Anxiety-Related Dizziness:

A Vestibular Physician's Deep Review of Panic Disorder, Generalised Anxiety, and Hyperventilation Syndrome

Vestibular Medicine for Vestibular Physicians

Functional and Psychiatric Vestibular Disorders — Module 5.3

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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 psychiatric 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

Anxiety-related dizziness encompasses a spectrum of presentations in which vestibular symptoms — dizziness, unsteadiness, vertigo, or spatial disorientation — are driven or significantly amplified by anxiety disorders, panic attacks, or hyperventilation. Far from being a diagnosis of exclusion, it is one of the most prevalent causes of chronic dizziness in expert vestibular practice, yet it remains systematically underdiagnosed, undertreated, and misattributed to structural inner-ear disease [1,2,3]. The vestibular physician occupies a unique position to identify this overlap, characterise it mechanistically, and deliver or coordinate evidence-based management that neither pure otology nor pure psychiatry typically provides [4,5].

Anxiety disorders are the most prevalent mental health conditions worldwide, affecting approximately 13–17% of the general population, with panic disorder affecting 2–5% and generalised anxiety disorder (GAD) a further 5–7% [16]. Among patients presenting to vestibular clinics, the prevalence of anxiety disorders rises dramatically: rates of panic disorder in dizziness-referral populations are five to fifteen times those in the general community [12,23]. Conversely, dizziness is the most common somatic symptom reported by patients with panic disorder, occurring in 65–80% of panic attacks across multiple studies, and persistent background dizziness between attacks is reported by over half of affected patients [6,23,45]. These bidirectional prevalence data alone make the vestibular-anxiety interface one of the highest-yield areas of clinical practice for any vestibular physician [2,3].

Hyperventilation syndrome (HVS) — chronic or episodic overbreathing sufficient to cause hypocapnia — is a closely related, mechanistically distinct contributor to anxiety-driven dizziness. Community prevalence estimates for HVS range from 6–11% among adults presenting with unexplained somatic complaints, with females affected approximately twice as often as males [13]. Hyperventilation is both a physiological correlate of acute anxiety and a precipitant of its own independent dizziness syndrome via cerebral vasoconstriction, altered neuronal excitability, and direct effects on vestibular afferent firing [13,47].

Table 1. Epidemiology of anxiety disorders and dizziness in the clinical context.

Measure	Value	Notes
Panic disorder in general population	2–5%	Lifetime prevalence [16]
Panic disorder in dizziness clinic patients	10–30%	5–15x general population [12,23]
Dizziness in panic disorder patients	65–80% of attacks	Most common somatic symptom [6,23]
GAD in vestibular clinic populations	15–25%	Underdiagnosed — majority presenting as persistent dizziness [5,22]
Hyperventilation syndrome prevalence	6–11%	Among adults with unexplained somatic complaints [13]

The clinical and functional burden is substantial. Patients with anxiety-related dizziness report higher Dizziness Handicap Inventory (DHI) scores than patients with many structural vestibular disorders, drive greater healthcare utilisation, and have longer time-to-diagnosis trajectories — often more than three years from symptom onset to correct diagnosis [2,22,33]. Avoidance behaviour is a key driver of disability: fear of triggering dizziness in social, occupational, or transport contexts progressively narrows the patient's world, reinforcing both the anxiety and the vestibular sensitivity that perpetuates symptoms [30,42]. The vestibular physician who recognises this dynamic early can prevent years of escalating investigations and treatment with vestibular suppressants that do not address the underlying mechanism [1,4,36].

Terminology has evolved over decades. Phobic postural vertigo (Brandt, 1994) described a syndrome of postural dizziness aggravated by specific situations and associated with anxiety and avoidance; chronic subjective dizziness (Staab, 2007) emphasised the perceptual and functional component; persistent postural-perceptual dizziness (PPPD, Barany Society 2017) now serves as the overarching functional dizziness label [10,20,22,41]. Anxiety-related dizziness as discussed in this review refers to presentations

in which a diagnosed anxiety disorder — most commonly panic disorder or GAD — is the primary or co-primary driver of vestibular symptoms, and where the management approach is necessarily psychiatric alongside vestibular. PPPD and anxiety-related dizziness overlap significantly, and many patients fulfil criteria for both; this review highlights the distinctions most relevant to the vestibular physician [10,20].

□ **Key Point:** Anxiety disorders are five to fifteen times more prevalent in vestibular clinic populations than in the general community. Every vestibular physician must have a systematic approach to identifying, characterising, and managing the anxiety-dizziness spectrum.

II. Pathophysiology — Vestibular-Autonomic Interactions and the Anxiety-Dizziness Loop

The neuroanatomical substrate for anxiety-vestibular co-morbidity is not coincidental but structural: vestibular and anxiety-related neural circuits share extensive brainstem, thalamic, and limbic connections [3,8]. Understanding these connections at a mechanistic level is essential for the vestibular physician, who must explain the symptom biology to patients, select the correct management modalities, and distinguish anxiety-amplified vestibular symptoms from primary structural disease [3,5,9].

Shared neuroanatomy

The parabrachial nucleus (PBN) of the dorsolateral pons occupies a critical relay position, receiving direct projections from the vestibular nuclei and projecting in turn to the amygdala, hypothalamus, and locus coeruleus [3,8]. The amygdala — the brain's primary fear-appraisal structure — receives vestibular input both directly via the PBN and indirectly via the thalamo-cortical stream, enabling vestibular perturbations to activate the fear network at a pre-cognitive level [3,8,9]. The locus coeruleus norepinephrine (NE) system, the principal driver of arousal and the fight-or-flight response, receives input from both the vestibular nuclei and the PBN, and projects back to the vestibular cortex (parietoinsular cortex, PIVC), providing a direct autonomic amplification pathway for vestibular symptoms [3,8]. Reciprocal projections from the amygdala to the vestibular nuclei complete the loop, meaning that elevated anxiety state directly modulates vestibular nucleus excitability [3,28].

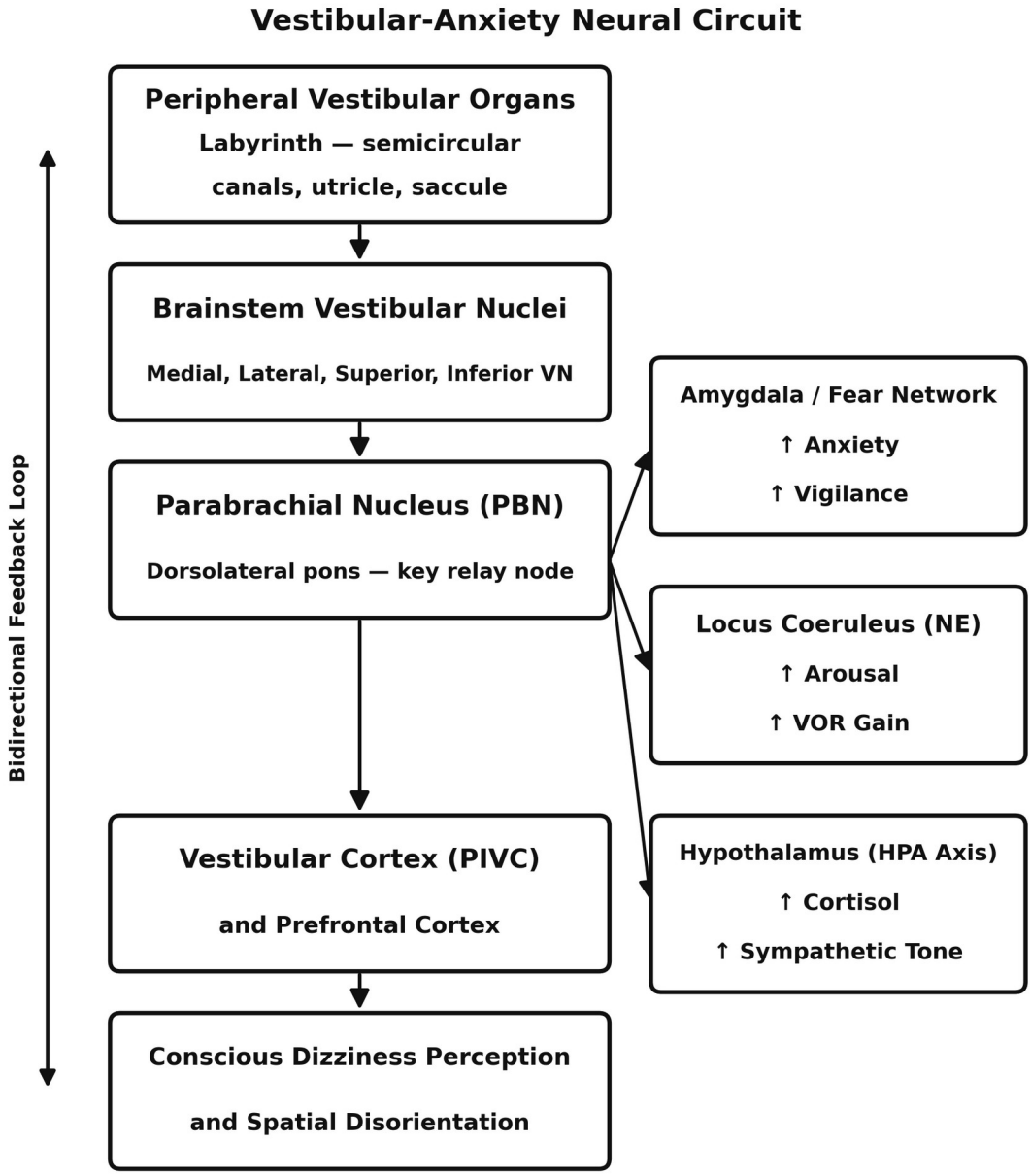

Figure 1. Simplified vestibular-anxiety neural circuit — key nodes and projection pathways linking the labyrinth to the fear network, autonomic output, and conscious dizziness perception.
Source: Adapted from Balaban and Thayer [3] and Gorman et al. [8].

