

Anxiety-Related Dizziness — Cheat Sheet for Vestibular Physicians

A positive diagnosis, not one of exclusion — but exclude structural disease first. Treat the vestibular axis and the anxiety axis together.

► Why anxiety-related dizziness matters

Anxiety and vestibular symptoms are bidirectionally linked. Panic disorder is roughly two to three times more common in patients with vestibular dysfunction, and up to 20–30% of patients attending a dizziness clinic carry a clinically significant anxiety disorder. The relationship is reciprocal: vestibular dysfunction triggers anxiety, and anxiety amplifies and perpetuates dizziness through the parabrachial–amygdala network. Recognising and treating the anxiety axis turns a chronic, refractory, over-investigated presentation into a treatable one — and is where a vestibular physician adds the most value.

► When to suspect it

- Chronic dizziness disproportionate to vestibular findings, worse in visually complex, crowded or moving environments.
- Situational or episodic dizziness with autonomic features — palpitations, derealisation, paraesthesiae, chest tightness.
- Dizziness that persists or worsens after an organic vestibular insult has resolved or compensated.
- Prominent avoidance, anticipatory anxiety, or catastrophic interpretation of normal motion cues.
- Voluntary hyperventilation reproduces the patient's everyday symptoms.

► Mechanism — the vestibular-anxiety circuit

Vestibular afferents reach the parabrachial nucleus, which projects to the amygdala, locus coeruleus and hypothalamus — the same network mediating fear and autonomic arousal. Three non-mutually-exclusive models explain the overlap and should guide which lever you pull first.

Model	Primary driver	Clinical signature
Psychosomatic	Anxiety/panic primary → hyperventilation, hypocapnic cerebral vasoconstriction	Dizziness is a secondary symptom; panic precedes and drives the dizziness
Somatopsychic	Vestibular dysfunction primary → fear, avoidance, conditioning	Anxiety follows a discrete vestibular event; postural instability is measurable
Network alarm theory	Sensitised amygdala-PBN-locus coeruleus circuit	Normal vestibular signals misread as threat; conditioned panic and avoidance

Pearl — The somatopsychic pathway dominates when anxiety clearly followed a vestibular insult — these patients respond best to VRT plus graded exposure rather than drugs alone.

► Clinical phenotypes

Condition	Key features	Vestibular link
Panic disorder	Discrete attacks, palpitations, derealisation, fear of dying	Dizziness in 60–70% of attacks; agoraphobic avoidance of triggers
Generalised anxiety (GAD)	Persistent worry, muscle tension, poor sleep	Chronic unsteadiness and visual dependence; symptom amplification
Hyperventilation syndrome	Overbreathing, paraesthesiae, carpopedal spasm, tetany	Hypocapnic dizziness; reproduced and relieved by provocation testing

Pearl — PPPD frequently coexists — treat the overlapping maintaining mechanisms (sensitisation, catastrophic cognition, avoidance) rather than forcing a single diagnostic label.

► Work-up — and what to exclude first

Step	Tool	Purpose
History	Trigger profile, autonomic symptoms, avoidance, timeline	Distinguish primary from secondary (post-vestibular) anxiety
Screening	HADS, DHI, Nijmegen Questionnaire (≥23 = HVS)	Quantify burden; document a treatment baseline
Bedside	HINTS, Dix-Hallpike, supine roll, Romberg	Exclude peripheral and central organic disease
Function	vHIT, caloric, VEMP, posturography	Identify subclinical vestibular asymmetry driving sensitisation
Provocation	Voluntary hyperventilation test	Reproduce and demystify symptoms; therapeutic in itself

Pearl — A positive diagnosis needs DSM-5 anxiety criteria PLUS examination consistent with functional or sensitised processing — never the mere absence of organic findings.

- **Red flags** — New or progressive unilateral hearing loss, focal neurology (INO, ataxia, diplopia, dysarthria), prominent spontaneous or direction-changing nystagmus, acute severe vertigo at rest, an orthostatic component, or onset after head or neck trauma → image and investigate.

Anxiety-related dizziness is a positive diagnosis, never one of exclusion, but structural and posterior-fossa disease must be actively ruled out before it is confirmed.

► **Management — treat both axes together**

Step	Intervention	Practice points
1 · Psychoeducation	Explain the vestibular-anxiety mechanism; breathing retraining	Frame dizziness as real but benign; warn it may worsen weeks 1-2 on an SSRI
2 · VRT	Gaze stabilisation; graded exposure to SMD-provoking settings	Directly targets sensitisation and avoidance; commence early
3 · Pharmacotherapy	SSRI at HALF dose, titrate at 2-4 wk; SNRI if inadequate	Sertraline 25 mg or Escitalopram 5 mg start; continue 12 months minimum
4 · CBT	Cognitive restructuring and graded exposure	First-line for catastrophic cognition; individual or group
5 · MDT review	Vestibular physician + psychologist + psychiatry	Refractory/severe panic; benzodiazepine only as a ≤4-week bridge, never chronic

Pearl — Start SSRIs at half dose and warn about the first-fortnight dizziness — unwarned patients stop early, convinced the tablet made their dizziness worse.

► **Pharmacotherapy quick-reference**

Agent	Start → target	Notes for the anxious vestibular patient
Sertraline (SSRI)	25 mg/d → 50-200 mg/d	First-line; start low to blunt the early dizziness spike
Escitalopram (SSRI)	5 mg/d → 10-20 mg/d	Well tolerated first-line alternative; few interactions
Venlafaxine (SNRI)	37.5 mg/d → 75-150 mg/d	Second-line if SSRI inadequate; monitor blood pressure
Buspirone / pregabalin	Buspirone 5 mg tds	Augmentation for residual anxiety; non-sedating
Clonazepam (BZD)	0.25-0.5 mg	Bridge only, ≤4 weeks, taper from day 1; never chronic

Pearl — SSRIs are the backbone; benzodiazepines reinforce avoidance and interfere with vestibular compensation — reserve them for a short, planned bridge only.

► **Prognosis and follow-up**

- Combined SSRI + VRT + CBT achieves ~60-75% clinically significant improvement at 12 months.
- Remission lags pure panic disorder — the vestibular sensitisation axis must be treated, not just the anxiety.
- Recurrence ~30-50% over 3 years, higher with residual avoidance or untreated GAD.
- Re-image or reassess if the picture changes — diagnostic labels are revisable.

Key references — Staab. *Curr Opin Neurol* 2013 · Balaban & Thayer. *J Anxiety Disord* 2001 · Gorman et al. *Am J Psychiatry* 2000 · Asmundson, Larsen & Stein 1998 · Bittar & Lins. *Braz J Otorhinolaryngol* 2015.