Persistent Postural-Perceptual Dizziness (PPPD):

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

Vestibular Medicine for Vestibular Physicians

Functional and Multisensory Vestibular Disorders — Module 4.2

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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 multisensory 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.

Table of Contents

I. Introduction and Epidemiology

Persistent postural-perceptual dizziness (PPPD) is the most common cause of chronic vestibular symptoms in adults of working age and the unifying diagnosis that finally consolidated a century of overlapping descriptions of chronic non-vertiginous dizziness [1,2]. It is defined by the Committee for the Classification of Vestibular Disorders of the Bárány Society as a chronic functional vestibular disorder characterised by persistent dizziness, unsteadiness or non-spinning vertigo, present on most days for three months or more, and exacerbated by upright posture, active or passive self-motion, and exposure to moving or complex visual stimuli [1]. The disorder was formally codified in 2017 and incorporated into the International Classification of Diseases, 11th revision, under code AB32.0, giving clinicians a single, internationally agreed label and case definition [1,42].

The conceptual lineage matters because it explains the disorder's clinical texture. Brandt and Dieterich described phobic postural vertigo in 1986 in patients with subjective unsteadiness, anxiety and a normal examination [3]. Bronstein characterised the visual vertigo syndrome in 1995, drawing attention to symptoms provoked by full-field visual motion such as supermarket aisles and traffic [4]. Staab and Ruckenstein then introduced chronic subjective dizziness in the 2000s, emphasising hypersensitivity to motion and a maladaptive postural control strategy independent of any persisting structural lesion [5,6]. PPPD subsumes these antecedents into one syndrome with explicit, operationalised criteria, while discarding the assumption that the symptoms are primarily psychiatric [1,8].

□ **Key Point:** PPPD is a positive clinical diagnosis defined by a characteristic symptom pattern — not a diagnosis of exclusion or a synonym for anxiety. It replaces and unifies phobic postural vertigo, visual vertigo and chronic subjective dizziness [1,8].

Epidemiologically, PPPD accounts for roughly 15–25 per cent of diagnoses in specialist dizziness clinics, and in several tertiary neuro-otology series it is the single most frequent diagnosis among patients with chronic symptoms [2,9,22,24]. A population-based analysis suggests that PPPD-type symptoms lie on a continuum within the general community rather than forming a discrete category, which is consistent with its emergence after a wide range of vestibular and non-vestibular precipitants [11]. Peak onset is in the fourth to sixth decades, and most series report a moderate female preponderance [2,9,24]. There is no confirmed geographic or ethnic predilection; reported prevalence tracks diagnostic awareness more than any biological gradient [2,21].

Table 1. Persistent postural-perceptual dizziness — epidemiology at a glance [2,9,11,22,24].

Parameter	Typical finding
Frequency in dizziness clinics	Approximately 15–25% of all diagnoses; often the most common chronic diagnosis
Typical age of onset	Fourth to sixth decade (mean in the 40s–50s)
Sex distribution	Moderate female preponderance (roughly 60–70% female in most series)
Most common precipitants	Peripheral vestibular disorders, vestibular migraine, panic/anxiety, concussion
Community pattern	Symptoms distributed on a continuum in the general population

The economic and personal burden is substantial. PPPD frequently coexists with anxiety, depression and other functional somatic syndromes, prolongs time away from work, and drives repeated, often unrewarding investigation when it is not recognised [2,9,43]. For the vestibular physician, confident early recognition is therefore the single highest-value intervention, short-circuiting the cycle of escalating tests and reinforcing the very threat appraisal that perpetuates the disorder [2,8].

Awareness remains the rate-limiting step. Despite formal codification, PPPD is still under-recognised in primary care and in parts of secondary care, and patients commonly accumulate years of symptoms and multiple normal investigations before the diagnosis is reached [2,43]. The delay is not benign: prolonged uncertainty entrenches avoidance, deconditioning and health anxiety, all of which worsen the prognosis and complicate later treatment [8,43]. For a vestibular service, embedding the diagnostic criteria into

routine assessment of every patient with chronic dizziness is therefore both a clinical and a system-level intervention [2,8].

II. Pathophysiology — Failed Recalibration and Visual Dependence

PPPD is a disorder of vestibular function, not structure. There is no destructive lesion; rather, the brain adopts and then fails to abandon a maladaptive mode of balance control [1,8,27]. The most widely accepted model proposes a stereotyped sequence: an acute precipitant provokes vertigo or unsteadiness, the brain shifts to a stiff, high-risk postural control strategy with heightened reliance on vision and proprioception, and in predisposed individuals this strategy persists long after the original insult has resolved or compensated [1,8,19].

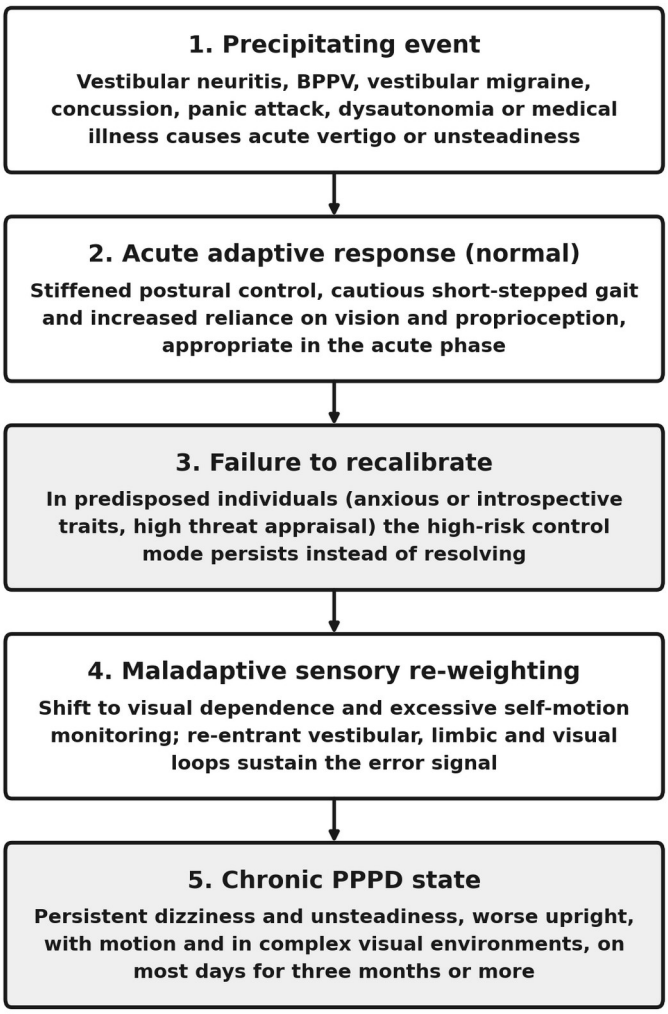

Figure 1. The proposed pathophysiological cascade of PPPD — from acute precipitant, through a normal protective adaptation, to failed recalibration and a self-sustaining chronic state.
Source: Adapted from Staab et al. [1] and Popkirov et al. [8].