Three explanatory models

Three non-mutually-exclusive models have been proposed to explain the association between panic disorder and vestibular dysfunction, each with distinct clinical implications [23].

- **Psychosomatic model —** Panic disorder is primary. Anxiety and autonomic arousal drive hyperventilation and sympathetic activation, which in turn produce dizziness via hypocapnia-induced cerebral vasoconstriction, altered vestibular afferent firing, and heightened somatic vigilance. The vestibular symptom is the consequence, not the cause [23,42].
- **Somatopsychic model —** Primary vestibular dysfunction — often subclinical or resolved by the time of presentation — triggers anxiety as a secondary adaptive response. Chronic postural instability, spatial disorientation, and movement-triggered symptoms generate fear, avoidance, and ultimately

panic disorder or GAD [1,23,43]. This is the dominant pathway in patients whose anxiety clearly followed a precipitating vestibular event.

- **Network alarm theory (Gorman et al.) —** An abnormally sensitised fear network centred on the amygdala-PBN-locus coeruleus circuit generates conditioned panic responses to normal vestibular stimuli. Slightly aberrant vestibular signals that a healthy individual ignores trigger disproportionate fear and autonomic arousal in a predisposed patient. This model best accounts for the overlap in neuroimaging, neurophysiology, and pharmacological response between vestibular dysfunction and panic disorder [8,9,23].

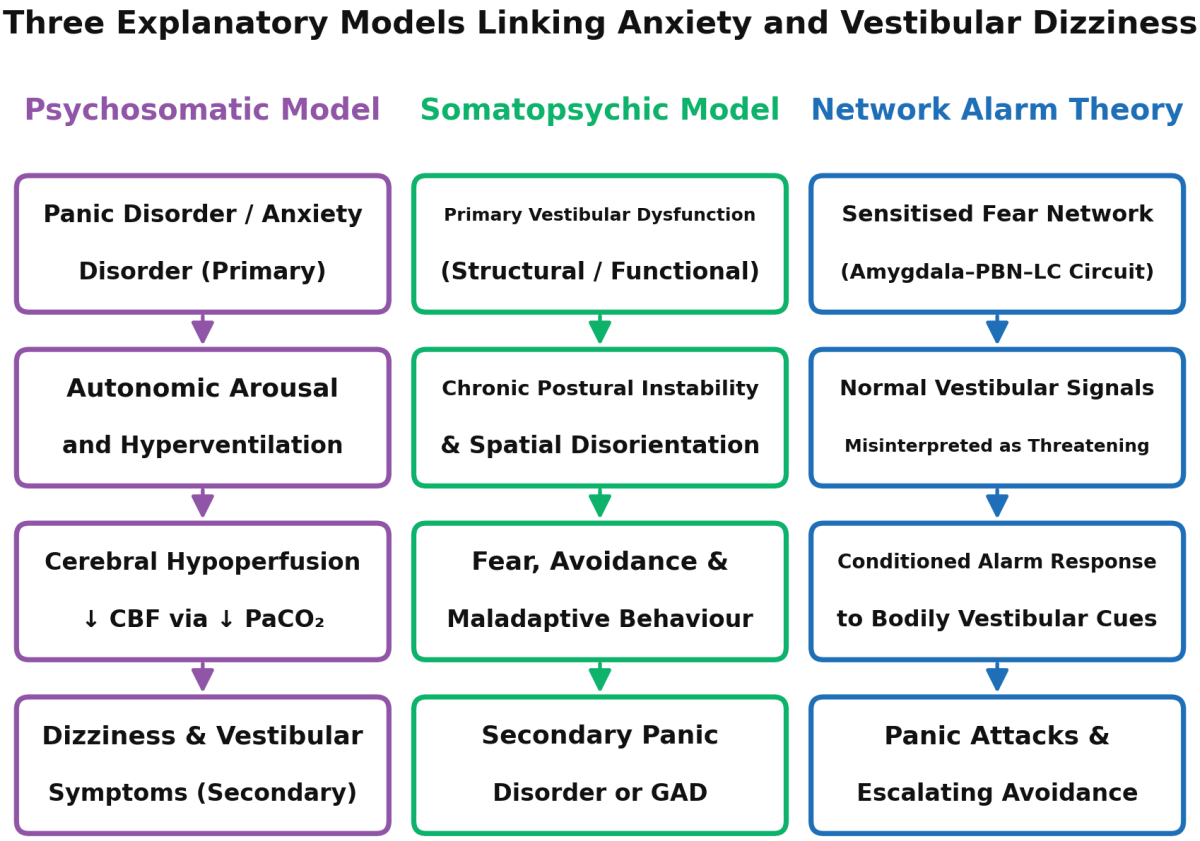

Figure 2. The three explanatory models linking anxiety and vestibular dizziness — psychosomatic, somatopsychic, and network alarm theory.
Source: Adapted from Asmundson et al. [23] and Gorman et al. [8].

Autonomic arousal and vestibular sensitisation

Acute anxiety activates the hypothalamic-pituitary-adrenal (HPA) axis and the sympathoadrenal system, producing elevated circulating cortisol, adrenaline, and norepinephrine [8]. These neurohumoral changes have direct effects on the vestibular periphery and central processing: elevated NE increases the gain of the vestibulo-ocular reflex (VOR) and amplifies the neural response to vestibular perturbation; elevated cortisol impairs habituation in vestibular compensation circuits; and heightened sympathetic tone alters cochlear blood flow with potential effects on endolymph composition [3,28,47]. The net effect is a vestibular system that is genuinely more sensitive to motion and positional change in an anxious patient, not merely one that is psychologically misinterpreting normal sensations [28,47].

This sensitisation is measurable. Studies using stabilometry and dynamic posturography demonstrate increased postural sway in patients with panic disorder compared with controls, and performance further deteriorates under conditions of reduced sensory input — analogous to the anxious sway seen in patients asked to stand on foam with eyes closed [15,28]. VOR gain asymmetries and postural control deficits

normalise with successful anxiety treatment, confirming their functional rather than structural basis [15,36,47].

Hyperventilation mechanism

Hyperventilation produces dizziness through a distinct but related pathway centred on hypocapnia. Rapid or deep breathing lowers arterial partial pressure of carbon dioxide (PaCO2) below the normal range of 35–45 mmHg; cerebral blood flow (CBF) decreases by approximately 2% per mmHg fall in PaCO2, so a drop from 40 to 25 mmHg — not unusual during acute hyperventilation — reduces CBF by roughly 30% [13]. This level of cerebral hypoperfusion is sufficient to cause dizziness, visual disturbance, perioral and extremity paraesthesias, and chest tightness — symptoms that patients and clinicians alike often attribute to a vestibular cause [13,47].

Alkalosis from hypocapnia also lowers ionised calcium, increasing neuronal membrane excitability and manifesting as perioral and hand paraesthesias, carpopedal spasm, and sometimes frank tetany [13]. Vestibular afferents are not immune: hyperventilation-induced nystagmus (HVIN) is a well-documented phenomenon in patients with vestibular asymmetry, in whom the differential alkalosis-mediated change in afferent firing rates on the two sides unmasks a latent canal imbalance [17]. In patients without a pre-existing vestibular lesion, HVIN may still be induced by severe hyperventilation via direct cortical suppression of vestibular compensatory mechanisms [17,47]. Voluntary hyperventilation testing in clinic is therefore both a diagnostic and a psychoeducational tool: reproducing the patient's own symptoms through voluntary overbreathing, then resolving them with rebreathing or slow breathing, provides compelling evidence of the mechanism to an often sceptical patient [13].

