ask specifically about childhood motion sickness in every patient with episodic vestibular symptoms. [7,11]

## VII. Non-Pharmacological Management

### Behavioural and environmental first-line measures

Initial management is behavioural and environmental, and should be counselled at first consultation regardless of whether pharmacotherapy is also planned [4,30,35]. Situating the patient in the lowest-motion zone of the vehicle (front seat of a car, midship on a vessel, over the wing of an aircraft) reduces vestibular stimulus amplitude. Visual anchor fixation on a stable horizon or dashboard suppresses the vestibular-visual conflict by providing a stable reference frame [4]. Reclining the head reduces gravitational otolith loading and can attenuate nausea in susceptible individuals, particularly on sea voyages [4]. Controlled timing of disclosure — informing the patient in advance of the vehicle's intended motion (speed, duration, route) — reduces the element of unexpected conflict and reduces symptom severity through anticipatory central processing [30].

### Diaphragmatic breathing

Controlled diaphragmatic breathing at 6 breaths per minute has demonstrated efficacy in two randomised crossover studies of subjects exposed to circularvection (a visual motion paradigm producing reliable nausea) [31]. The mechanism operates through two parallel pathways: restoration of autonomic balance (reducing sympathetic arousal that amplifies NTS nausea perception) and through attentional redirection from the interoceptive nausea signal [30,31]. The technique must be taught in clinic and practised in a non-provoking environment before deployment during motion exposure. A practical target is 5–10 minutes of diaphragmatic breathing practice daily in the week preceding anticipated travel [31].

### Graded habituation

The neural mismatch model predicts that repeated sub-threshold exposure to the conflict stimulus progressively updates the internal model, reducing mismatch amplitude with successive exposures [1,17,18]. This is the mechanistic basis for habituation training — the standard vestibular rehabilitation approach to MSS. Graduated exposure protocols begin with brief, low-intensity stimuli (optokinetic stripe viewing at slow speeds, short car rides of 5 minutes) and systematically advance stimulus intensity and duration over 4–8 weeks [17,18]. Golding et al. [18] demonstrated that sensitisation from prior provocative physical motion (i.e., the worsening of VIMS after physical car travel) is a real phenomenon that must be accounted for in protocol design — habituation sessions should not be immediately preceded by uncontrolled provocative exposures in early training. Rine et al. [17] reported improvements in MSS scores and VOR suppression after an 8-week visual-vestibular habituation programme. Migraine comorbidity does not contraindicate habituation but may require slower progression and scheduling sessions outside the migrainous peri-ictal window [7,49].

### Vestibular rehabilitation for VIMS

Specific VRT protocols for VIMS (the PPPD/cybersickness phenotype) incorporate: horizontal and vertical gaze stabilisation exercises; optokinetic stimulation at incrementally graded speeds; vestibular adaptation exercises; and sensory reweighting tasks on compliant surfaces [17,26]. The PPPD-specific vestibular rehabilitation programme (Staab et al. framework) [26] includes both vestibular and visual-exposure components and demonstrates reduction in VIMSSQ-Short scores alongside PPPD functional outcome measures. The target response criterion for rehabilitation is a minimum 3-point reduction in MSSQ-Short and tolerance of a 10–20 minute optokinetic challenge without symptom onset beyond mild transient nausea [17].

## Cognitive-behavioural adjuncts

Biofeedback targeting heart rate variability and cognitive-behavioural approaches addressing catastrophic appraisal of dizziness are evidence-based adjuncts in patients with high anxiety burden [25,30,43]. The neurobiological rationale is direct: locus coeruleus hyper-arousal amplifies NTS activity and lowers the nausea threshold; any intervention that reduces LC arousal (CBT, biofeedback, SSRI/SNRI) will partially attenuate MSS [25,41,43]. This should be framed for patients as treating a brain-level sensitivity, not as a psychological "mind over matter" approach, which can inadvertently invalidate the somatic reality of their symptoms.