The shift to visual dependence

A defining physiological signature of PPPD is increased visual dependence — an over-weighting of visual input in the multisensory estimate of self-motion and verticality [4,16]. Patients who later develop chronic

dizziness after vestibular neuritis show greater visual dependence in the acute phase than those who recover, suggesting that the trait both predisposes to and is amplified by the disorder [16,17]. Posturography demonstrates excessive reliance on visual cues and abnormal high-frequency postural sway, while gait analysis reveals a cautious, co-contracted, attention-demanding walking pattern reminiscent of walking on ice [18,19,23]. Crucially, these abnormalities improve with distraction and worsen with self-focused attention, a hallmark of functional rather than structural disease [23].

A complementary and increasingly influential framework casts PPPD in terms of predictive processing. The brain continuously predicts the sensory consequences of self-motion and tests those predictions against incoming vestibular, visual and proprioceptive signals; in PPPD the system appears to assign excessive precision, or confidence, to visual and interoceptive predictions while down-weighting vestibular input, so that ordinary sensory mismatches are read as salient errors that demand postural correction [46,47]. This account accommodates the visual-vestibular mismatch that patients describe in full-field motion and explains why the disorder behaves as a functional neuro-otological condition rather than a fixed structural deficit [47,48]. Neurophysiological studies that combine clinical assessment with evoked and resting-state measures localise the abnormality to higher-order integrative processing rather than to the labyrinth or the primary vestibular pathways [27,41].

□ **Clinical Insight:** The paradox of PPPD gait — unsteadiness that improves when the patient is distracted (for example performing a cognitive task) and worsens under deliberate self-observation — is a useful bedside pointer toward a functional mechanism [19,23].

Posturographic and gait studies add quantitative substance to the model. On the sensory organisation test, patients perform disproportionately poorly in conditions that remove or destabilise reliable visual and proprioceptive cues, consistent with their over-reliance on those channels, while high-frequency, low-amplitude body sway and increased co-contraction of antigravity muscles reflect the stiff, effortful control strategy [18,19]. These measures normalise as patients recover, and the same studies show that the abnormalities are modulated by attention rather than fixed, reinforcing that the substrate is a control policy the brain can in principle relearn [19,23,41].

Cortical networks and the threat overlay

Functional neuroimaging supports a network rather than a lesion. Resting-state and task-based studies show reduced connectivity within and between central vestibular cortical regions — particularly the parieto-insular vestibular cortex — alongside altered engagement of visual motion areas and anxiety-related limbic structures including the amygdala and anterior insula [12,13,14,15]. The pattern is interpreted as a reweighting of multisensory integration toward visual and interoceptive signals, coupled with a heightened threat appraisal that tags ordinary self-motion as dangerous [13,46]. This is the neurobiological substrate of the clinical observation that PPPD sits at the intersection of vestibular and affective processing without being reducible to either [8,13].

Personality and affective traits are genuine pathophysiological contributors, not incidental comorbidity. Anxious, introverted and introspective traits are over-represented and predict both the development and the persistence of symptoms [7,20,28]. High pre-morbid trait anxiety and a tendency to catastrophic interpretation of bodily sensations bias the system toward sustained hypervigilance, which prevents the normal down-regulation of the protective control mode [7,20,29]. The result is a self-reinforcing loop in which symptoms generate anxiety, anxiety heightens vestibular vigilance, and vigilance sustains symptoms [8,38].

□ **Clinical Pearl:** Think of PPPD as a software fault, not a hardware fault. The balance hardware works; the control software stays locked in a high-alert, vision-dependent mode that no longer matches the patient's actual risk [8].

III. Clinical Features — The Postural, Motion and Visual Triad

The clinical picture is remarkably consistent and is the basis of the diagnostic criteria [1,8]. Patients describe a near-constant sense of dizziness, unsteadiness, swaying or rocking — frequently likened to being on a boat — rather than discrete rotational vertigo [1,26]. Symptoms are present on most days, often waxing and waning in intensity through the day, and characteristically building from relative ease on waking to greater unsteadiness by afternoon or evening as sensory and cognitive demands accumulate [26,40].

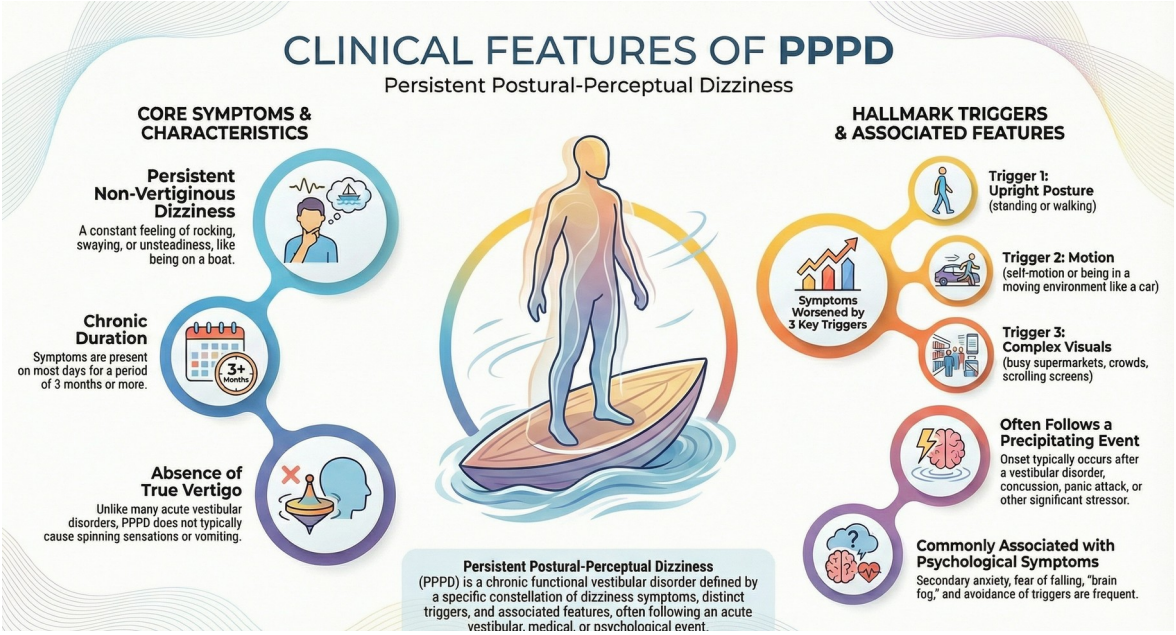

Figure 2. The clinical signature of PPPD — core non-vertiginous symptoms, the three exacerbating factors, and the associated features that complete the picture.

Source: Australian Dizziness Clinics, synthesising the Bárány Society criteria [1] and Popkirov et al. [8].

Three exacerbating factors define the syndrome and should be sought explicitly in every history [1,4,8]. First, upright posture: symptoms worsen on standing or walking and ease on lying down. Second, active or passive motion: the patient's own movements and passive transport such as car or escalator travel aggravate symptoms regardless of direction. Third, exposure to moving or complex visual stimuli: busy supermarkets, scrolling screens, patterned floors and traffic provoke a characteristic spike in dizziness — the visual dependence described in Section II made clinically visible [4,8].