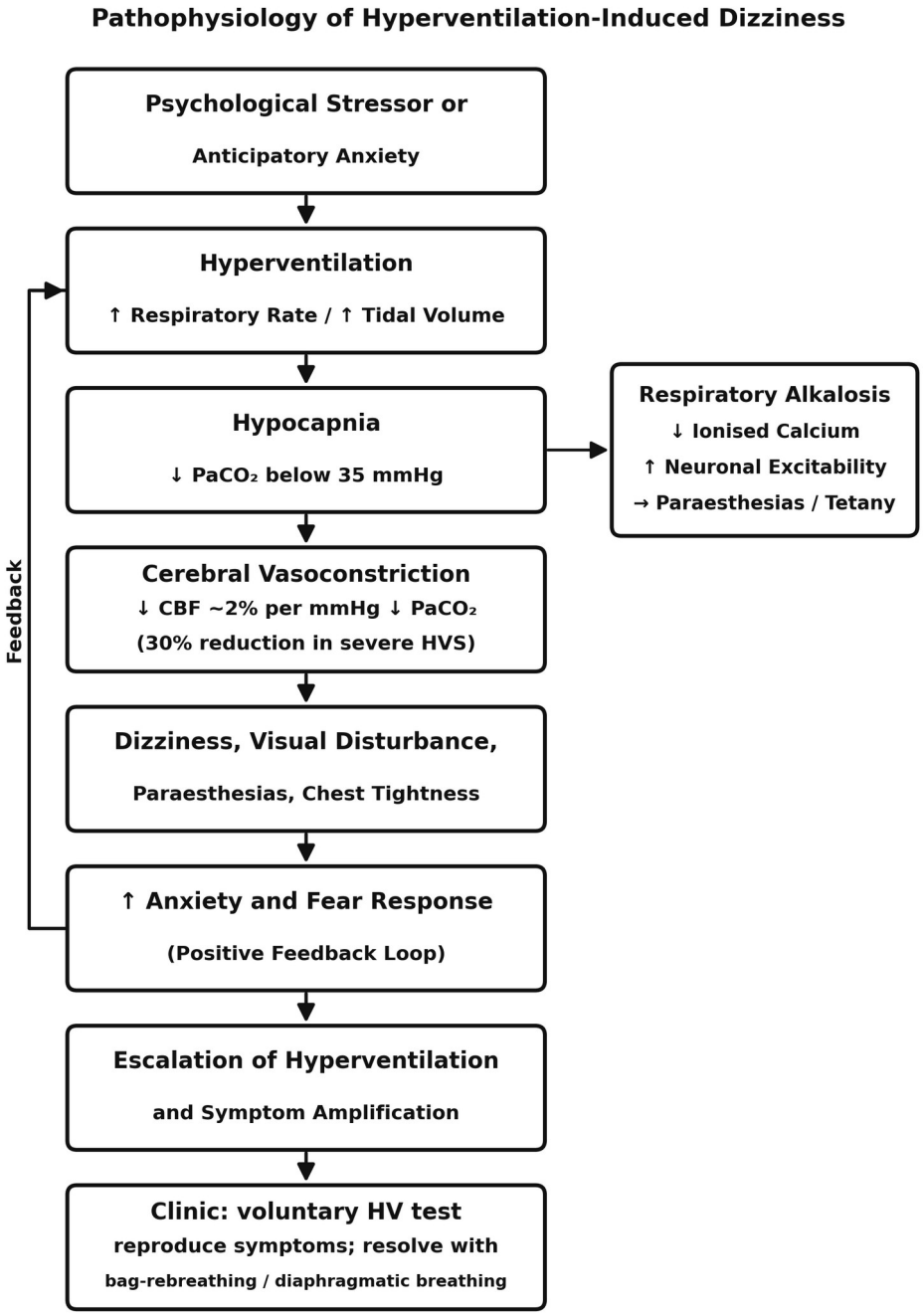

Figure 3. Pathophysiology of hyperventilation-induced dizziness — the positive feedback loop between anxiety, overbreathing, hypocapnia, and escalating vestibular symptoms.

Source: Adapted from MedLink Neurology hyperventilation syndrome review and Zwergal and Dieterich [17].

Perceptual amplification and space and motion discomfort

Space and motion discomfort (SMD) is a consistently reported feature of panic disorder with vestibular dysfunction and refers to the situational specificity of symptoms in environments characterised by excessive or conflicting visual-vestibular input: busy supermarkets, escalators, crowds, open spaces, motorway driving, and shopping centres are classic precipitants [6,23]. The mechanism is perceptual: in patients whose vestibular-anxiety circuit is sensitised, situations demanding high reliance on visual or vestibular input (rather than proprioceptive anchoring) trigger disproportionate spatial disorientation and fear responses [6,30]. SMD is not anxiety avoidance alone; it represents a genuine perceptual deficit in multi-sensory integration that is amenable to specific rehabilitative intervention [30,40].

□ **Clinical Insight:** Vestibular symptoms in anxious patients are neurophysiologically real —

elevated norepinephrine increases VOR gain, hypocapnia reduces cerebral blood flow, and amygdala feedback modulates vestibular nucleus excitability. These are not imagined symptoms. Validating the mechanism to the patient is a therapeutic act, not a concession.

III. Clinical Phenotypes — Panic Disorder, Generalised Anxiety, and Hyperventilation Syndrome

Panic disorder with vestibular features

Panic disorder (PD) is defined by DSM-5 as recurrent unexpected panic attacks — abrupt surges of intense fear or discomfort reaching a peak within minutes — accompanied by persistent worry about future attacks or maladaptive behavioural change [5]. Dizziness is one of the thirteen defined DSM-5 panic attack symptoms and is among the most common, reported by 65–80% of patients [5,6,23]. In the vestibular clinic context, PD most often presents as episodic dizziness with associated palpitations, dyspnoea, chest tightness, paraesthesias, and diaphoresis — a cluster that strongly resembles a peripheral vestibular attack and is regularly referred from emergency departments, cardiology, or neurology after normal ECG and imaging [6,12,31].

Three distinguishing features separate PD from structural vestibular disease in the history. First, unprovoked attacks are characteristic: the dizziness of PD arises without a clear positional, movement, or acoustic trigger, arising instead in association with psychosocial stress, anticipation of feared situations, or apparently at random — particularly at night or in apparently safe environments [5,6]. Second, multi-system symptoms are prominent: vestibular disease primarily causes vestibular symptoms; PD causes dizziness accompanied by cardiac, respiratory, gastrointestinal, and dermatological symptoms simultaneously [5,23]. Third, situational clustering is striking: attacks cluster in environments associated with SMD as described above — escalators, crowds, bridges, driving — not in the positional or acceleratory patterns of BPPV, Meniere's, or vestibular neuritis [6,30].

Generalised anxiety disorder and chronic dizziness

GAD presents as pervasive, excessive worry across multiple domains that is difficult to control, accompanied by restlessness, fatigue, difficulty concentrating, irritability, muscle tension, and sleep disturbance [5]. In the vestibular clinic, GAD typically presents as chronic, persistent background dizziness — not episodic — often described as rocking, floating, unsteadiness, or a sense of being off-balance that is present most of the day, every day [2,10,22]. This presentation overlaps heavily with PPPD, and many patients with GAD meet criteria for both diagnoses simultaneously [10,20].

The chronic dizziness of GAD is maintained by a combination of perpetual low-level autonomic arousal, heightened somatic vigilance (hypervigilance to vestibular sensations), and the downstream consequences of avoidance and deconditioning [22,30,42]. GAD patients commonly describe worsening in response to stress, fatigue, or social demands, and temporary relief in distraction, relaxation, or controlled environments — a pattern that differs from structural disease but is easily misread as malingering or somatisation [2,22]. The Hospital Anxiety and Depression Scale (HADS) and the DHI, when administered routinely, reliably identify this population before extensive vestibular investigations [33,46].

Hyperventilation syndrome

HVS is characterised by recurrent episodes of inappropriate hyperventilation — either acutely or chronically at low amplitude — that generate symptoms in excess of those attributable to organic disease [13]. The clinical picture includes dizziness, light-headedness, visual disturbance (blurring, tunnel vision), perioral and extremity paraesthesias, chest tightness, palpitations, and fatigue [13]. Unlike panic disorder, the subjective emotional arousal is often secondary rather than primary — many HVS patients report more respiratory and somatic distress than fear per se, complicating the psychological characterisation [13].