### Non-Pharmacological Management

Stepwise algorithm — behavioural and rehabilitative

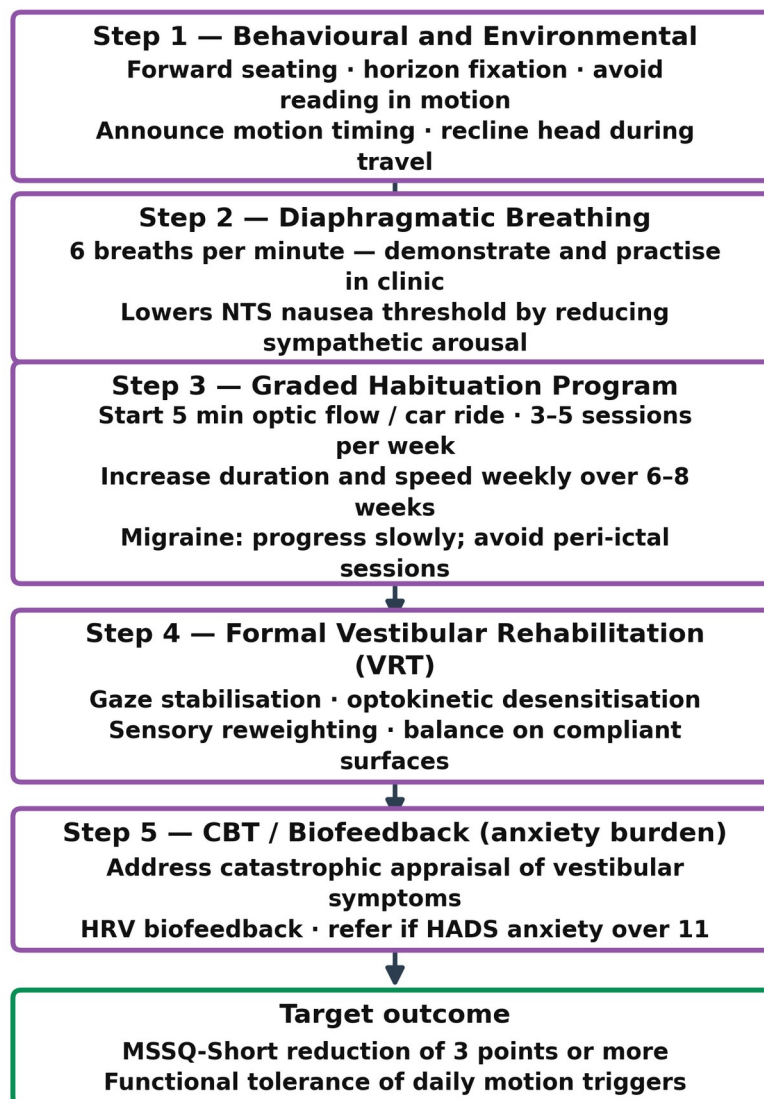


Figure 6. Non-pharmacological management stepwise algorithm — behavioural measures and graded habituation precede pharmacotherapy in all patient categories. VRT = vestibular rehabilitation therapy; OKN = optokinetic nystagmus; HRV = heart rate variability.

Source: Adapted from Rine et al. [17], Golding et al. [18], and Stromberg et al. [31].

## VIII. Pharmacological Management

Pharmacological management of MSS is evidence-based, with the highest-quality data from two Cochrane systematic reviews [16,32] and multiple randomised controlled trials. Drug selection should be guided by: expected exposure duration; migraine comorbidity; patient age; operator duties (driving, flying, surgery); and pregnancy status [4,16,32,35].

## Scopolamine (hyoscine)

Transdermal scopolamine 1.5 mg (Scopoderm® patch) is the most comprehensively supported pharmacological agent for motion sickness prophylaxis [16,29]. The 2004 Cochrane review (Spinks et al.) and subsequent meta-analyses confirm superiority to placebo in controlled trials of sea, air, and experimental rotation sickness [16]. Onset of protection requires approximately 4 hours after patch application; duration of action is 72 hours [16]. The requirement for pre-exposure application is the most consistently misunderstood aspect of scopolamine prescribing — patches applied after symptom onset do not provide meaningful relief and may contribute to anticholinergic side effects without therapeutic benefit [16,29]. Key caveats: anticholinergic side effects (dry mouth, blurred vision, urinary retention, constipation) limit use in elderly patients and those on anti-dementia medications; central anticholinergic syndrome (confusion, agitation, tachycardia) is a rare but serious complication in frail elderly patients [29,35].

## First-generation H1 antihistamines

The 2022 Cochrane review (Karrim et al.) [32] examined six first-generation agents: cinnarizine, dimenhydrinate, meclizine, promethazine, cyclizine, and hydroxyzine. Under naturalistically induced motion sickness, antihistamines as a class produce a probably significant reduction in symptoms compared to placebo (moderate certainty evidence); compared to scopolamine, no agent demonstrated superiority — scopolamine prevented symptoms in 81% versus 71% for antihistamines in the comparative trial subset [32]. Cinnarizine (25 mg TDS) and dimenhydrinate (50 mg every 4–6 hours) are the most widely used in clinical practice and have the most favourable tolerability profiles [4,32,34]. Promethazine offers the highest anti-emetic efficacy of the class but produces the most sedation — restricting its use to situations where sedation is acceptable (overnight cruise, air travel) and is absolutely contraindicated for patients with operator duties [4].

## Dopaminergic adjuncts and combination therapy

In vestibular migraine patients with nausea-dominant MSS incompletely controlled by H1 antihistamines, adding a D2 antagonist (prochlorperazine 5–10 mg PO or domperidone 10 mg PO) targets the parallel area postrema emetic pathway and can provide superior combined efficacy [24,42]. Metoclopramide is an alternative but carries a higher risk of extrapyramidal side effects with repeated dosing. In clinical practice, the H1 + D2 combination should be reserved for VM patients with refractory nausea after establishing that anti-migraine prophylaxis has been optimised, as prophylaxis — not as-needed antiemetics — is the preferred long-term strategy [49].

## Special prescribing considerations

Ginger extract (*Zingiber officinale*, 1–2 g orally before exposure) has modest evidence in pregnancy-associated nausea and at least one small crossover RCT in rotational motion sickness [35]. Its safety profile makes it an appropriate recommendation for pregnant patients who cannot take antihistamines in the first trimester. The Beers Criteria flag scopolamine and diphenhydramine as potentially inappropriate medications in older adults due to anticholinergic cognitive burden; cinnarizine and cyclizine are preferable in this population [35]. No established pharmacological treatment exists for CGRP-pathway targeting of MSS, though anecdotal VM series suggest anti-CGRP monoclonals may improve inter-ictal MSS scores as part of their broader anti-migraine effect [49].

