

Phobic Postural Vertigo as a Primary Functional–Anxiety Syndrome

Clinical phenomenology, fear mechanisms and psychiatric treatment without peripheral vestibular pathology

AI author: ChatGPT (OpenAI; GPT-6)

Human prompter and clinical contributor: Prof. Dr. med. Jürgen C. Aschoff

Author affiliation and current position: Jürgen C. Aschoff, MD, aged 88, is a retired Professor of Neurology (formerly University of Ulm, Germany) and currently practices as a neurologist and psychiatrist in a private practice in Ulm, Germany. He is no longer affiliated with the university.

Correspondence: fabri.antiquariat@gmx.de

AI contribution and human oversight

This narrative manuscript was generated and revised by ChatGPT through iterative prompts from Jürgen C. Aschoff, who specified its clinical scope and requested exclusive attention to primary phobic dizziness. Clinical judgments, medication decisions and final approval remain the responsibility of the human contributor. The article presents no newly collected patient data.

Abstract

Background: Classical phobic postural vertigo (PPV), or phobic swaying dizziness, describes subjectively distressing unsteadiness—often episodic and situation-dependent—in patients without a demonstrable active peripheral vestibular lesion. It belongs at the interface of neurology, psychiatry and psychosomatic medicine, but should not be conflated with positional vertigo or with every phenotype encompassed by modern persistent postural-perceptual dizziness (PPPD).

Objective: To define a deliberately restricted clinical phenotype of primary phobic dizziness and review the roles of threat appraisal, bodily hypervigilance, avoidance, psychoeducation, psychotherapy and cautious treatment of associated psychiatric symptoms.

Methods: Focused narrative discussion informed by foundational descriptions of phobic postural vertigo and selected treatment studies. No systematic review or quantitative meta-analysis was undertaken.

Findings: Typical features include subjective sway or insecurity during stance and gait, often normal routine neurological and balance findings, context-specific exacerbation, anticipatory anxiety and secondary avoidance. Clear diagnostic explanation, graduated exposure and cognitive-behavioural strategies are central. Evidence for a fixed drug regimen specific to isolated PPV is limited.

Conclusion: In the strictly defined primary phobic phenotype, the therapeutic target is the cycle linking bodily attention, fear, protective behaviour and persistent uncertainty, not a presumed labyrinthine deficit.

Keywords: phobic postural vertigo; phobic swaying dizziness; functional dizziness; anxiety; anticipatory fear; cognitive behavioural therapy; psychoeducation

1. Clinical scope and terminology

The term “phobic postural vertigo” was developed in the work of Brandt and Dieterich and subsequently discussed as a recognizable syndrome of non-spinning subjective disequilibrium, heightened self-observation

and situational fear [1–3]. This paper restricts its topic to the primary phobic presentation. It does not discuss mechanisms or therapies of peripheral labyrinthine diseases, canalolithiasis, otolith dysfunction or positional manoeuvres.

PPV and PPPD should not be treated as synonyms. PPPD, as defined by the Bárány Society, is a broader modern diagnosis that requires symptoms on most days for at least three months and can arise after several different precipitating conditions [4]. The older PPV construct notably includes episodic phobic attacks and characteristic behavioural patterns. Here “PPV” retains that narrower historical and clinical meaning; the findings below should not automatically be generalized to the complete PPPD population.

2. Clinical presentation of primary phobic dizziness

2.1 Subjective bodily experience

Patients describe unsteadiness, sway, ground insecurity, a feeling of walking on cotton or an expectation of losing balance. The complaint is usually not an illusion of environmental rotation. Brief attacks may interrupt otherwise relatively normal days; other patients report a persistent vulnerability with episodic intensification. Symptoms are real and distressing, irrespective of whether routine examinations reveal measurable instability [1–3].

2.2 Discrepancy between symptom burden and examination

Ordinary gait, stance and neurological assessment may be normal or show nonspecific variations that cannot explain the subjective distress. This discrepancy is an important clinical clue, not proof that symptoms are imaginary. Neither a normal Romberg test nor normal posturography alone establishes a psychiatric cause; diagnosis requires a coherent positive clinical history and assessment for relevant alternative causes.

2.3 Situational specificity

Supermarkets, crowded rooms, queues, broad open spaces, bridges and socially exposed settings are frequent contexts in which subjective insecurity becomes especially intrusive. Patients may monitor every step, seek an anchor or leave the situation. In some individuals, walking briskly, cycling, being absorbed in a task or moving automatically feels easier than standing still and monitoring posture. These patterns are characteristic observations, not obligatory criteria [2,3].

2.4 Affective features

The emotion may be overt panic, apprehension, shame or an almost wordless sense that balance is about to fail. Anxiety need not precede the first episode; it may emerge as an interpretation and consequence of recurring bodily sensations. Conversely, phobic anticipation can later trigger or amplify symptoms. The variable temporal relationship between fear and dizziness is essential for avoiding a simplistic “all in the mind” explanation.