The Nijmegen Questionnaire (NQ) is the standard screening instrument for HVS in clinic: sixteen items each scored 0–4 on symptom frequency; a total score of 23 or more has sensitivity of approximately 68% and specificity of 92% for HVS [13]. The hyperventilation provocation test — voluntary overbreathing at 30 breaths/minute for three minutes in clinic — reproduces the patient's characteristic symptoms in a controlled setting and can be reversed by bag-rebreathing or controlled abdominal breathing, providing both diagnosis and psychoeducation in a single encounter [13]. End-tidal CO2 monitoring during the test (target below 30 mmHg) confirms the physiological correlate [13,47].

Agoraphobia and space and motion discomfort

Agoraphobia — marked fear of situations where escape might be difficult or help unavailable — frequently complicates panic disorder with vestibular features and is itself a major driver of functional impairment [5]. In the vestibular context, agoraphobic avoidance specifically targets environments with high SMD load: shopping centres, public transport, bridges, open squares, and unfamiliar buildings [6,30]. The overlap between agoraphobia and vestibular avoidance behaviour is so substantial that the two are difficult to distinguish on clinical history alone — the vestibular examination and psychometric tools are necessary to decompose the contribution of each [6,23,30].

Table 2. Clinical features differentiating the three primary anxiety-related dizziness phenotypes.

Feature	Panic Disorder	GAD / Chronic Anxiety	Hyperventilation Syndrome
Dizziness pattern	Episodic attacks, unprovoked or situationally cued	Persistent daily unsteadiness / rocking	Episodic or chronic; provoked by stress / exertion
Associated symptoms	Palpitations, dyspnoea, chest pain, paraesthesias, diaphoresis	Muscle tension, fatigue, insomnia, restlessness	Perioral paraesthesias, chest tightness, dyspnoea, carpopedal spasm
Triggers	SMD environments, stress, anticipatory anxiety; unprovoked	Psychosocial stress, fatigue, social demands	Exertion, anxiety, emotional arousal; chronic low-amplitude pattern
Key diagnostic tool	DSM-5 criteria; HADS-A; SMDQ	DSM-5 GAD criteria; HADS-A; Worry scale	Nijmegen Questionnaire; hyperventilation provocation test

□ **Clinical Pearl:** The history 'I get dizzy in supermarkets, on escalators, and when driving on motorways' is the space and motion discomfort signature. It is not exclusively PPPD — it is also the situational profile of panic disorder with vestibular features and agoraphobia.

IV. Diagnostic Criteria, Assessment Tools, and Clinical Red Flags

DSM-5 diagnostic criteria

Panic disorder requires: (a) recurrent unexpected panic attacks (abrupt surge of intense fear or discomfort reaching a peak within minutes, with 4 or more of the 13 defined symptoms); (b) at least one attack followed by 1 month or more of persistent worry about future attacks or their consequences, or significant maladaptive behavioural change; and (c) the disturbance is not attributable to substances, a medical condition, or better explained by another mental disorder [5,14]. Crucially, panic attacks can be expected (cued) or unexpected — and agoraphobia is coded separately and requires its own assessment [5].

GAD diagnosis requires: (a) excessive anxiety and worry about multiple topics on more days than not for 6 months or more; (b) difficulty controlling worry; (c) 3 or more of six associated symptoms in adults: restlessness, easy fatigability, concentration difficulty, irritability, muscle tension, sleep disturbance; and (d) clinically significant distress or functional impairment not attributable to a substance or medical condition [5]. In the vestibular clinic population, sleep disturbance, muscle tension, and restlessness are commonly present and easily overlooked when the presenting complaint is dizziness [2,22].

Psychometric instruments

A standard battery of psychometric instruments, administered as a patient-completed questionnaire set before the clinical encounter, provides objective severity data and enables efficient screening without extending the consultation time [33,46]. The following instruments have the strongest evidence in the vestibular clinic context:

- **Dizziness Handicap Inventory (DHI) —** 25 items assessing physical, emotional, and functional impact of dizziness. Total scores of 29–60 indicate moderate handicap; above 60 indicate severe handicap. Emotional sub-scores are particularly elevated in anxiety-related dizziness [33,46].
- **Hospital Anxiety and Depression Scale (HADS) —** 14-item scale, 7 items each for anxiety (HADS-A) and depression (HADS-D). Scores of 8–10 suggest probable case; 11 or more suggest definite case. Sensitivity 80%, specificity 85% for anxiety disorders in vestibular populations [33,46].
- **Nijmegen Questionnaire (NQ) —** 16-item self-report for hyperventilation symptoms. Score 23 or above indicates likely HVS (sensitivity 68%, specificity 92%) [13].
- **Space and Motion Discomfort Questionnaire (SMDQ) —** Quantifies situational avoidance in SMD-provoking environments. High scores correlate with panic disorder and agoraphobia in vestibular clinic populations [6,30].
- **Panic Disorder Severity Scale (PDSS) —** 7-item clinician-administered scale for panic disorder. Useful for tracking treatment response; a reduction of 40% or more represents clinically significant improvement [5,52].

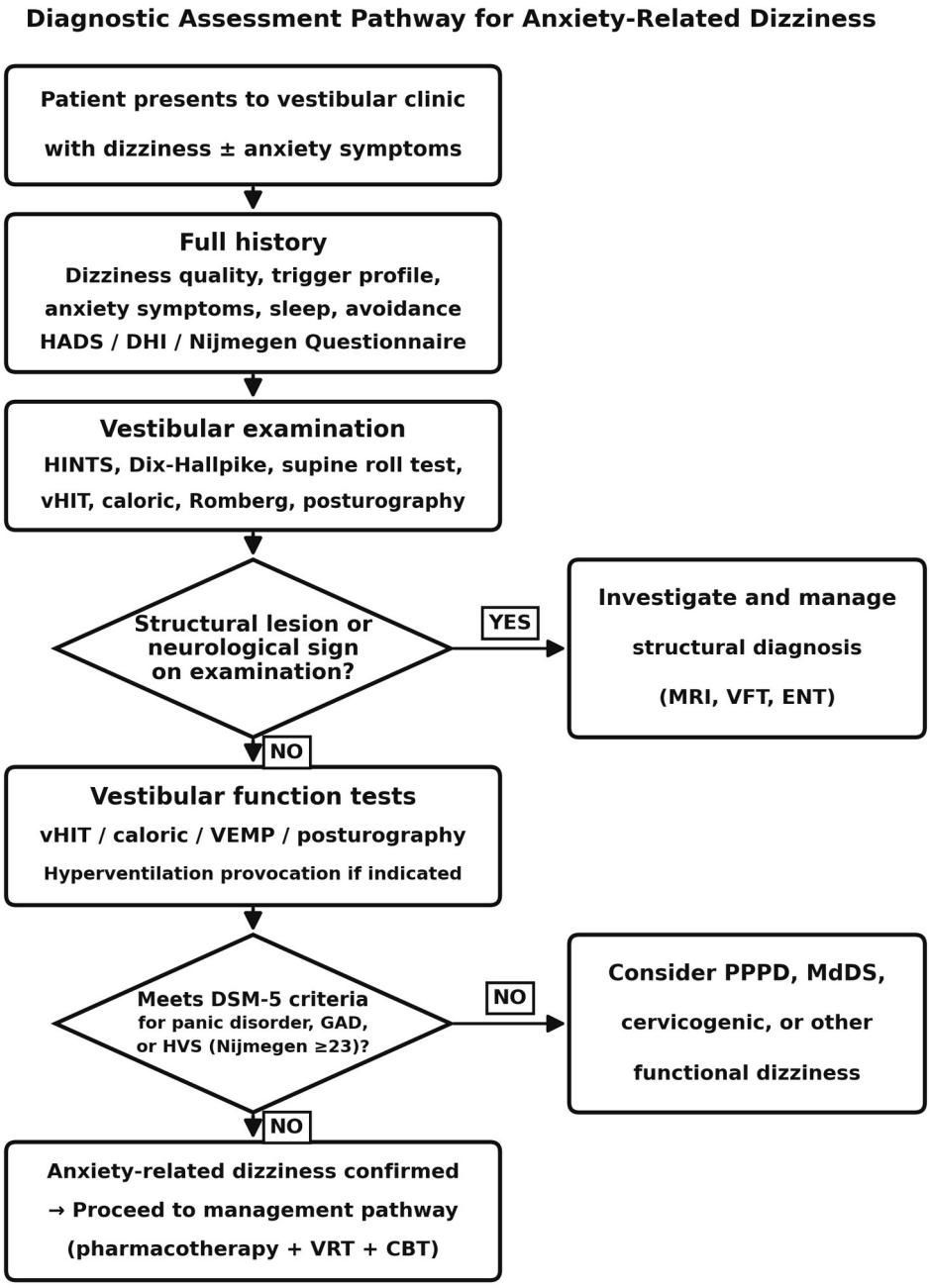

Figure 4. Diagnostic assessment pathway for suspected anxiety-related dizziness in the vestibular clinic setting.
Source: Adapted from Staab et al. [10] and Asmundson et al. [23].