□ **Clinical Insight:** Two screening questions capture the syndrome with high yield: "Do busy or moving visual environments such as supermarkets or scrolling screens make you worse?" and "Are you better lying down than standing or walking?" Consistent yes answers should raise PPPD strongly [8,26].

Onset typically follows an identifiable precipitant — most often an acute or episodic vestibular disorder, but also vestibular migraine, concussion, dysautonomia, a medical illness, or a panic attack [1,10,39]. Symptoms may begin intermittently and consolidate into a daily pattern as the precipitant resolves [1]. Secondary anxiety, depression, fear of falling, avoidance behaviour and a subjective "brain fog" of impaired concentration are common and amplify disability [8,29,43]. By definition the neurological and bedside vestibular examination is normal or shows only a well-compensated old deficit that does not explain the ongoing symptoms [1,8].

The internal structure of the symptom set is clinically useful. Validated instruments such as the Niigata PPPD Questionnaire separate the contributions of the three provoking factors — upright posture, motion, and visual stimulation — and many patients show a dominant subscale, for example a strongly visual-dominant phenotype that drives supermarket and screen intolerance, or a motion-dominant phenotype that limits travel [25]. Recognising the dominant provoking factor helps tailor rehabilitation, since the desensitisation programme can be weighted toward the patient's most disabling trigger [25,32]. The intensity of symptoms is characteristically variable from hour to hour and day to day, and patients

frequently report that fatigue, poor sleep and emotional stress amplify the dizziness, a pattern that fits the attentional and affective modulation seen experimentally [23,26,29].

Equally important is what PPPD is not. It does not cause true spinning vertigo attacks, spontaneous nystagmus, or prominent autonomic features such as vomiting; when these are present, an active vestibular disorder such as BPPV, Ménière's disease or vestibular migraine should be sought, recognising that such disorders may coexist with and continue to trigger PPPD [1,8].

□ **Important:** New spontaneous or positional nystagmus, fresh focal neurological signs, acute hearing loss, or a first severe headache are not features of PPPD. Their presence demands a search for active vestibular or central pathology before the symptoms are attributed to PPPD [1,8].

IV. Diagnosis — Bárány Society Criteria and the Bedside

PPPD is diagnosed clinically against the five Bárány Society criteria, all of which must be satisfied [1]. The criteria operationalise the clinical features described above and, importantly, do not require the demonstration of a psychiatric disorder or the complete absence of any other vestibular condition [1,8]. The diagnostic act is therefore a positive pattern recognition supported by a targeted exclusion of mimics, not an open-ended hunt for a structural lesion [2,8].

Table 2. Bárány Society diagnostic criteria for PPPD — all five (A–E) required [1].

Criterion	Requirement
A. Persistent symptoms	Dizziness, unsteadiness or non-spinning vertigo on most days for ≥ 3 months, lasting prolonged (hours) but often fluctuating
B. Provoking factors	Symptoms occur without specific provocation but are exacerbated by upright posture, active or passive motion, and moving or complex visual stimuli
C. Precipitant	Triggered by a condition causing vertigo, unsteadiness, dizziness or balance problems — acute, episodic or chronic vestibular, neurological, medical or psychological
D. Distress or impairment	Symptoms cause significant distress or functional impairment
E. Not better explained	Symptoms are not better accounted for by another active disease or disorder

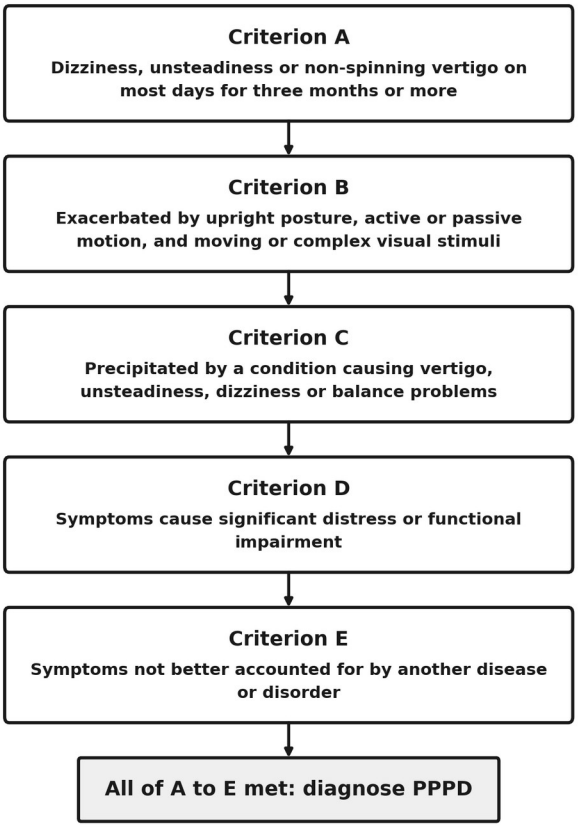

Figure 3. The five Bárány Society criteria for PPPD shown as a sequential checklist; the diagnosis requires that all of A through E are satisfied.
Source: Adapted from Staab et al. [1].

Applying the criteria at the bedside

In practice, criterion C is the one most often overlooked, because patients and previous clinicians may have forgotten or normalised the original precipitant [1,10]. A careful timeline that links a remote episode of vertigo, a concussion or a period of intense anxiety to the onset of daily symptoms frequently unlocks the diagnosis [10,39]. Criterion E does not demand a normal examination in the absolute sense; it demands that no active disorder better explains the picture, which permits PPPD to be diagnosed alongside a compensated vestibular neuritis or well-controlled migraine [1,8]. Structured instruments such as the Niigata PPPD Questionnaire can quantify symptom load and the relative contribution of the three provoking factors, aiding both diagnosis and the tracking of treatment response [25].

□ **Clinical Pearl:** When a patient says "every test has been normal but I am still dizzy every day," do not reach for another scan — reach for the PPPD criteria. Normal investigations in the right symptom pattern support, rather than undermine, the diagnosis [2,8].

V. Investigations and the Role of Excluding Structural Disease

There is no confirmatory test for PPPD; investigation serves two defined purposes — to exclude an active vestibular, neurological or medical disorder that would better explain the symptoms, and to characterise any compensated deficit that acted as the precipitant [1,8]. The work-up should be proportionate and

hypothesis-driven; over-investigation reinforces illness beliefs and is itself anti-therapeutic [2,8]. Figure 4 sets out a pragmatic bedside-to-diagnosis pathway.