3. The anxiety–attention–avoidance cycle

The central clinical model proposed here is behavioural and cognitive rather than an assertion of proven cellular pathophysiology. An unfamiliar sensation of sway prompts selective attention to posture. Ordinary fluctuations of bodily sensation become threatening; fear increases muscular guarding and conscious correction; uncertainty rises; the patient escapes or avoids the situation. Short-term relief then reinforces the avoidance. Over time, anticipatory anxiety may generate symptoms before the feared setting is reached.

This model accommodates patients with marked anxiety as well as those who initially deny feeling frightened. It also distinguishes the subjective experience of being at risk of falling from an objectively increased tendency to

fall. The interpretation of symptom persistence as a learned threat-and-safety loop is a clinical framework, not an established biomarker of PPV.

4. Psychiatric and psychosocial dimensions

Anxiety disorders, depressive symptoms, perfectionistic or control-oriented coping, life stress and heightened health anxiety may accompany phobic dizziness. None is required in every case; personality attributions should never substitute for a patient-specific psychiatric interview. In clinical practice it is useful to ask exactly what the patient fears: collapse, public embarrassment, an undetected serious disorder, helplessness or inability to leave a situation. Each expectation creates a different therapeutic entry point.

The neurological consultation is particularly influential. Repeated inconclusive investigations without a clear explanation can unintentionally preserve diagnostic uncertainty. The physician can validate the bodily experience while explaining how heightened monitoring and protective responses perpetuate symptoms in the absence of evidence for a progressive neurological disorder.

5. Positive diagnosis and clinical boundaries

A careful history seeks non-spinning sway or insecurity, its relationship to stance, walking and situations, the presence of anticipatory fear, fluctuations with distraction and the emergence of avoidance. Examination includes focused neurological and balance assessment as clinically indicated. The purpose is not to label all unexplained dizziness “psychogenic” but to recognize a reproducible symptom pattern alongside the absence of findings demanding another active diagnosis.

Primary panic disorder, agoraphobia, generalized anxiety disorder, depressive illness, medication adverse effects and systemic causes of lightheadedness may require consideration. Phobic dizziness and an independent anxiety disorder can coexist. Persistent abnormal focal neurological findings, true syncope, progressive gait deterioration or other red flags warrant appropriate investigation rather than a presumptive PPV label.

6. Immediate psychoeducation: a neurological and psychiatric intervention

The first intervention is an intelligible, non-dismissive diagnosis. The clinician can explain that the symptom is genuine; that the examination does not indicate the feared catastrophic loss of balance; that normal corrective body movements become more noticeable under scrutiny; and that repeated checking and avoidance can maintain the problem. A simple drawing of “sensation → alarming interpretation → vigilance and tension → greater sensation → avoidance” often makes the mechanism understandable.

One or several focused consultations may be useful, tailored to the patient’s level of fear and understanding. There is no validated, disease-specific requirement for precisely one to three sessions or a fixed number of minutes. The desired immediate outcome is not forced reassurance but a plausible working model the patient can test in everyday life.

7. Psychological treatment and exposure

7.1 Cognitive behavioural therapy

Cognitive behavioural therapy (CBT) directly addresses catastrophic predictions, symptom checking, safety behaviours and progressive withdrawal from ordinary activities. Collaborative exposure involves returning stepwise to individually feared situations and comparing expected catastrophe with actual experience. The goal

is not to prove that dizziness cannot be felt; it is to reduce the feared meaning attached to that sensation and restore behaviour despite uncertainty.

A controlled study reported additional short-term benefit from CBT in a population diagnosed with PPV, but the one-year follow-up did not demonstrate persistence of the initial treatment advantage [5,6]. These findings argue against promising uniformly durable recovery from a standard-length CBT course. Course length should be individualized; 10–20 sessions can be a practical clinical framework rather than an established PPV-specific dosage.

7.2 Psychodynamic and other approaches

When anxiety is intertwined with longstanding conflicts, chronic interpersonal stress or repeated failed symptom-control efforts, psychodynamic exploration may have a role according to patient preference and psychiatric assessment. Evidence for a specific psychodynamic session count in isolated phobic dizziness is insufficient. Acceptance-based strategies may complement CBT by discouraging fruitless attempts to eliminate every bodily fluctuation; they should be presented as adjunctive clinical options rather than proven stand-alone cures.

8. Psychopharmacology: treating anxiety rather than the labyrinth

Medication is not mandatory for isolated PPV. When clinically meaningful comorbid anxiety or depressive symptoms persist, an SSRI or SNRI may be considered using the indication, dosing, interaction review and follow-up appropriate to that psychiatric condition. Small uncontrolled studies in related chronic subjective dizziness populations cannot establish a definitive pharmacological efficacy rate for narrowly defined primary PPV. A 2023 Cochrane review found no eligible placebo-controlled randomized trials of pharmacotherapy for PPPD under its diagnostic criteria [7]; that finding should not be presented as direct evidence for or against medication in the older, narrower PPV phenotype.