Red flags requiring urgent exclusion

Before attributing dizziness to anxiety, the vestibular physician must actively exclude structural vestibular and neurological disease, as anxiety frequently co-exists with organic pathology and can obscure it. The following features mandate investigation before an anxiety diagnosis is confirmed [17,29]:

- New or progressive unilateral hearing loss — Meniere's disease, vestibular schwannoma, or autoimmune inner-ear disease.
- Focal neurological signs — cranial neuropathy, INO, ataxia, diplopia, dysarthria, or limb signs require MRI.
- Prominent spontaneous or direction-changing nystagmus — central vestibular pathology until proven otherwise.

- Acute onset severe vertigo at rest — AICA or PICA infarction, labyrinthitis, or superior canal dehiscence.
- Orthostatic component — autonomic failure, POTS, or medication effect, not anxiety.
- Onset after head or neck trauma — perilymph fistula, BPPV, or cervicogenic dizziness [34].

□ **Important:** A diagnosis of anxiety-related dizziness is a positive clinical diagnosis based on meeting DSM-5 anxiety criteria plus vestibular history and examination findings consistent with functional or sensitised vestibular processing. It is NOT a diagnosis of exclusion, but structural vestibular and posterior-fossa disease must be actively ruled out before it is confirmed.

V. Investigations — Vestibular Function Tests and Psychometric Instruments

Vestibular function testing (VFT) in anxiety-related dizziness occupies a nuanced position: the majority of patients with primary anxiety-driven dizziness have normal or near-normal VFT results, which is diagnostically informative in its own right [1,2,36]. However, subclinical peripheral vestibular dysfunction — detectable only on formal testing — may co-exist and contribute a somatopsychic pathway to anxiety [23]. Additionally, VFT objectively excludes compensated unilateral vestibulopathy that has been mislabelled as anxiety for years [15,21,29].

Video head impulse test (vHIT)

vHIT is the most informative first-tier test for unilateral canal hypofunction. In anxiety-related dizziness, VOR gain is typically normal bilaterally with no catch-up saccades, which supports a central or functional mechanism [15,29]. Subtly reduced gain with covert saccades may signal a compensated prior unilateral vestibulopathy — important because this represents a structural precipitant for the anxiety through the somatopsychic pathway [29]. Asymmetric VOR gain with overt refixation saccades in a patient labelled as anxiety demands reassessment for active structural disease [15].

Caloric testing

Caloric testing assesses horizontal canal function at low frequencies. Canal paresis (CP) greater than 25% suggests unilateral hypofunction and warrants structural investigation; directional preponderance (DP) greater than 30% may indicate an asymmetric tonic bias from central pathology or ongoing compensation [29]. In anxiety-related dizziness, calorics are typically normal or show mild, non-specific DP related to sympathetic tone. A normal caloric response alongside a positive anxiety screen is diagnostically reassuring but not sufficient alone to exclude all vestibular pathology [15,29].

VEMPs and posturography

Cervical VEMPs (cVEMPs) and ocular VEMPs (oVEMPs) assess saccular and utricular function respectively. In anxiety-related dizziness without structural disease, VEMPs are typically normal in amplitude and latency. Heightened VEMP amplitudes have been reported in some anxious patients, possibly reflecting generalised sympathetic-mediated neural gain — an interesting correlate of the documented vestibular sensitisation [47]. Computerised dynamic posturography (CDP) in anxious patients classically shows increased sway on sensory organisation conditions with reduced or conflicting sensory input — the aphysiological pattern where patients perform worse with eyes closed on a stable surface than on conditions that are genuinely more challenging for a structural vestibular lesion [15].

Hyperventilation testing

Voluntary hyperventilation at 30 breaths per minute for three minutes, performed in a controlled clinic setting with resuscitation equipment available, is the definitive diagnostic test for HVS and is informative in suspected panic disorder [13]. A positive test reproduces the patient's characteristic symptoms within 2–3 minutes and is reversed by bag-rebreathing or controlled breathing. End-tidal CO2 monitoring (target below 30 mmHg for test validity) adds objectivity. HVIN may emerge during hyperventilation in patients with a pre-existing latent vestibular asymmetry, providing a visual correlate that reinforces the mechanistic

explanation [17]. The hyperventilation provocation test is contraindicated in patients with a history of epilepsy, known ischaemic heart or cerebrovascular disease, or severe hypertension [13].

Neuroimaging

Routine MRI is not required in patients meeting criteria for anxiety-related dizziness with normal examination and VFT. However, MRI is indicated when red flags are present — any focal neurological sign, unilateral hearing loss, progressive course, or atypical nystagmus pattern [17,29]. Brain MRI in patients with established anxiety disorders and dizziness may show non-specific white matter changes or subtle hippocampal volume reduction, findings that are well documented in chronic anxiety states but do not constitute a vestibular diagnosis [26,27].

Table 3. Vestibular function test findings in anxiety-related dizziness versus structural unilateral vestibulopathy.

Test	Anxiety-Related Dizziness	Structural Unilateral Vestibulopathy
vHIT (VOR gain)	Normal bilaterally, no saccades	Reduced gain ipsilesional, catch-up saccades
Calorics	Normal or mild DP; no CP	CP over 25% ipsilesional
Posturography (CDP)	Increased sway on visual conflict; aphysiological pattern	Pattern matches peripheral or central loss
VEMP	Normal amplitude; possibly mildly elevated	Asymmetric or absent ipsilesionally
Hyperventilation test	Positive — reproduces symptoms; HVIN may emerge	HVIN only if latent asymmetry present; symptoms differ

□ **Key Point:** Normal vHIT and calorics in a patient with chronic dizziness and high HADS-A is strong diagnostic support for anxiety-related dizziness. The combination of normal structural testing with elevated anxiety scores is more informative than either in isolation.

VI. Differential Diagnosis

The differential diagnosis of anxiety-related dizziness is broad and mirrors the differential for any chronic vestibular presentation. The vestibular physician's task is threefold: exclude structural disease that requires disease-specific treatment; correctly sub-classify functional vestibular presentations (PPPD, MdDS, HVS, phobic postural vertigo) where treatment differs; and identify co-morbid anxiety in patients with confirmed structural disease who require simultaneous psychiatric management [1,5,10].

Persistent postural-perceptual dizziness (PPPD)

PPPD is the closest nosological neighbour of anxiety-related dizziness and shares pathophysiology, phenomenology, and treatment approach [10,20]. PPPD requires symptoms of dizziness, unsteadiness, or non-spinning vertigo present on most days for 3 months or more, exacerbated by upright posture, motion, and complex visual environments [10]. Anxiety comorbidity is present in 50–70% of PPPD patients [10,20]. The key distinction: in anxiety-related dizziness, the anxiety disorder is primary and the dizziness is predominantly a symptom of the anxiety; in PPPD, the functional dizziness is primary and anxiety is a maintaining factor or co-morbidity. In practice, many patients fulfil both sets of criteria and benefit from treatment addressing both [10,20,40].

Mal de debarquement syndrome (MdDS)

MdDS is characterised by persistent rocking, swaying, or bobbing dizziness — typically following passive motion exposure (sea travel, air travel) — that is relieved by motion and worsens with stationary posture [41]. The rocking quality and the paradoxical relief with movement distinguish MdDS from anxiety-related dizziness, which lacks this characteristic motion-relief pattern. Anxiety co-morbidity in MdDS is common but secondary [41].

Vestibular migraine (VM)

VM frequently coexists with anxiety disorders and shares features of episodic dizziness, SMD-type aggravation, and chronic background imbalance. Distinguishing features: migraine headache or typical migraine accompaniments (photophobia, phonophobia, osmophobia) associated with at least 50% of vestibular episodes; duration of episodes 5 minutes to 72 hours; and response to migraine-specific treatment [18,44]. VM is not accompanied by the multi-system autonomic features of panic disorder, and the Nijmegen Questionnaire is typically negative [11,18].

Orthostatic dizziness and POTS

Postural orthostatic tachycardia syndrome (POTS) is easily confused with anxiety because it presents with palpitations, dizziness, anxiety-like symptoms, and hyperventilation on standing — all triggered by upright posture [24]. Differentiating features: in POTS, symptoms are reliably position-dependent, heart rate rises 30 bpm or more on standing (or 40 bpm or more in those aged under 19), and symptoms are partially relieved by lying down. In anxiety-related dizziness, the positional component is typically less prominent and the multi-system autonomic storm of panic attacks is not consistently position-triggered [24].