**Table 4. Pharmacological agents for motion sickness — mechanism, evidence level, dose, and caveats.**

Agent	Mechanism	Dose (Adult)	Evidence Level	Key Caveats
<b>Scopolamine (transdermal)</b>	M1/M3 anticholinergic (central)	1.5 mg patch 4 h before exposure	Cochrane: high [16]	Apply early; anticholinergic SE; avoid in elderly/frail
<b>Cinnarizine</b>	H1 antagonist + calcium channel	25 mg TDS	Cochrane: moderate [32]	Sedation; avoid with alcohol;

				weight gain long-term
<b>Dimenhydrinate</b>	H1 antagonist (central)	50 mg Q4-6 h	Cochrane: moderate [32]	Sedation; impairs psychomotor performance
<b>Promethazine</b>	H1 + D2 + M antagonist	25 mg PO or IM	High efficacy; highest sedation	Contraindicated if driving; paediatric warnings
<b>Prochlorperazine</b>	D2 antagonist	5-10 mg PO	Adjunct evidence [24]	EPS risk; migraine comorbidity use; not in pregnancy
<b>Ginger extract</b>	Uncertain (5-HT3 weak antagonism?)	1-2 g PO before exp.	Modest RCT [35]	Safe in pregnancy; modest effect
<b>Ondansetron</b>	5-HT3 antagonist	4-8 mg PO	Weak for MSS [6]	Effective for postop/chemo; NOT first-line for MSS

### Pharmacotherapy Decision Tree

Motion sickness prophylaxis and acute management

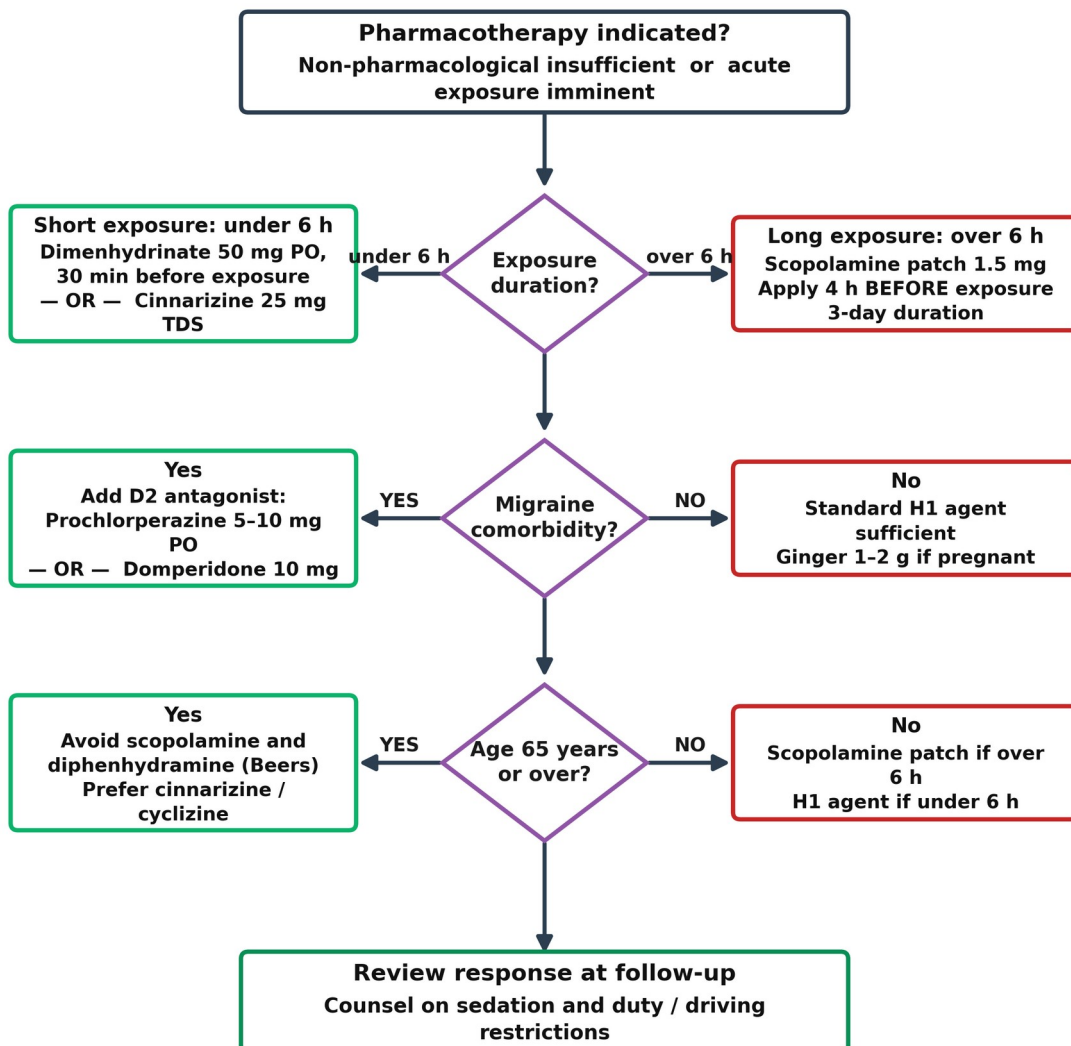


Figure 7. Pharmacotherapy decision tree for motion sickness prophylaxis and treatment — drug selection driven by exposure duration, comorbidity, age, and patient context.