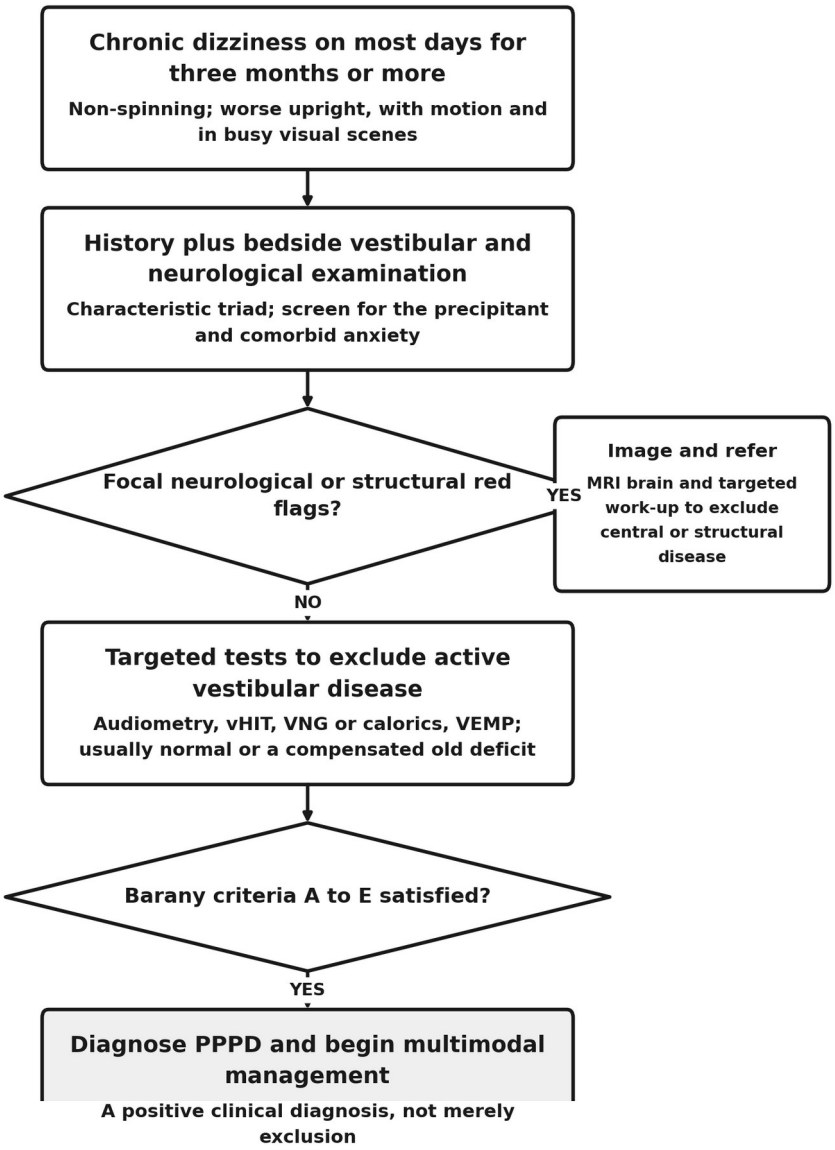

Figure 4. A pragmatic diagnostic pathway for the patient with chronic dizziness, moving from history and examination through red-flag screening and targeted vestibular testing to a positive PPPD diagnosis.
Source: Australian Dizziness Clinics, adapted from Staab et al. [1] and Dieterich and Staab [8].

A focused vestibular battery — audiometry, video head impulse testing, videonystagmography with caloric testing, and vestibular evoked myogenic potentials — is typically normal or shows only a stable, compensated unilateral deficit [1,8,18]. Posturography may demonstrate the characteristic visual dependence and abnormal sway pattern but is not required for diagnosis [18]. Where the history includes orthostatic lightheadedness or syncope, orthostatic vital signs or autonomic testing should be added; where vestibular migraine is plausible, its criteria should be applied in parallel [1,8]. Neuroimaging is reserved for cases with red flags or an atypical course rather than performed reflexively [8].

Table 3. Targeted investigations in suspected PPPD — purpose and indication [1,8,18].

Investigation	Purpose	When to order
Audiometry	Exclude Ménière's disease and other cochleovestibular pathology	Most patients, especially with any aural symptoms
vHIT, VNG and calorics	Detect or characterise a peripheral deficit (usually old and compensated)	Most patients, to confirm no active loss

VEMP	Assess otolith pathways; supports characterisation	Selected patients where otolith disease is considered
MRI brain	Exclude central or structural disease	Red flags, atypical course or focal signs only
Orthostatic and autonomic testing	Identify orthostatic intolerance contributing to symptoms	History of lightheadedness on standing or syncope

□ **Important:** Reflexive serial imaging in a patient who already meets PPPD criteria is not benign. It delays effective treatment, escalates health anxiety, and entrenches the threat appraisal that drives the disorder [2,8].

VI. Differential Diagnosis

The differential for chronic dizziness is broad, and PPPD is distinguished by its specific triad of provoking factors, its non-vertiginous quality, and a normal or compensated examination [1,8]. The central task is to separate PPPD from active disorders that require their own treatment, while recognising that several of these commonly coexist with — and continue to trigger — PPPD [1,10]. Vestibular migraine is the most important and most frequent companion, and bilateral assessment against both sets of criteria is often necessary [8,39].

Table 4. Differential diagnosis of chronic dizziness and the features that distinguish PPPD [1,8].

Condition	Distinguishing features from PPPD
Vestibular migraine	Discrete episodes of vertigo with migrainous features and headache; PPPD is continuous and non-episodic, though the two frequently coexist
BPPV	Brief positional spinning vertigo with diagnostic positional nystagmus; PPPD lacks true vertigo and positional nystagmus
Ménière's disease	Episodic vertigo with fluctuating low-frequency hearing loss, tinnitus and aural fullness; PPPD has no cochlear progression
Uncompensated vestibular hypofunction	Active deficit on vHIT/calorics with directional symptoms; PPPD shows compensated or normal testing
Orthostatic hypotension / POTS	Symptoms tied to standing with measurable blood-pressure or heart-rate change; PPPD provoked by motion and visual load too
Cerebellar or central disorder	Abnormal eye movements, ataxia or focal signs; PPPD examination is normal or only compensated

□ **Clinical Insight:** Coexistence is the rule, not the exception. A patient can have well-controlled Ménière's disease, a compensated neuritis or active vestibular migraine and still meet PPPD criteria — treat both layers rather than forcing a single label [1,10].

VII. Management — Education, Rehabilitation, CBT and Pharmacology

PPPD responds to a coordinated, multimodal programme that targets both the maladaptive balance control and the threat appraisal sustaining it [8,44]. No single modality is sufficient; the evidence favours combining patient education, vestibular rehabilitation, cognitive-behavioural therapy and serotonergic medication, matched to severity and comorbidity [8,30,32,44]. Figure 5 summarises the elements and the realistic outlook, and Figure 6 sets them out as a stepped-care pathway.