Structural vestibular disease with secondary anxiety

Approximately 40–60% of patients with confirmed unilateral vestibular lesions develop clinically significant anxiety within two years of onset — the somatopsychic pathway [46,48]. This group requires both disease-specific vestibular management and concurrent anxiety treatment [49]. The danger is attributing the anxiety component to the vestibular disease alone, neglecting the independent psychiatric disorder that will maintain dizziness even after the vestibular lesion has compensated [3,46].

Table 4. Differential diagnosis of anxiety-related dizziness — key discriminating features.

Diagnosis	Key distinguishing features	Primary management
Panic disorder	Episodic attacks, 4 or more DSM-5 panic symptoms, SMD, agoraphobia	SSRI/SNRI + CBT
GAD	Chronic daily dizziness, pervasive worry, muscle tension, HADS-A 8 or above	SSRI/SNRI + CBT + VRT
HVS	Paraesthesias, Nijmegen score 23 or above, symptoms reproduced and reversed in clinic	Breathing retraining + CBT
PPPD	Persistent 3 months or more, upright posture triggers, motion-visual sensitivity	VRT + SSRI + CBT
MdDS	Post-motion onset, rocking/bobbing quality, relieved by motion	Optokinetic retraining
VM	Headache history, migraine accompaniments, 5 min–72 hr episodes	Migraine prophylaxis
POTS	Positional HR rise 30 bpm or more on standing, lying relief	Autonomic rehabilitation, fludrocortisone

□ **Clinical Insight:** When a patient with confirmed structural vestibular disease continues to report disproportionate functional impairment after vestibular compensation appears complete, always screen for secondary anxiety disorder. Treating the anxiety is often what resolves the residual dizziness.

VII. Pharmacological Management

Pharmacological treatment of anxiety-related dizziness targets the underlying anxiety disorder using agents with established efficacy in panic disorder, GAD, or both [35,36,52]. Vestibular suppressants — antihistamines, benzodiazepines used chronically, and meclizine — are inappropriate as primary management: they impair central vestibular compensation, reinforce somatic vigilance, and generate dependency without addressing the anxiety substrate [1,36]. The serotonergic agents have the best evidence across the anxiety-vestibular spectrum and have the additional benefit of direct serotonergic

action within the vestibular nuclei and inner ear — a mechanism distinct from, and additive to, their anxiolytic effect [36].

Selective serotonin reuptake inhibitors (SSRIs)

SSRIs are first-line pharmacotherapy for both panic disorder and GAD, and have the most extensive evidence base in anxiety-related dizziness specifically [35,36,52]. Sertraline, escitalopram, paroxetine, and citalopram have all been studied in vestibular clinic populations with consistent results: reduction in dizziness frequency, severity, and functional impairment alongside improvement in anxiety scores [35,36,52].

Practical prescribing considerations for anxiety-related dizziness: SSRIs paradoxically worsen anxiety and dizziness transiently in the first 1–2 weeks of treatment due to initial serotonergic activation — a phenomenon particularly disruptive in patients whose presenting complaint is dizziness [36,52]. Starting at half the standard anxiety dose — sertraline 25 mg/d or escitalopram 5 mg/d — for the first two weeks before titrating to therapeutic levels substantially improves tolerability and adherence [35,52]. Patients must be counselled that dizziness may temporarily worsen before improving, and that the therapeutic window is 4–8 weeks [35,52]. Dose escalation to the upper end of the therapeutic range (sertraline 150–200 mg/d, escitalopram 20 mg/d) may be needed for full response in panic disorder; lower doses often suffice for GAD [35,36,52].

Serotonin-norepinephrine reuptake inhibitors (SNRIs)

Venlafaxine and duloxetine are effective alternatives and have dual serotonergic and noradrenergic activity [36,52]. Venlafaxine (37.5 mg/d starting dose, titrated to 75–150 mg/d) has shown superiority to placebo in both panic disorder and PPPD (chronic subjective dizziness), and is a reasonable first-choice in patients with comorbid depression or those who have failed an SSRI [36,52]. The noradrenergic component of SNRIs may be particularly relevant given the locus coeruleus role in the anxiety-vestibular circuit, though direct evidence for differential benefit is limited [3,36].

Benzodiazepines

Benzodiazepines (BZDs) — clonazepam, lorazepam, diazepam — have a limited but legitimate role in short-term management of acute panic attacks and severe anticipatory anxiety when commencing SSRI therapy [52]. Their use should be restricted to the shortest effective period (typically 4 weeks or fewer), at the lowest effective dose, with a clear tapering plan communicated to the patient from the outset [52]. Chronic BZD use in anxiety-related dizziness is firmly contraindicated: it impairs vestibular compensation, causes dependence and withdrawal symptoms that mimic vestibular symptoms, and creates a pharmacological maintenance cycle that prevents recovery [1,36,52]. The vestibular physician should actively identify and address existing BZD dependence as part of the assessment, as it is a common and underappreciated obstacle to improvement [36].

Beta-blockers and other agents

Propranolol and other non-selective beta-blockers are useful for the autonomic symptoms of panic — tachycardia, sweating, tremor — particularly in performance or situational anxiety contexts. They do not address the core anxiety disorder and should not be used as monotherapy [52]. Buspirone is a partial 5-HT1A agonist with specific efficacy for GAD and anxious rumination; it lacks anti-panic effect and has limited vestibular data but may be useful as an augmenter in treatment-resistant cases [52]. Pregabalin has evidence for GAD and is used as an augmenter in refractory cases; its sedating properties can worsen vestibular symptoms at higher doses [52]. Antihistamines such as promethazine and prochlorperazine should be reserved for acute severe vertigo episodes only; their role in chronic anxiety-related dizziness management is nil [1].

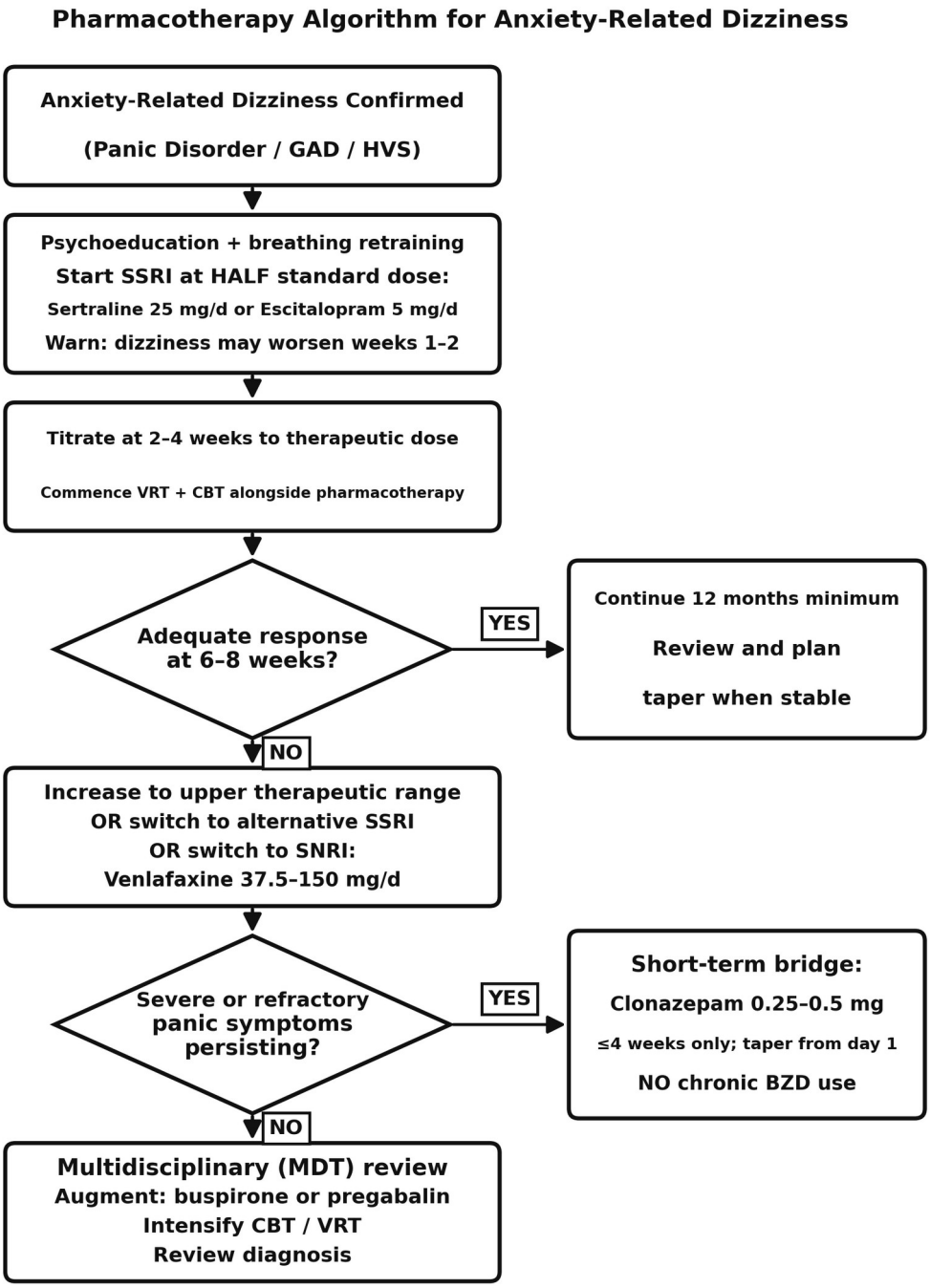

Figure 5. Pharmacotherapy algorithm for anxiety-related dizziness — stepwise approach from first-line SSRI through to expert augmentation strategies.