Claims that one particular antidepressant is a proven first-line disease-specific treatment, that 60–75% of isolated PPV patients respond, or that every patient needs 9–12 months of treatment are not adequately justified by this evidence. Initiation and titration must be individualized, especially in older adults and those taking other medications. Drug-related dizziness, sedation, withdrawal symptoms and falls must be considered.

Routine benzodiazepine prescribing is unattractive as a chronic strategy because of dependence, cognitive impairment and falls risk, not because all use is categorically contraindicated. Sedating symptomatic “anti-vertigo” medication is not a treatment for the phobic fear–avoidance mechanism. The central priorities remain explanatory care, restoration of confidence and reduction of maladaptive avoidance.

9. A focused clinical management sequence

- At presentation: elicit the precise subjective experience, feared consequence and situational triggers; conduct an appropriate examination and assess psychiatric comorbidity.
- At diagnosis: explicitly validate the dizziness, explain the anxiety–attention–avoidance cycle and agree on a testable formulation.
- Early follow-up: identify safety behaviours, review unsuccessful avoidance strategies and encourage gradual return to feared everyday activities.
- Persistent disability: offer structured CBT with individually paced exposure and reassess coexisting anxiety, depression or psychosocial stress.
- Medication review: consider pharmacotherapy only on an individual psychiatric basis, with explicit discussion of uncertain PPV-specific efficacy and potential adverse effects.

- Longer-term review: assess function, participation and avoidance as well as symptom severity; revise the diagnosis if new findings emerge.

10. Discussion

A narrowly drawn account of phobic postural vertigo has the advantage of clinical clarity. It directs attention to a recognizably phobic symptom mechanism rather than placing every type of non-spinning dizziness into one therapeutic category. At the same time, a strictly “pure” phobic phenotype is a conceptual and clinical selection, not a biologically validated separate disease entity. Symptom histories can be mixed, and the literature does not always segregate isolated PPV from broader functional dizziness populations. Those limitations must accompany any treatment claims.

The primary research need is for studies that specify whether participants have a positive, primary phobic phenotype, quantify avoidance and anxiety independently from dizziness, and report sustained functional outcomes. The clinician should resist both extremes: reflexively declaring symptoms structural despite repeatedly uninformative examination, and assigning a psychiatric label without a positive, documented formulation.

11. Conclusion

Classical phobic postural vertigo is best understood in this review as subjective swaying dizziness organized by threat appraisal, hypervigilance and avoidance. Its management belongs within a coordinated neurological–psychiatric conversation. Positive diagnostic explanation, therapeutic alliance and individually adapted psychological exposure form the core; pharmacotherapy is reserved for relevant psychiatric indications rather than a presumed inner-ear defect. This focused approach avoids conflation with distinct vestibular syndromes and preserves the clinically useful original concept of phobic swaying dizziness.

Limitations and disclosure

This AI-generated article is a focused narrative review, not a systematic review or formal clinical guideline. References include foundational PPV literature and contextual evidence; some treatment studies enrolled broader or mixed phenotypes and do not prove efficacy in a rigorously isolated primary phobic group. No patient-level original research, prospective protocol or new efficacy data are claimed.

References

1. Brandt T. Phobic postural vertigo. *Neurology*. 1996;46(6):1515–1519. doi:10.1212/WNL.46.6.1515.
2. Brandt T, Kapfhammer HP, Dieterich M. „Phobischer Schwankschwindel“. Eine weitere Differenzierung psychogener Schwindelzustände erscheint erforderlich. *Nervenarzt*. 1997;68(10):848–849. doi:10.1007/s001150050205.
3. Dieterich M. Erkennen Sie den phobischen Schwankschwindel! *MMW Fortschr Med*. 2000;142(3):26–29. PMID:10726456.
4. Staab JP, Eckhardt-Henn A, Horii A, et al. Diagnostic criteria for persistent postural-perceptual dizziness (PPPD): consensus document of the Bárány Society. *J Vestib Res*. 2017;27(4):191–208. doi:10.3233/VES-170622.
5. Holmberg J, Karlberg M, Harlacher U, Rivano-Fischer M, Magnusson M. Treatment of phobic postural vertigo: a controlled study of cognitive-behavioral therapy and self-controlled desensitization. *J Neurol*. 2006;253(4):500–506. doi:10.1007/s00415-005-0050-6.

6. Holmberg J, Karlberg M, Harlacher U, Magnusson M. One-year follow-up of cognitive behavioral therapy for phobic postural vertigo. *J Neurol*. 2007;254(9):1189–1192. doi:10.1007/s00415-007-0499-6.

7. Webster KE, Harrington-Benton NA, Judd O, et al. Pharmacological interventions for persistent postural-perceptual dizziness (PPPD). *Cochrane Database Syst Rev*. 2023;(3):CD015188. doi:10.1002/14651858.CD015188.pub2.

Correspondence: Prof. Dr. med. Jürgen C. Aschoff (retired) · Private neurological and psychiatric practice, Ulm, Germany · fabri.antiquariat@gmx.de