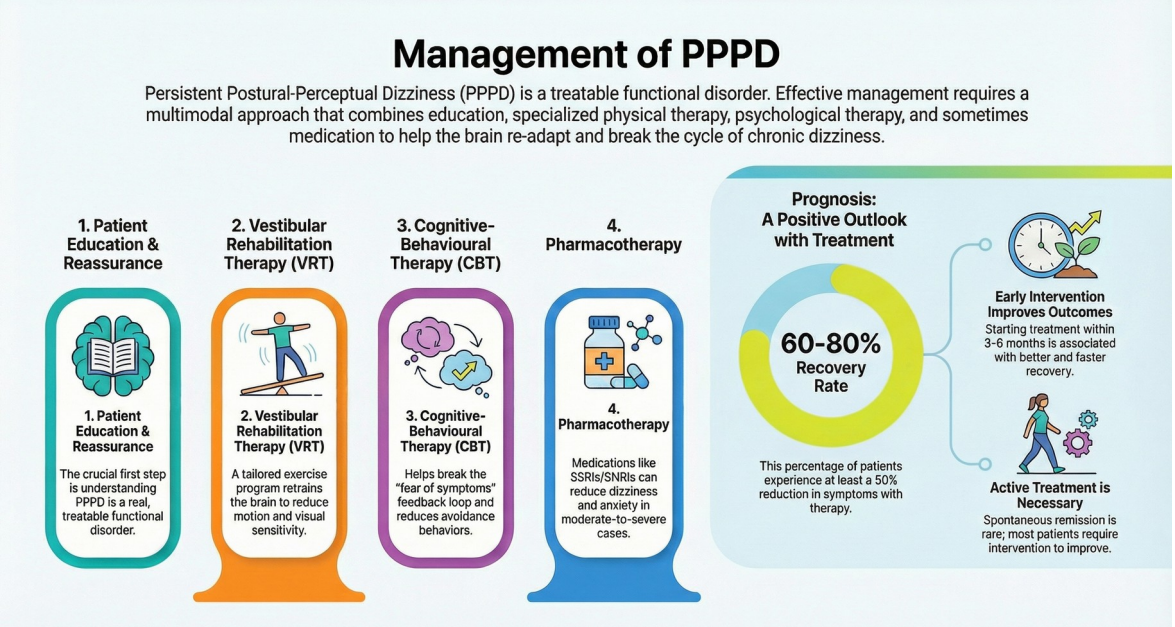

Figure 5. The four pillars of PPPD management and the realistic prognosis — education, vestibular rehabilitation, cognitive-behavioural therapy and pharmacotherapy, delivered together.
Source: Australian Dizziness Clinics, synthesising Popkirov et al. [8] and Axer et al. [44].

Education and reassurance

Explaining the diagnosis is itself therapeutic and is the indispensable first step [8]. Naming PPPD, validating that the symptoms are real, and framing them as a reversible fault in balance control rather than a hidden catastrophe directly counteracts the threat appraisal that perpetuates the disorder [8,38]. Patients who understand the mechanism engage far better with the active therapies that follow [8].

Vestibular rehabilitation

Vestibular rehabilitation is a core therapy. Graded habituation and motion- and visual-desensitisation exercises retrain the brain to tolerate the stimuli it has learned to avoid, progressively reducing visual dependence [32,33]. Programmes must be carefully paced — too aggressive a start can flare symptoms and reinforce avoidance — and outcomes are best when therapy is delivered by a vestibular physiotherapist familiar with the disorder [32,33]. Psychologically informed rehabilitation, which integrates cognitive techniques into the exercise programme, is an active area of trial development [45].

Cognitive-behavioural therapy

Cognitive-behavioural therapy targets the catastrophic interpretation, hypervigilance and avoidance that maintain PPPD, and randomised and controlled studies show meaningful symptom reduction, particularly when CBT is delivered early or combined with medication [30,31,37]. Brief, disorder-specific CBT appears more effective than generic supportive psychotherapy for established disease [31,37].

Pharmacotherapy

Serotonergic antidepressants are the pharmacological mainstay, used for their effect on the vestibular-limbic network rather than purely for mood [8,34,35]. Open-label and prospective data support selective serotonin reuptake inhibitors such as sertraline, paroxetine, escitalopram and fluoxetine, and serotonin-noradrenaline reuptake inhibitors such as venlafaxine, with response rates commonly in the 60–70 per cent range when treatment is sustained [34,35,36]. The governing principle is to start low and increase slowly, because PPPD patients are often sensitive to the initial activating side effects, and to continue an adequate trial of at least eight to twelve weeks before judging efficacy [8,34]. Medication works best alongside rehabilitation and CBT, not instead of them [8,44].

The evidence base, while still maturing, is internally consistent. Prospective and open-label trials of SSRIs and SNRIs report symptom reduction in the majority of patients who complete an adequate course, and

controlled studies of cognitive-behavioural therapy demonstrate benefit that is greatest when treatment begins early in the disease course [30,31,34,36]. Studies of vestibular rehabilitation tailored to PPPD show reduced handicap and improved balance confidence, and combination approaches that integrate rehabilitation with psychological therapy and medication consistently outperform single modalities [32,33,44]. Augmenting medication with disorder-specific CBT improves outcomes over medication alone, supporting the principle that the maladaptive control policy and the affective overlay must both be addressed [37]. Across modalities the recurring message is that coordination and adequate dose and duration, rather than the choice of any single agent, determine success [8,44].

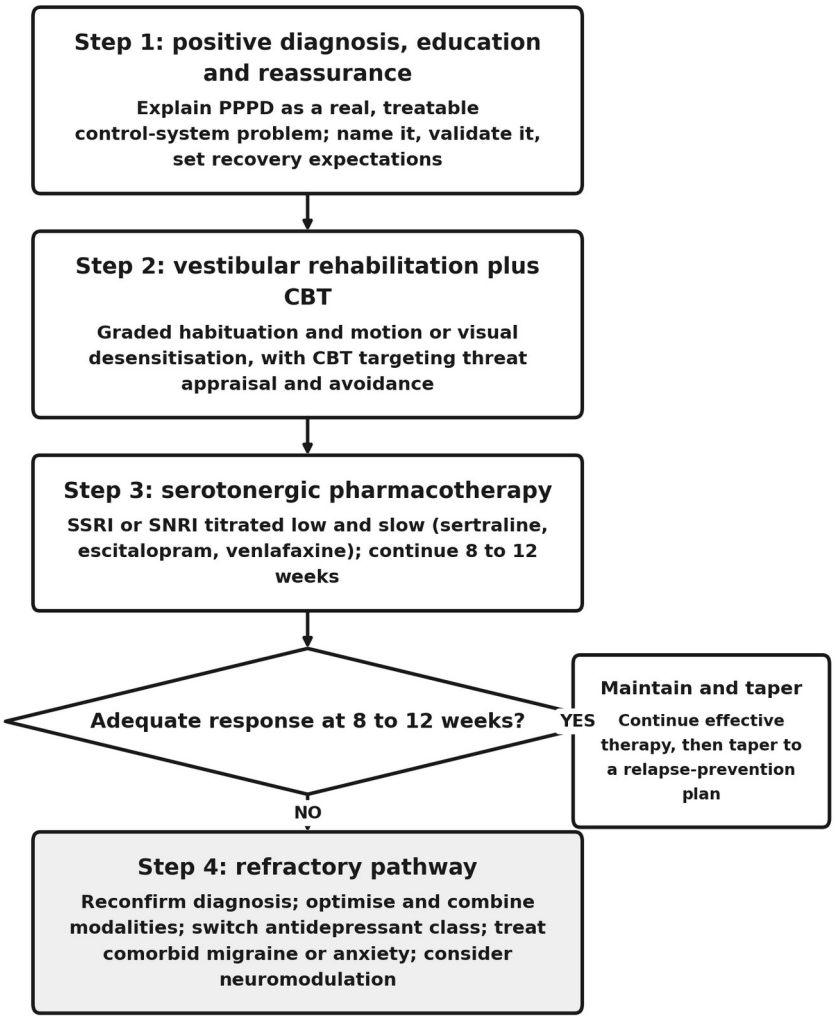

Figure 6. A stepped-care pathway for PPPD — beginning with diagnosis and education, adding vestibular rehabilitation and CBT, then serotonergic pharmacotherapy, with a defined route for refractory disease.
Source: Australian Dizziness Clinics, adapted from Popkirov et al. [8] and Staab et al. [34].