Source: Adapted from Spinks et al. [16] and Karrim et al. [32].

□ **Important:** Scopolamine must be applied 4 hours BEFORE exposure — not after symptom onset. Patches applied reactively are ineffective and carry the full anticholinergic side-effect burden without therapeutic benefit. This is the single most common scopolamine prescribing error in clinical practice. [16,29]

## IX. Special Populations, Occupational Contexts, and Cybersickness

### Cybersickness and visually induced motion sickness (VIMS)

VIMS — symptoms triggered entirely by visual motion without physical movement — has emerged as the fastest-growing MSS presentation in vestibular clinics over the past five years [15,27]. Triggers include: scrolling social media feeds on smartphones; wide-field cinema (IMAX, Dolby); VR gaming headsets; flight simulation; radiological review stations; and occupational screen-heavy environments [15,27]. The underlying mechanism is classical sensory conflict in the reverse direction: optic flow signals movement that the stationary labyrinth does not confirm [12,15,48]. Up to 80% of VR headset users experience some degree of cybersickness on first exposure; 50% abandon the VR session within 20 minutes due to symptoms [27]. VIMSSQ-Short scores above 12 identify patients at high risk for occupational VIMS interference and warrant specific VRT [15,27].

### Cybersickness in VR rehabilitation

A clinically relevant paradox exists: VR platforms are increasingly deployed for vestibular rehabilitation, but high-MSS patients — the very patients most needing rehabilitation — are most susceptible to cybersickness from VR, potentially limiting participation and creating a barrier to graded exposure [27]. Progressive desensitisation protocols that begin with 2D screen-based optic flow (standard monitor, slow scroll, horizontal movement) before advancing to immersive VR head-mounted displays mitigate this effectively in practice [27]. The 2024 study by Ngan et al. [27] on within-day interval optimisation confirmed that multiple short VR exposure sessions within a single day (with 2-hour gaps) produce faster adaptation than single longer sessions, providing a protocol basis for structured clinic VR habituation in VIMS patients.

### Pregnancy and peripartum

First-trimester pregnancy dramatically amplifies MSS, with partial overlap between pregnancy-induced nausea/vomiting (and hyperemesis gravidarum) and motion sickness mediated by oestrogen-sensitised cholinergic pathways and hCG stimulation of the area postrema [9,13]. Safe pharmacological options include: dimenhydrinate (considered compatible with pregnancy, widely used); cyclizine (limited human data, generally used in practice); and ginger extract (1–2 g, well-tolerated) [9,35]. Scopolamine and promethazine are classified as category C in pregnancy and are not recommended in the first trimester [35]. For Ménière's disease patients requiring motion sickness management during pregnancy, cinnarizine carries limited pregnancy data and referral to maternal-foetal medicine for individualised risk assessment is appropriate [9].

### Occupational and military contexts

Seafarers, naval personnel, military vehicle crews, and fighter pilots experience high baseline MSS attrition rates in initial training, with rates of 28–60% in naval aviation candidates before habituation programmes [33,36]. Graded habituation exposure protocols — standard in naval aviation training since the 1960s — reliably reduce MSS scores across training periods and reduce attrition [33,36]. For commercial aviation, scopolamine and antihistamine use by flight crew is generally prohibited by civil aviation authority regulations due to sedation and psychomotor effects [35]. In military special operations contexts, scopolamine-dextroamphetamine combinations have been used under specific operational protocols, but are outside civilian vestibular practice [33].

### Elderly patients

MSS scores decline with advancing age — the elderly are paradoxically less susceptible to motion sickness, likely due to physiological shortening of velocity storage time constants and progressive reduction in labyrinthine sensitivity [14,38]. However, when motion sickness does occur in elderly patients, the pharmacological risk profile is substantially different. The Beers Criteria list scopolamine and diphenhydramine as potentially inappropriate medications in older adults due to cognitive, urinary, and cardiovascular anticholinergic burden [35]. Cinnarizine or cyclizine, with lower anticholinergic profiles, are preferred first-line agents in this cohort. New or worsening MSS in a patient over 65 years should prompt a systematic review of all new medications for anticholinergic or dopaminergic contributions, as drug-induced vestibular and motion sickness sensitivity shifts are well-documented [5,35].

**Table 5. Special population management considerations for motion sickness susceptibility.**

Population	Key Issue	Preferred Agents	Agents to Avoid
<b>Pregnancy (1st trimester)</b>	hCG + oestrogen sensitisation of area postrema	Dimenhydrinate · Cyclizine Ginger 1–2 g	Scopolamine · Promethazine (Category C)
<b>Age ≥ 65 years</b>	Beers Criteria anticholinergic cognitive burden	Cinnarizine 25 mg Cyclizine 50 mg	Scopolamine · Diphenhydramine (cognitive SE)
<b>Commercial aviation crew</b>	Regulatory prohibition (sedation / psychomotor)	Non-pharmacological only; graded habituation	All sedating agents during duty (civil aviation regulations)
<b>Post-concussion</b>	VIMSSQ-Short elevated; central sensitisation	Graded optokinetic desensitisation; avoid rest	Prolonged visual rest (delays adaptation)
<b>VR rehabilitation patients</b>	Cybersickness paradox (rehab triggers MSS)	Progressive 2D → VR protocol; short sessions	Immediate full-immersion VR (causes session failure)



*Figure 8. Immersive head-mounted virtual reality delivers compelling visual motion (strong optic flow) without corresponding vestibular input — a potent provocateur of visually induced motion sickness, or cybersickness.*  
Source: Photograph by InclusiveGameLab, Wikimedia Commons. CC BY-SA 4.0.