Source: Adapted from Staab et al. [36] and Campos et al. [52].

□ **Clinical Pearl:** Start the SSRI at half-dose for two weeks before titrating. The first-fortnight dizziness worsening with SSRIs in anxious vestibular patients is predictable and preventable with counselling. Patients who are not warned will discontinue treatment prematurely — often convinced that the tablet made their dizziness worse.

VIII. Non-Pharmacological Therapies — CBT, VRT, and Integrated Approaches

Non-pharmacological treatment is the cornerstone of management for anxiety-related dizziness and should be commenced alongside — not instead of — pharmacotherapy in the majority of patients [38,39,40,50]. The combination of vestibular rehabilitation therapy (VRT), cognitive behavioural therapy (CBT), and psychoeducation addresses the three maintaining mechanisms of the disorder: vestibular sensitisation, catastrophic cognitions, and avoidance behaviour respectively [38,39,40].

Psychoeducation

Psychoeducation is the first and most immediately accessible intervention and has a measurable therapeutic effect in its own right [39,40]. Explaining the mechanism — your dizziness is real; it is caused by your anxiety system amplifying normal vestibular signals rather than by an ear problem — reduces the fear of a missed diagnosis, decreases safety-seeking behaviour, and facilitates engagement with the treatment plan [40,42]. A brief structured explanation using the neural circuit model and, where applicable, a clinic hyperventilation demonstration, converts an abstract psychiatric label into a mechanistically comprehensible biological phenomenon — a distinction that matters enormously to the patient's therapeutic engagement [13,42].

Breathing retraining

For HVS specifically, and for anxiety-related dizziness broadly, diaphragmatic breathing retraining is the most immediately effective non-pharmacological intervention [13]. The technique — slow nasal breathing at 6–8 breaths per minute with active diaphragmatic excursion and passive exhalation — normalises PaCO2 within 2–4 minutes of a provoked episode and, with regular practice, raises the resting respiratory set-point above the hyperventilation threshold [13,50]. It is best taught by a trained physiotherapist or respiratory physiotherapist, supplemented by app-guided home practice. Capnography biofeedback (end-tidal CO2 display during sessions) accelerates acquisition of the skill in HVS patients [13].

Vestibular rehabilitation therapy (VRT)

VRT in anxiety-related dizziness targets the behavioural avoidance and the heightened sensory sensitivity maintained by non-use of vestibular and motion inputs [38,50]. The programme for anxious dizzy patients differs from VRT for structural vestibulopathy in two critical respects: first, graded exposure to SMD-provoking environments is added to the standard gaze-stabilisation and balance exercise programme; and second, the physiotherapist must have sufficient understanding of panic disorder and avoidance behaviour to manage the anxiety responses that emerge during exposure [7,38,40,50].

Evidence for VRT in anxiety-related dizziness: a 2023 Cochrane-level systematic review found small-to-moderate improvements in dizziness disability from VRT alone, and larger and more consistent reductions when VRT was combined with CBT or psychologically informed components [38]. A 2025 RCT found that VRT significantly improved quality of life in PPPD, particularly when pre-treatment HADS-A was elevated — suggesting that anxious patients paradoxically respond better to VRT than non-anxious PPPD patients when given an appropriately graduated programme [38].

Cognitive behavioural therapy (CBT)

CBT for anxiety-related dizziness targets the three components of the anxiety-avoidance cycle: catastrophic misinterpretation of vestibular sensations (cognitive restructuring); safety behaviours and avoidance (behavioural experiments and graduated exposure); and physiological arousal (relaxation and breathing techniques) [39,42,51]. The Clark cognitive model of panic (1986) provides the theoretical framework: perceived threat generates autonomic arousal; the somatic symptoms of arousal are misinterpreted as evidence of physical catastrophe; this misinterpretation amplifies arousal and perpetuates the attack [42,51].

Efficacy of CBT in anxiety-related dizziness: Holmberg et al. (2007) demonstrated significant reduction in avoidance and dizziness handicap at one-year follow-up after six sessions of individualised CBT for phobic postural vertigo [37]. Edelman et al. (2012) conducted an RCT of CBT versus control in chronic subjective dizziness, finding statistically significant and clinically meaningful reductions in DHI and anxiety at three months [39]. Interdisciplinary treatment programmes combining VRT and CBT achieve the largest treatment effects for anxiety-related dizziness, with effect sizes comparable to CBT for primary anxiety

disorders [40]. Group-based CBT formats are cost-effective alternatives to individual therapy and have demonstrated similar efficacy in PPPD and chronic dizziness populations [37,39].

Mindfulness-based interventions

Mindfulness-based stress reduction (MBSR) and mindfulness-based cognitive therapy (MBCT) have preliminary evidence in anxiety-related dizziness and are increasingly used as adjuncts to VRT and CBT [40]. The mechanism is decentring from vestibular symptoms — developing a non-judgemental observational stance toward dizziness sensations rather than reacting with catastrophic appraisal — which reduces the emotional amplification of vestibular signals [40]. MBSR is particularly useful in patients for whom traditional CBT is unavailable, refused, or failed, and as a relapse-prevention strategy [40].

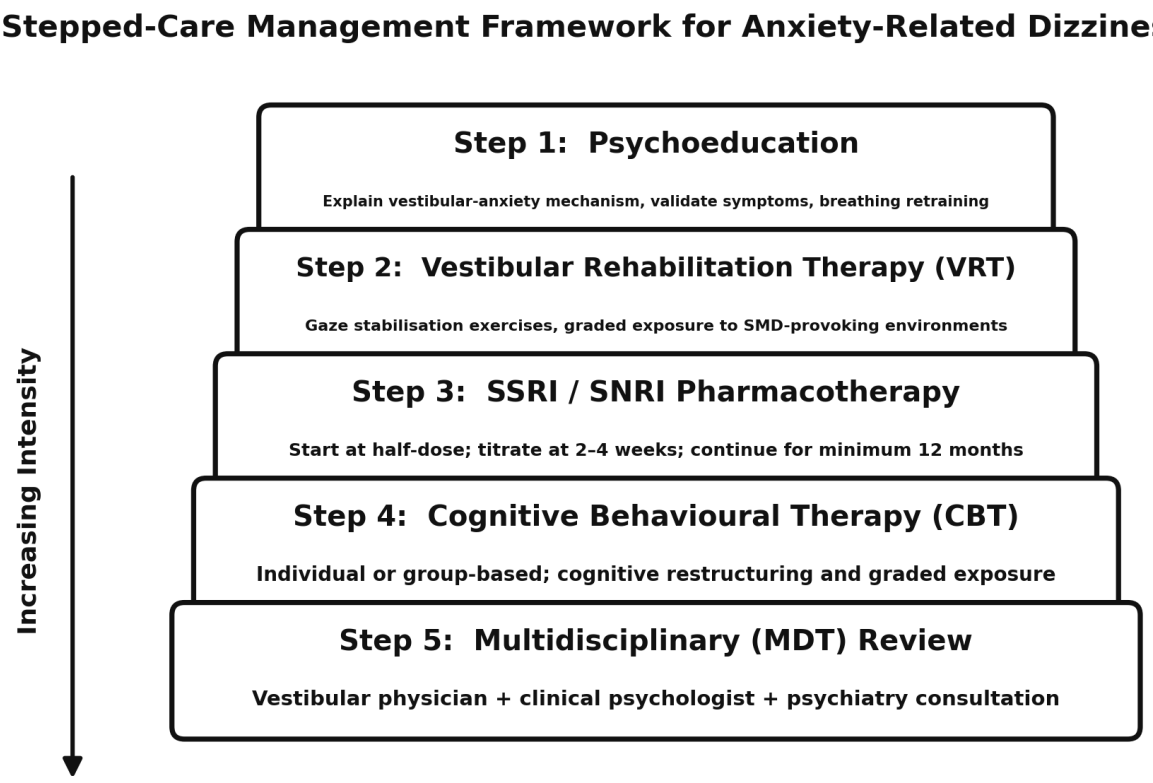

Figure 6. Stepped-care management framework for anxiety-related dizziness — from first-contact psychoeducation through to expert interdisciplinary care.
Source: Adapted from Staab [22] and Naber et al. [40].