Table 5. Pharmacological options in PPPD — agents, rationale and cautions [8,34,35,36].

Class / agent	Rationale and dosing principle	Cautions
SSRI (sertraline, paroxetine, escitalopram, fluoxetine)	First-line; act on vestibular-limbic network; start low and titrate slowly	Early activation, nausea, sleep and sexual side effects; 8–12 week trial
SNRI (venlafaxine, duloxetine)	Alternative first-line; venlafaxine also benefits coexisting vestibular migraine	Blood pressure, insomnia; taper to avoid discontinuation effects
Low-dose tricyclic (selected)	Occasional adjunct where SSRIs/SNRIs not	Anticholinergic effects,

	tolerated	sedation; use with care
Vestibular suppressants (benzodiazepines)	Generally avoided; impede central adaptation and risk dependence	Not for maintenance; undermine rehabilitation

□ **Clinical Pearl:** The winning combination is rarely one thing. Education plus vestibular rehabilitation plus CBT plus a serotonergic agent, titrated slowly and given time to work, outperforms any single modality [8,44].

VIII. Refractory Disease, Neuromodulation and Emerging Therapies

A minority of patients fail to improve with optimised first-line care. The initial step in apparent refractoriness is to reconfirm the diagnosis and audit delivery — verifying that vestibular rehabilitation was correctly paced, that CBT was disorder-specific, that an antidepressant reached an adequate dose for an adequate duration, and that a coexisting active disorder such as vestibular migraine has not been left untreated [8,39,44]. Genuine treatment resistance is far less common than incomplete or uncoordinated treatment [44].

For true refractory disease, options include switching or combining antidepressant classes, intensifying combined rehabilitation and psychological therapy within a multidisciplinary programme, and aggressively treating comorbid migraine and anxiety [8,44]. Integrated multimodal programmes that bring these elements together report benefit in patients who had not responded to single modalities [44]. Non-invasive neuromodulation, including transcranial magnetic and transcranial direct-current stimulation directed at vestibular and affective cortical targets, is under investigation and remains experimental, appropriate only within expert or research settings [13,46]. Interest also continues in better characterising the brain-network signature so that treatment can eventually be targeted to the dominant mechanism in a given patient [13,46].

□ **Important:** Before labelling PPPD refractory, confirm the diagnosis and the dose: an under-dosed or short antidepressant trial, mis-paced rehabilitation, or an untreated coexisting vestibular migraine accounts for most apparent treatment failures [8,44].

Delivery model matters as much as content. The combination of education, vestibular rehabilitation, psychological therapy and pharmacotherapy is most reliably achieved within a coordinated multidisciplinary service, where a vestibular physician, a vestibular physiotherapist and a clinical psychologist share a common formulation and reinforce a consistent message to the patient [44]. Fragmented care — in which each clinician addresses only one element in isolation — is a frequent and remediable cause of apparent treatment resistance, because mixed or contradictory messages reactivate the threat appraisal the programme is trying to dismantle [8,44]. Where a full multidisciplinary team is not available, a single clinician who understands the whole model and sequences the elements deliberately can still deliver effective care [8].

IX. Prognosis, Relapse and Special Populations

With coordinated treatment the prognosis is good: most patients achieve a substantial reduction in symptoms and recover function and quality of life [8,44]. Early intervention matters — shorter symptom duration before diagnosis and prompt initiation of combined therapy are associated with better and faster recovery [8,10,44]. Spontaneous remission, by contrast, is uncommon; untreated PPPD tends to persist, which is the central argument for confident early diagnosis rather than watchful waiting [8].

Outcome is shaped by the same factors that predispose to the disorder, which can be organised within the three-P framework of predisposing, precipitating and perpetuating influences [8,20]. High trait anxiety, prominent catastrophic thinking, entrenched avoidance and a long delay to diagnosis all predict slower or partial recovery unless they are addressed directly [8,20,29]. Conversely, resilient coping and good psychological adjustment are protective and predict better outcomes [29].

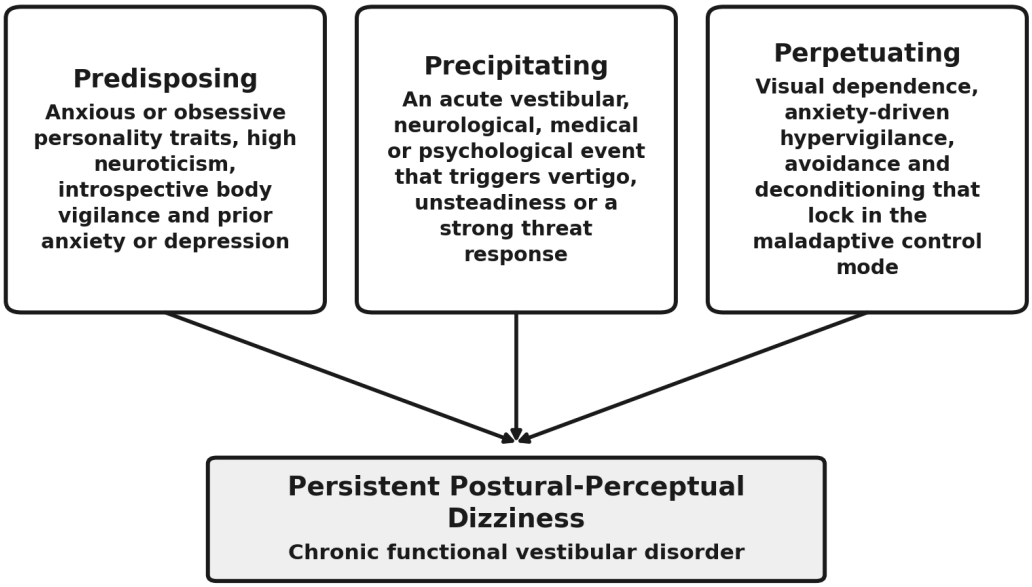

Figure 7. The three-P formulation of PPPD — predisposing traits, a precipitating event and perpetuating mechanisms combine to produce and maintain the disorder, and each layer is a treatment target.
Source: Adapted from Popkirov et al. [8] and Staab et al. [20].

Quantitatively, the trajectory under treatment is encouraging but rarely instantaneous. Most series describe meaningful improvement over weeks to months rather than days, with a substantial proportion of patients maintaining gains at one to two years when they continue some form of maintenance therapy [8,28,44]. Long-term follow-up of patients carrying PPPD or its historical equivalents shows that a large fraction return to work and resume curtailed activities, although a residual minority remain symptomatic, particularly where treatment was delayed or comorbid anxiety was left unmanaged [28,29,44]. These figures justify a confident, hopeful framing at the point of diagnosis, tempered by realistic expectations about the pace of recovery [8].