## VIMS / Cybersickness — Vestibular Physician Pathway

Visually induced motion sickness: assessment to management

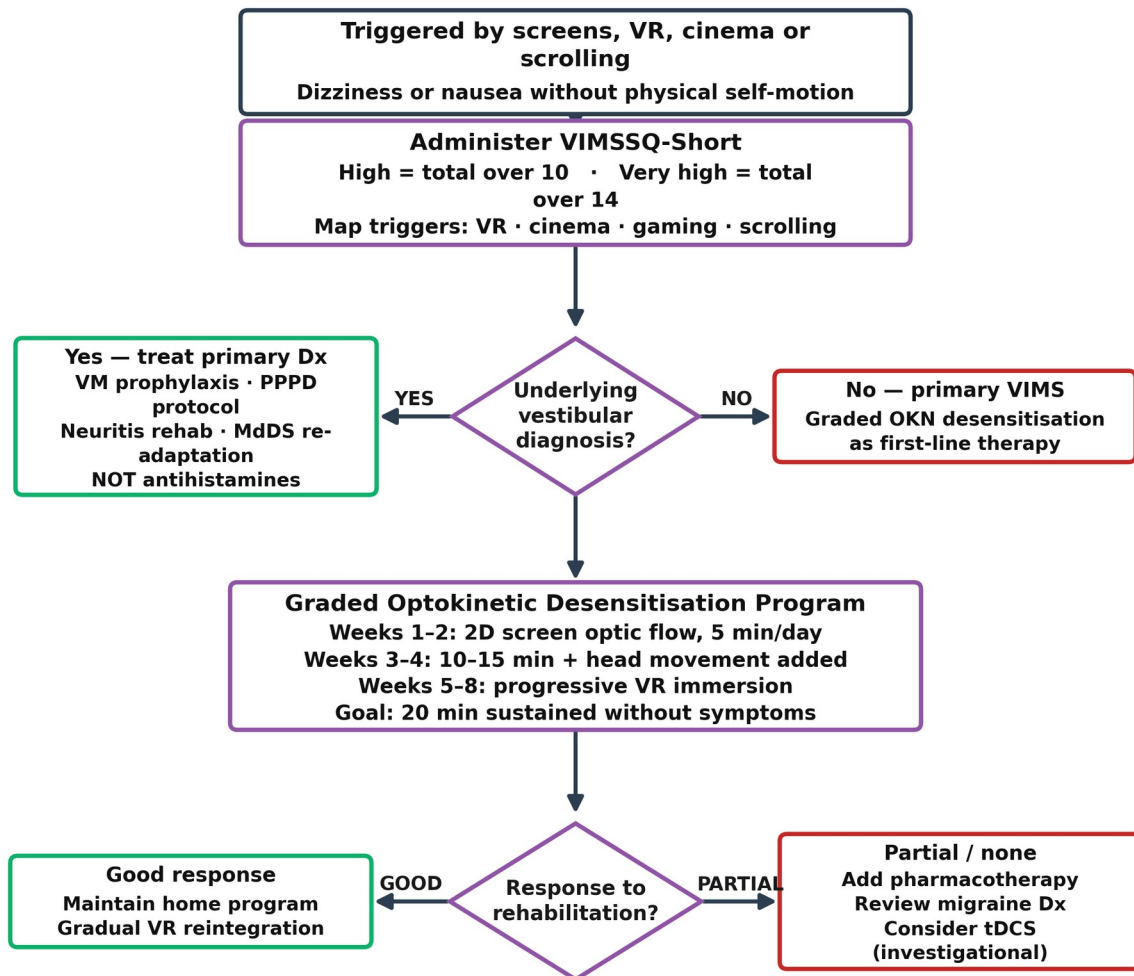


Figure 9. Visually induced motion sickness (VIMS) and cybersickness vestibular physician pathway — structured assessment, identification of underlying vestibular diagnosis, and graduated desensitisation protocol.  
Source: Adapted from Golding and Keshavarz [15] and Ngan et al. [27].

## X. Controversies, Future Directions, and Research Frontiers

### The 'toxin detector' theory and evolutionary frameworks

Two competing evolutionary frameworks explain the vomiting reflex being triggered by motion. Treisman's 1977 poisoning-mimicry theory [37] proposes that emesis represents a fortuitous cross-activation of the toxin-detection system — the abnormal movement signal mimics the postural ataxia of neurotoxin ingestion, triggering protective emesis. The neurotoxin hypothesis (Bles et al.) more specifically proposes that nausea and vomiting evolved to purge ingested substances before they could produce movement-associated ataxia [6,37]. Neither framework fully accounts for the dose-response characteristics of habituation (the toxin model predicts that habituation to "poisoning" would be maladaptive), nor for the fact that antihistamines — which have no direct relationship to the emetic reflex or toxin signalling — are consistently efficacious [6]. The evolutionary debate has practical relevance: it informs why non-pharmacological management is mechanistically preferable in the long term — habituation aligns with the internal model update that evolution "designed" the mismatch detector to achieve [1,37].