□ **Key Point:** CBT and VRT are not alternative treatments for anxiety-related dizziness — they address different components of the same disorder. The combination consistently produces larger treatment effects than either alone. Vestibular physicians should develop relationships with vestibular physiotherapists and clinical psychologists who have experience at this interface.

IX. Prognosis, Recurrence, and Special Populations

The natural history of anxiety-related dizziness, untreated, is one of persistent or progressive functional impairment in the majority of patients — with remission rates in clinic populations significantly lower than those for primary panic disorder in psychiatric settings [43,45]. This discrepancy is explained by the additional vestibular sensitisation axis of the disorder, which is not addressed by psychiatric treatment alone and which perpetuates dizziness symptoms even when anxiety cognitions resolve [22,43].

Predictors of outcome

Favourable prognostic factors include: shorter symptom duration before treatment; absence of significant avoidance behaviour; patient motivation for psychological treatment; absence of major depression; and engagement with both pharmacological and non-pharmacological components of management [43,45]. Unfavourable predictors include: symptom duration greater than two years; prominent agoraphobic avoidance; comorbid depression; prior unsuccessful treatment with vestibular suppressants; health anxiety or somatisation disorder; and active compensation claims related to dizziness disability [31,43,45].

Among patients treated with combined SSRI, VRT, and CBT, approximately 60–75% report clinically significant improvement (at least 40% reduction in DHI or PDSS) at 12 months, with around 40–50% reaching remission [35,37,39]. These outcomes compare favourably with purely psychiatric treatment of panic disorder (60–70% response to SSRI alone) but require consistent multi-modal treatment to be achieved [35,37]. Recurrence after treatment cessation is common — estimated at 30–50% over 3 years — particularly in patients with residual avoidance or underlying GAD [19,43,52].

Elderly patients

Anxiety-related dizziness in older adults (age over 65) is systematically underdiagnosed because the anxiety component is frequently attributed to age-related dizziness, underlying cardiac or cerebellar disease, or polypharmacy — all of which may co-exist [24]. Late-life anxiety is associated with more prominent somatic symptoms and less overt psychological complaint than in younger patients, making the clinical detection of anxiety more difficult [24]. Specific considerations in the elderly: benzodiazepine avoidance is even more critical given the associated falls risk; starting SSRI doses should be lower and titration slower; and VRT programmes should prioritise balance function alongside exposure [24,32]. Falls risk assessment is mandatory before any vestibular exposure programme in older anxious patients [24,25].

Women and hormonal influences

Anxiety disorders are approximately twice as prevalent in women as men, and dizziness comorbidity with anxiety is correspondingly female-predominant [16]. Hormonal milestones — perimenopause, menstrual cycle variation, and postpartum period — are associated with heightened anxiety and increased sensitivity to vestibular stimuli [16,33]. Perimenopause is associated with new-onset anxiety disorders in women without prior psychiatric history, presenting with palpitations, dizziness, and mood instability that frequently reaches the vestibular physician before the gynaecologist or psychiatrist [33]. A routine hormonal history is warranted in women presenting with new anxiety-related dizziness in the peri- and postmenopausal window [33].

PPPD evolution and the anxiety-maintenance cycle

A clinically important and underappreciated progression pathway is from primary anxiety with dizziness to established PPPD. In this trajectory, a panic disorder patient develops chronic vestibular sensitisation, avoidance behaviour, and progressive SMD that outlasts the acute anxiety disorder and becomes a self-sustaining functional dizziness syndrome meeting PPPD criteria [10,20,22]. By the time PPPD is established, the original anxiety disorder may be in partial remission while the functional dizziness has become the dominant complaint — yet SSRI and CBT remain the most effective treatments precisely because the central sensitisation pathway is serotonergically mediated [10,36,40].

□ **Clinical Insight:** Anxiety-related dizziness should be thought of as a dynamic condition that evolves over time. Early identification and treatment prevents the hardening of avoidance behaviour into established PPPD — a transition that significantly worsens the prognosis and lengthens the treatment course.

X. Guidelines, Controversies, and Future Directions

Current guidelines

There is no single dedicated guideline for anxiety-related dizziness as a combined entity. Management draws from: the Barany Society PPPD consensus document (2017) for the functional dizziness component; NICE guidelines CG113 (Generalised Anxiety Disorder and Panic Disorder, 2011, updated 2019) for the psychiatric component; and the AAO-HNS vestibular rehabilitation evidence review for the rehabilitation component [10,38]. This fragmentation of guidance across disciplines reflects the historical siloing of otology and psychiatry — precisely the gap that the vestibular physician is positioned to bridge [1,5]. Several European centres have implemented joint vestibular-liaison psychiatry clinics with measurable improvements in time-to-treatment and outcome [2,41].

Controversies

Several active controversies in this field deserve explicit acknowledgement by the practising vestibular physician:

- **Terminology proliferation —** At least six diagnostic labels (phobic postural vertigo, chronic subjective dizziness, space and motion phobia, psychophysiological dizziness, visual vertigo, PPPD) have been applied to overlapping presentations. PPPD is the current consensus label but does not fully capture the primary anxiety phenotype; anxiety-related dizziness as a term is not formally codified. This creates diagnostic confusion, insurance-coding problems, and hinders research synthesis [20,22].
- **Is hyperventilation syndrome underdiagnosed? —** The Nijmegen Questionnaire has reasonable sensitivity and high specificity, yet formal hyperventilation testing is rarely performed in vestibular clinics. Undiagnosed HVS may account for a substantial proportion of patients labelled as PPPD or unexplained dizziness; routine hyperventilation testing in all persistent-dizziness cases may improve diagnostic yield [13].
- **Organic versus functional —** The binary organic/functional distinction is unhelpful and increasingly unsupported by neuroimaging evidence. Functional dizziness with anxiety has measurable neurobiological substrates — reduced hippocampal volume, altered PIVC connectivity, amygdala hyperreactivity — that demonstrate clear physical correlates of the disorder [26,27]. Vestibular physicians who communicate this framework to patients improve treatment engagement and outcomes [40,42,53].

Future directions

Several emerging developments are likely to reshape the management of anxiety-related dizziness in the coming decade. Transcranial magnetic stimulation (TMS) and transcranial direct current stimulation (tDCS) targeting the PIVC and prefrontal cortex have entered early trials for PPPD and anxiety-related dizziness, with preliminary evidence for symptom reduction — a potential option for pharmacotherapy-refractory cases [27]. Virtual reality (VR)-based vestibular exposure therapy, combining the graded motion exposure of VRT with the cognitive modelling of CBT in a controlled virtual environment, is in Phase II trial for PPPD and panic disorder with agoraphobia, with preliminary results suggesting advantages over standard graded exposure in terms of adherence and dropout rate [40].

Biomarker development is an active area: salivary cortisol diurnal patterns, heart rate variability indices, and fMRI connectivity signatures of the amygdala-vestibular network are candidate markers for treatment response stratification — potentially identifying at baseline which patients require combined VRT-CBT versus SSRI monotherapy [26,27]. Digital CBT platforms — app-based programmes with clinical oversight — have been validated for primary panic disorder and are now being adapted for vestibular contexts, offering scalable access in health systems with limited clinical psychology capacity [40,51].

Table 5. Summary of key guidelines and evidence levels for treatment modalities.

Treatment	Evidence level / guideline	Recommendation
SSRI (sertraline, escitalopram, paroxetine)	Level B (multiple open RCTs); NICE CG113 [35,36,52]	First-line; start low, titrate slowly
SNRI (venlafaxine, duloxetine)	Level B; NICE CG113 [36,52]	Alternative first-line or SSRI failure
CBT (individual or group)	Level A (RCTs); NICE CG113 [37,39,51]	First-line non-pharmacological

Vestibular rehabilitation therapy	Level B (RCTs for PPPD) [38,50]	First-line alongside pharmacotherapy
Breathing retraining (HVS)	Level B; consensus expert opinion [13]	First-line for HVS phenotype
Benzodiazepines (short-term)	Level C; avoid chronic use [52]	Short-term bridge only; taper plan required

□ **Clinical Pearl:** The future of anxiety-related dizziness management is integration — not the sequential referral from vestibular to psychiatry, but a co-designed programme that addresses vestibular sensitisation, anxiety cognitions, and avoidance behaviour simultaneously, in the same clinical environment, with a unified therapeutic narrative.

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