Table 6. Prognostic and relapse factors in PPPD and their influence on outcome [8,10,20,29,44].

Factor	Influence on outcome
Short symptom duration before diagnosis	Favourable — faster and more complete recovery
Early coordinated multimodal treatment	Favourable — the strongest modifiable predictor of success
Resilient coping and good psychological adjustment	Favourable — independently protective
High trait anxiety and catastrophic thinking	Unfavourable unless treated directly
Entrenched avoidance and deconditioning	Unfavourable — a primary target of rehabilitation
Untreated comorbid vestibular migraine	Unfavourable — treat in parallel
Medication non-adherence or early discontinuation	Unfavourable — a common driver of relapse

PPPD can relapse, typically when a new vestibular insult, a period of high stress or an intercurrent illness re-triggers the maladaptive control mode [8,10]. Patients should therefore leave treatment with a relapse-prevention plan — maintained habituation exercises, retained cognitive strategies, and a low threshold

for early re-engagement rather than a return to avoidance [8,32]. Non-adherence and premature discontinuation of medication, reported in around a fifth of patients in chronic-dizziness treatment, are themselves drivers of relapse and warrant proactive counselling [8].

In special populations, the syndrome is increasingly recognised in adolescents and young adults, where it often follows concussion, an episode of intense stress, or an acute vestibular event, and where attention to school participation and family understanding is important [8,11]. The same three-P logic and the same multimodal treatment apply across the age range, with dosing and psychological approach adapted to the individual [8].

□ **Clinical Insight:** Frame recovery for the patient around the three-P model: you cannot change what predisposed or precipitated you, but the perpetuating factors — avoidance, hypervigilance, deconditioning — are exactly what treatment dismantles [8].

X. Controversies, Guidelines and Future Directions

Although the Bárány Society criteria have brought welcome consensus, several questions remain open [1,8]. The first concerns visual dependence — whether it is a primary trait that predisposes to PPPD or a learned consequence of the disorder; the evidence that pre-existing visual dependence predicts chronic dizziness after neuritis suggests it is at least partly causal, but the relationship is likely bidirectional [16,17]. The second concerns nosological placement: PPPD straddles the boundary between neuro-otology and functional neurological disorder, and debate continues over whether it is best framed as a vestibular disorder with an affective overlay or as a functional disorder expressed in the vestibular system [8,13,46].

A further debate concerns the breadth of the criteria and the risk of over-diagnosis when symptoms overlap with ongoing vestibular disease, reinforcing the need to identify and treat coexisting active disorders rather than attributing everything to PPPD [1,10]. The role of short-term vestibular suppressants remains contested; the prevailing view is that they impede central adaptation and should be avoided in maintenance, even if they offer brief symptomatic relief [8]. Population data indicating that PPPD-like symptoms lie on a continuum raise the deeper question of where normal variation ends and disorder begins [11].

Future directions are converging on mechanism-guided care. Network neuroimaging may eventually stratify patients by their dominant abnormality — visual-cortical, vestibular-cortical or limbic — and match them to the most effective modality, while validated severity instruments such as the Niigata PPPD Questionnaire are improving the measurement of treatment response [13,25,46]. Trials of psychologically informed vestibular rehabilitation and of neuromodulation will clarify how best to help the refractory minority [44,45,46]. For now, the highest-value actions remain unchanged: recognise PPPD early, explain it well, and deliver coordinated multimodal treatment [2,8].

□ **Key Point:** The single most important determinant of outcome in PPPD is not a drug or a device but recognition: a confident, early, positively framed diagnosis that ends the cycle of investigation and opens the door to coordinated treatment [2,8].

References

[1] Staab JP, Eckhardt-Henn A, Horii A, Jacob R, Strupp M, Brandt T, Bronstein A. Diagnostic criteria for persistent postural-perceptual dizziness (PPPD): consensus document of the Committee for the Classification of Vestibular Disorders of the Bárány Society. J Vestib Res. 2017;27(4):191-208.

[2] Bisdorff A, Staab JP, Newman-Toker DE. Overview of the international classification of vestibular disorders. Neurol Clin. 2015;33(3):541-550.

[3] Brandt T, Dieterich M. Phobischer Attacken-Schwankschwindel, a new syndrome? Munch Med Wochenschr. 1986;128:247-250.

[4] Bronstein AM. Visual vertigo syndrome: clinical and posturography findings. J Neurol Neurosurg Psychiatry. 1995;59(5):472-476.

[5] Staab JP, Ruckenstein MJ. Expanding the differential diagnosis of chronic dizziness. Arch Otolaryngol Head Neck Surg. 2007;133(2):170-176.

[6] Staab JP. Chronic subjective dizziness. Continuum (Minneap Minn). 2012;18(5 Neuro-otology):1118-1141.

[7] Jacob RG, Furman JM, Durrant JD, Turner SM. Space and motion discomfort and abnormal balance control in patients with anxiety disorders. J Neurol Neurosurg Psychiatry. 2009;80(1):74-78.

[8] Popkirov S, Staab JP, Stone J. Persistent postural-perceptual dizziness (PPPD): a common, characteristic and treatable cause of chronic dizziness. Pract Neurol. 2018;18(1):5-13.

[9] Dieterich M, Staab JP. Functional dizziness: from phobic postural vertigo and chronic subjective dizziness to persistent postural-perceptual dizziness. Curr Opin Neurol. 2017;30(1):107-113.

[10] Trinidade A, Cabreira V, Goebel JA, Staab JP, Kaski D, Stone J. Predictors of persistent postural-perceptual dizziness (PPPD) and similar forms of chronic dizziness precipitated by peripheral vestibular disorders: a systematic review. J Neurol Neurosurg Psychiatry. 2023;94(11):904-915.

[11] Powell G, Derry-Sumner H, Rajenderkumar D, Rushton SK, Sumner P. Persistent postural perceptual dizziness is on a spectrum in the general population. Neurology. 2020;94(18):e1929-e1938.

[12] Indovina I, Riccelli R, Chiarella G, Petrolini C, Augimeri A, Giofre L, et al. Role of the insula and vestibular system in patients with chronic subjective dizziness: an fMRI study using sound-evoked vestibular stimulation. Front Neurol. 2015;6:91.

[13] Indovina I, Passamonti L, Mucci V, Chiarella G, Lacquaniti F, Staab JP. Brain correlates of persistent postural-perceptual dizziness: a review of neuroimaging studies. J Clin Med. 2021;10(18):4274.

[14] Riccelli R, Passamonti L, Toschi N, Nigro S, Chiarella G, Petrolo C, et al. Altered insular and occipital responses to simulated vertical self-motion in patients with persistent postural-perceptual dizziness. Front Neurol. 2017;8:529.

[15] Lee JO, Lee ES, Kim JS, Lee YB, Jeong Y, Choi BS, et al. Altered brain function in persistent postural perceptual dizziness: a study on resting state functional connectivity. Hum Brain Mapp. 2018;39(8):3340-3353.

[16] Cousins S, Cutfield NJ, Kaski D, Palla A, Seemungal BM, Golding JF, et al. Visual dependency and dizziness after vestibular neuritis. PLoS One. 2014;9(9):e105426.