### Motion sickness immunity with labyrinthine loss

Complete bilateral vestibulopathy produces paradoxical immunity to classical physical motion sickness by eliminating the velocity storage signal that is the substrate for prolonged NTS activation [14,50]. The seminal observation by Kellogg et al. [50] in labyrinthine-defective individuals during zero-gravity manoeuvres confirmed this immunity. However, patients with partial bilateral loss may retain a dysregulated velocity storage signal and experience worsened VIMS in certain environmental contexts — a distinction that is currently clinically underappreciated [14]. The clinical implication is that vHIT showing complete bilateral absence of canal function should accompany counselling that classical MSS is effectively resolved; VIMS from remaining cortical visual processing may persist as a separate phenomenon [14,40].

### Non-invasive neuromodulation of velocity storage

Transcranial direct current stimulation (tDCS) targeting cerebellar pathways has been explored as a method to modulate velocity storage time constants and reduce cybersickness severity [27]. A 2025 cathodal cerebellar tDCS study demonstrated reduced cybersickness symptom scores and altered functional near-infrared spectroscopy (fNIRS) cortical patterns during VR exposure, establishing cerebellar tDCS as a credible investigational treatment candidate for refractory VIMS [27]. No clinical trials have advanced to Phase II for vestibular clinic populations, but the rationale is mechanistically sound given the nodular inhibitory control of velocity storage [14,22].

### CGRP pathway and pharmacotherapy frontiers

The CGRP–vestibular–migraine interface is an active research frontier with direct implications for MSS pharmacotherapy. Whether CGRP-targeted therapies — gepants (rimegepant, ubrogepant) and anti-CGRP monoclonal antibodies (erenumab, fremanezumab, galcanezumab) — reduce MSS trait scores in VM patients is under active investigation [49]. Early case series in VM suggest that patients achieving anti-CGRP monoclonal responder status report improved inter-ictal tolerance of motion triggers alongside headache reduction, but prospective MSS-specific outcome data are lacking [42,49]. A well-designed prospective cohort study measuring MSSQ-Short before and at 6 and 12 months of anti-CGRP monoclonal therapy in VM patients would substantially advance this field.

### Wearable technology and MSS quantification

Head-worn wearables capable of capturing head kinematics, heart rate variability, electrodermal activity, and eye movement metrics may enable continuous, passive, ecological MSS quantification outside the clinic — a significant advance over retrospective questionnaire tools [38]. These devices could provide real-time feedback for habituation protocol adherence, monitor rehabilitation progress, and generate objective symptom burden data for clinical trials [38]. The convergence of wearable vestibular sensing with AI-based pattern recognition is anticipated to substantially change MSS assessment within the next decade, with implications for both clinical practice and vestibular research methodology [38].

□ **Clinical Pearl:** Patients with complete bilateral vestibulopathy are effectively immune to classical physical motion sickness but may still experience VIMS from cortical optic flow processing. Counsel accordingly and assess both MSSQ-Short and VIMSSQ-Short independently in this group. [14,40,50]

## References

- [1] Reason JT, Brand JJ. Motion Sickness. London: Academic Press; 1975.
- [2] Golding JF. Motion sickness susceptibility questionnaire revised and its relationship to other forms of sickness. *Brain Res Bull.* 1998;47(5):507–16. doi:10.1016/S0361-9230(98)00091-4.
- [3] Reavley CM, Golding JF, Cherkas LF, Spector TD, MacGregor AJ. Genetic influences on motion sickness susceptibility in adult women: a classical twin study. *Curr Biol.* 2006;16(24):2442–4. doi:10.1016/j.cub.2006.10.058.
- [4] Leung AK, Hon KL. Motion sickness: an overview. *Drugs Context.* 2019;8:2019-9-4. doi:10.7573/dic.2019-9-4.
- [5] Koch A, Cascorbi I, Westhofen M, Dafotakis M, Klapa S, Kuhtz-Buschbeck JP. The neurophysiology and treatment of motion sickness. *Dtsch Arztebl Int.* 2018;115(41):687–96. doi:10.3238/arztebl.2018.0687.
- [6] Schmäl F. Neuronal mechanisms and the treatment of motion sickness. *Pharmacology.* 2013;91(3–4):229–41. doi:10.1159/000350454.
- [7] Marcus DA, Furman JM, Balaban CD. Motion sickness in migraine sufferers. *Expert Opin Pharmacother.* 2005;6(15):2691–7. doi:10.1517/14656566.6.15.2691.
- [8] Abouzari M, Cheung D, Pham T, Goshtasbi K, Sarna B, Tajran S, et al. The relationship between vestibular migraine and motion sickness in a multidisciplinary tertiary dizziness clinic population. *Otol Neurotol.* 2021;42(4):e492–6. doi:10.1097/MAO.0000000000003027.
- [9] Akdal G, Özçelik P, Balçi B, Halmágyi GM, Ünal B. One simple question detects motion sickness susceptibility in migraine patients. *J Clin Neurosci.* 2023;111:41–5. doi:10.1016/j.jocn.2023.03.003.
- [10] Gedik-Soyuyuce O, Yalinay-Dikmen P, Korkut N. The effect of migraine and motion sickness on symptoms evoked by the caloric and rotatory chair test. *J Vestib Res.* 2022;32(3):233–9. doi:10.3233/VES-210145.
- [11] Golding JF, Patel M. Meniere's, migraine, and motion sickness. *Acta Otolaryngol.* 2017;137(5):495–502. doi:10.1080/00016489.2016.1248793.
- [12] Drummond PD. Triggers of motion sickness in migraine sufferers. *Headache.* 2005;45(6):653–6. doi:10.1111/j.1526-4610.2005.05133.x.
- [13] Saman Y, Sharif M, Lee A, Ahmed S, Pagán A, McGuirk M, et al. Sex-disease dimorphism underpins enhanced motion sickness susceptibility and protracted vestibular dysfunction. *Front Neurol.* 2022;13:853994. doi:10.3389/fneur.2022.853994.
- [14] Maruta J. On labyrinthine function loss, motion sickness immunity, and velocity storage. *Front Neurol.* 2024;15:1426213. doi:10.3389/fneur.2024.1426213.
- [15] Golding JF, Keshavarz B. Norms and correlations of the Visually Induced Motion Sickness Susceptibility Questionnaire Short Form (VIMSSQ-Short). *Exp Brain Res.* 2023;241(5):1325–37. doi:10.1007/s00221-023-06570-0.
- [16] Spinks AB, Wasiak J, Villanueva EV, Bernath V. Scopolamine (hyoscine) for preventing and treating motion sickness. *Cochrane Database Syst Rev.* 2004;(3):CD002851. doi:10.1002/14651858.CD002851.pub3.
- [17] Rine RM, Schubert MC, Balkany TJ. Visual-vestibular habituation and balance training for motion sickness. *Phys Ther.* 1999;79(10):949–57.
- [18] Golding JF, Alund D, Gresty MA, Flynn MB. Sensitization of visually induced motion sickness by prior provocative physical motion. *J Vestib Res.* 2009;19(3–4):125–34. doi:10.3233/VES-2009-0334.
- [19] Ugur E, Konukseven BO, Topdag M, Cakmakci ME, Topdag DO. Expansion to the Motion Sickness Susceptibility Questionnaire applied to tinnitus patients. *J Int Adv Otol.* 2022;18(1):8–13. doi:10.5152/iao.2022.20290.
- [20] Patel M. Increased perception of head tilt to galvanic vestibular stimulation correlates to motion sickness susceptibility. *Exp Brain Res.* 2019;237(3):751–61. doi:10.1007/s00221-019-05484-y.
- [21] Yates BJ, Miller AD, Lucot JB. Physiological basis and pharmacology of motion sickness: an update. *Brain Res Bull.* 1998;47(5):395–406. doi:10.1016/S0361-9230(98)00092-6.
- [22] Hain TC, Helminski JO. Anatomy and physiology of the normal vestibular system. In: Herdman SJ, Clendaniel RA, editors. *Vestibular Rehabilitation.* 4th ed. Philadelphia: FA Davis; 2014. p. 2–18.
- [23] Takeda N, Morita M, Kubo T, Yamatodani A, Tohyama M, Watanabe T. Neuropharmacological mechanisms of motion sickness. *Am J Otolaryngol.* 1989;10(5):351–9. doi:10.1016/0196-0709(89)90117-3.
- [24] Soto E, Vega R, Seseña E. Neuropharmacological basis of vestibular system disorder treatment. *J Vestib Res.* 2013;23(3):119–37. doi:10.3233/VES-130495.
- [25] Balaban CD, Jacob RG. Background and history of the interface between anxiety and vertigo. *J Anxiety Disord.* 2001;15(1–2):27–51. doi:10.1016/S0887-6185(00)00043-0.