[17] Cousins S, Kaski D, Cutfield N, Arshad Q, Ahmad H, Gresty MA, et al. Predictors of clinical recovery from vestibular neuritis: a prospective study. Ann Clin Transl Neurol. 2017;4(5):340-346.

[18] Soto-Varela A, Rossi-Izquierdo M, et al. Posturographic findings in patients with persistent postural-perceptual dizziness on the sensory organization test. J Vestib Res. 2016;26(3):319-326.

[19] Wuehr M, Pradhan C, Novozhilov S, Krafczyk S, Brandt T, Jahn K, Schniepp R. Inadequate interaction between open- and closed-loop postural control in phobic postural vertigo. J Neurol. 2013;260(5):1314-1323.

[20] Staab JP, Rohe DE, Eggers SDZ, Shepard NT. Anxious, introverted personality traits in patients with chronic subjective dizziness. J Psychosom Res. 2014;76(1):80-83.

[21] Yan Z, Cui L, Yu T, Liang H, Wang Y, Chen C. Analysis of the characteristics of persistent postural-perceptual dizziness: a clinical-based study in China. Int J Audiol. 2017;56(1):33-37.

[22] Kim HJ, Lee JO, Choi JY, Kim JS. Etiologic distribution of dizziness and vertigo in a referral-based dizziness clinic in South Korea. J Neurol. 2020;267(8):2252-2259.

[23] Schniepp R, Wuehr M, Huth S, Pradhan C, Brandt T, Jahn K. Gait characteristics of patients with phobic postural vertigo: effects of fear of falling, attention, and visual input. J Neurol. 2014;261(4):738-746.

[24] Habs M, Strobl R, Grill E, Dieterich M, Becker-Bense S. Primary or secondary chronic functional dizziness: does it make a difference? A DizzyReg study in 356 patients. J Neurol. 2020;267(Suppl 1):212-222.

[25] Yagi C, Morita Y, Kitazawa M, Nonomura Y, Yamagishi T, Ohshima S, et al. A validated questionnaire to assess the severity of persistent postural-perceptual dizziness (PPPD): the Niigata PPPD Questionnaire (NPQ). Otol Neurotol. 2019;40(7):e747-e752.

[26] Staab JP. Persistent postural-perceptual dizziness. Semin Neurol. 2020;40(1):130-137.

[27] Holle D, Schulte-Steinberg B, Wurthmann S, Naegel S, Ayzenberg I, Diener HC, et al. Persistent postural-perceptual dizziness: a matter of higher, central dysfunction? PLoS One. 2015;10(11):e0142468.

[28] Best C, Eckhardt-Henn A, Tschan R, Dieterich M. Why do subjective vertigo and dizziness persist over one year after a vestibular vertigo syndrome? Ann N Y Acad Sci. 2009;1164:334-337.

[29] Tschan R, Best C, Beutel ME, Knebel A, Wiltink J, Dieterich M, Eckhardt-Henn A. Patients' psychological well-being and resilient coping protect from secondary somatoform vertigo and dizziness. J Neurol. 2011;258(1):104-112.

[30] Edelman S, Mahoney AEJ, Cremer PD. Cognitive behavior therapy for chronic subjective dizziness: a randomized, controlled trial. Am J Otolaryngol. 2012;33(4):395-401.

[31] Holmberg J, Karlberg M, Harlacher U, 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.

[32] Nada EH, Ibraheem OA, Hassaan MR. Vestibular rehabilitation therapy outcomes in patients with persistent postural-perceptual dizziness. Ann Otol Rhinol Laryngol. 2019;128(4):323-329.

[33] Thompson KJ, Goetting JC, Staab JP, Shepard NT. Retrospective review and telephone follow-up to evaluate a physical therapy protocol for treating persistent postural-perceptual dizziness. J Vestib Res. 2015;25(2):97-104.

[34] Staab JP, Ruckenstein MJ, Amsterdam JD. A prospective trial of sertraline for chronic subjective dizziness. Laryngoscope. 2004;114(9):1637-1641.

[35] Horii A, Mitani K, Kitahara T, Uno A, Takeda N, Kubo T. Paroxetine, a selective serotonin reuptake inhibitor, reduces depressive symptoms and subjective handicaps in patients with dizziness. Otol Neurotol. 2004;25(4):536-543.

[36] Simon NM, Parker SW, Wernick-Robinson M, Oppenheimer JE, Hoge EA, Worthington JJ, et al. Fluoxetine for vestibular dysfunction and anxiety: a prospective pilot study. Psychosomatics. 2005;46(4):334-339.

[37] Yu YC, Xue H, Zhang YX, Zhou J. Cognitive behavior therapy as augmentation for sertraline in treating patients with persistent postural-perceptual dizziness. Biomed Res Int. 2018;2018:8518631.

[38] Whalley MG, Cane DA. A cognitive-behavioral model of persistent postural-perceptual dizziness. Cogn Behav Pract. 2017;24(1):72-89.

[39] Waterston J, Chen L, Mahadevan M, Bester C, Castle N, Karavitaki K, et al. Persistent postural-perceptual dizziness: precipitating conditions, comorbidities and treatment with cognitive behavioral therapy. Front Neurol. 2021;12:795516.

[40] Bittar RSM, Lins EMDvS. Clinical characteristics of patients with persistent postural-perceptual dizziness. Braz J Otorhinolaryngol. 2015;81(3):276-282.

[41] Adamec I, Juren Measki S, Krbot Skoric M, Jazbec V, Habek M. Persistent postural-perceptual dizziness: clinical and neurophysiological study. J Clin Neurosci. 2020;72:26-30.

[42] World Health Organization. International Classification of Diseases, 11th Revision (ICD-11): AB32.0 Persistent postural-perceptual dizziness. Geneva: World Health Organization; 2019.

[43] Sarna B, Risbud A, Lee A, Muhonen E, Abouzari M, Djalilian HR. Implications of persistent postural-perceptual dizziness: a systematic review. Otolaryngol Head Neck Surg. 2021;164(5):952-961.

[44] Axer H, Finn S, Wassermann A, Guntinas-Lichius O, Klingner CM, Witte OW. Multimodal treatment of persistent postural-perceptual dizziness. Brain Behav. 2020;10(12):e01864.

[45] Herdman D, Norton S, Pavlou M, Murdin L, Moss-Morris R. Protocol and methods for a randomised feasibility trial of psychologically informed vestibular rehabilitation for persistent dizziness (INVEST). J Neurol. 2022;269(8):4032-4044.

[46] Castro P, Bancroft MJ, Arshad Q, Kaski D. Persistent postural-perceptual dizziness (PPPD) from brain imaging to behaviour and perception. Brain Sci. 2022;12(6):753.

[47] Yagi C, Morita Y, Kitazawa M, Yamagishi T, Ohshima S, Izumi S, et al. Persistent postural-perceptual dizziness: a functional neuro-otological disorder. Auris Nasus Larynx. 2024;51(4):588-598.

[48] Mallinson AI, Longridge NS. Visual vestibular mismatch in patients treated with chronic subjective dizziness. J Otolaryngol Head Neck Surg. 2014;43(1):18.

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