- [26] Staab JP, Eckhardt-Henn A, Horii A, Jacob R, Strupp M, Brandt T, et al. Diagnostic criteria for persistent postural-perceptual dizziness (PPPD). *J Vestib Res.* 2017;27(4):191–208. doi:10.3233/VES-170622.
- [27] Ngan L, Keshavarz B, So RHY. Effects of within-day intervals on adaptation to visually induced motion sickness in a virtual-reality motorcycling simulator. *Sci Rep.* 2024;14(1):20441. doi:10.1038/s41598-024-71526-9.
- [28] Cha YH, Brodsky J, Ishiyama G, Sabatti C, Baloh RW. Clinical features and natural history of mal de débarquement syndrome. *J Neurol.* 2008;255(7):1053–9. doi:10.1007/s00415-008-0830-0.
- [29] Golding JF, Stott JR. Comparison of the effects of a selective muscarinic receptor antagonist and hyoscine (scopolamine) on motion sickness. *Br J Clin Pharmacol.* 1997;43(4):411–6. doi:10.1046/j.1365-2125.1997.00576.x.
- [30] Dobie TG, May JG. Cognitive-behavioral management of motion sickness. *Aviat Space Environ Med.* 1994;65(10 Suppl):C1–20.
- [31] Stromberg SE, Russell ME, Carlson CR. Diaphragmatic breathing and its effectiveness for the management of motion sickness. *Aerosp Med Hum Perform.* 2015;86(5):452–7. doi:10.3357/AMHP.4152.2015.
- [32] Karrim N, Byrne M, Magula N, Saman Y, Swain S. Antihistamines for motion sickness. *Cochrane Database Syst Rev.* 2022;12(12):CD012715. doi:10.1002/14651858.CD012715.pub2.
- [33] Lackner JR, DiZio P. Space motion sickness. *Exp Brain Res.* 2006;175(3):377–99. doi:10.1007/s00221-006-0697-y.
- [34] Melamed Y, Paz A, Bitterman N. Comparison of cinnarizine and scopolamine for prevention of seasickness. *Aviat Space Environ Med.* 1986;57(7):655–9.
- [35] Brainard A, Gresham C. Prevention and treatment of motion sickness. *Am Fam Physician.* 2014;90(1):41–6.
- [36] Huppert D, Benson J, Brandt T. A historical view of motion sickness — a plague at sea and on land, also with military impact. *Front Neurol.* 2017;8:114. doi:10.3389/fneur.2017.00114.
- [37] Treisman M. Motion sickness: an evolutionary hypothesis. *Science.* 1977;197(4302):493–5. doi:10.1126/science.301659.
- [38] Golding JF. Motion sickness. *Handb Clin Neurol.* 2016;137:371–90. doi:10.1016/B978-0-444-63437-5.00027-3.
- [39] Bronstein AM. Vision and vertigo: some visual aspects of vestibular disorders. *J Neurol.* 2004;251(4):381–7. doi:10.1007/s00415-004-0410-7.
- [40] Dilda V, MacDougall HG, Curthoys IS, Moore ST. Effects of Gz acceleration on otolith-ocular and perceptual responses in humans. *J Vestib Res.* 2011;21(3):129–43. doi:10.3233/VES-2011-0406.
- [41] Balaban CD. Neural substrates linking balance control and anxiety. *Physiol Behav.* 2002;77(4–5):469–75. doi:10.1016/S0031-9384(02)00935-6.
- [42] Furman JM, Marcus DA, Balaban CD. Vestibular migraine: clinical aspects and pathophysiology. *Lancet Neurol.* 2013;12(7):706–15. doi:10.1016/S1474-4422(13)70107-8.
- [43] Saman Y, Bamiou DE, Gleeson M, Dutia MB. Interactions between stress and vestibular compensation: a review. *Front Neurol.* 2012;3:116. doi:10.3389/fneur.2012.00116.
- [44] Hromatka BS, Tung JY, Kiefer AK, Do CB, Hinds DA, Eriksson N. Genetic variants associated with motion sickness point to roles for inner ear development, neurological processes and glucose homeostasis. *Hum Mol Genet.* 2015;24(9):2700–8. doi:10.1093/hmg/ddv028.
- [45] Keshavarz B, Golding JF. Measuring the susceptibility to visually induced motion sickness and its relationship with vertigo, dizziness, migraine, syncope and personality traits. *Exp Brain Res.* 2023;241(4):1143–56. doi:10.1007/s00221-023-06552-2.
- [46] Staab JP. Chronic subjective dizziness. *Continuum (Minneap Minn).* 2012;18(5 Neuro-otology):1118–41.
- [47] Lackner JR. Motion sickness: more than nausea and vomiting. *Exp Brain Res.* 2014;232(8):2493–510. doi:10.1007/s00221-014-3968-6.
- [48] Bos JE, Bles W, Groen EL. A theory on visually induced motion sickness. *Displays.* 2008;29(2):47–57. doi:10.1016/j.displa.2007.09.002.
- [49] Furman JM, Marcus DA, Balaban CD. Migrainous vertigo: development of a pathogenetic model and structured diagnostic interview. *Curr Opin Neurol.* 2003;16(1):5–13. doi:10.1097/00019052-200302000-00003.
- [50] Kellogg RS, Kennedy RS, Graybiel A. Motion sickness symptomatology of labyrinthine defective and normal subjects during zero gravity manoeuvres. *Aerosp Med.* 1965;36:315–8.

## Disclaimer and Copyright

### © Copyright Notice

Copyright © 2026 Australian Dizziness Clinics. All rights reserved. This document and its contents are the intellectual property of Australian Dizziness Clinics. No part of this publication may be reproduced, distributed, transmitted, or stored in any retrieval system in any form or by any means without the prior written permission of Australian Dizziness Clinics.

### Educational Use Only

This review is produced solely for the continuing professional development of healthcare clinicians. It is not intended for lay distribution and does not constitute individualised medical advice. Clinical decisions must always be made in the context of each treating clinician's professional judgement and the specific circumstances of each patient.

### Accuracy and Currency

Whilst every effort has been made to ensure accuracy at the time of publication, vestibular medicine is a rapidly evolving field. Australian Dizziness Clinics makes no warranties, express or implied, regarding the accuracy, completeness, or fitness for purpose of the content.

**Australian Dizziness Clinics**  
[www.AustralianDizzinessClinics.com](http://www.AustralianDizzinessClinics.com)