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Treatment of Persistent Postural-Perceptual Dizziness (PPPD) and Related Disorders

Stoyan Popkirov, MD[1,*]
Jon Stone, PhD[2]
Dagny Holle-Lee, MD[3]

Address
[*,1]Department of Neurology, University Hospital Knappschaftskrankenhaus Bochum, Ruhr University Bochum, Bochum, Germany
Email: popkirov@gmail.com
[2]Centre for Clinical Brain Sciences, Western General Hospital, University of Edinburgh, Edinburgh, UK
[3]Dizziness and Vertigo Center Essen and Department of Neurology, University of Duisburg-Essen, Essen, Germany

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Abstract

Purpose of review Persistent postural-perceptual dizziness (PPPD) is a newly defined disorder of functional dizziness that in the International Classification of Diseases in its 11th revision (ICD-11) supersedes phobic postural vertigo and chronic subjective dizziness. Despite efforts to unify the diagnosis of functional (somatoform) dizziness, patients will present with a variety of triggers, perpetuating factors, and comorbidities, requiring individualized treatment. This article will review different treatment strategies for this common functional neurological disorder and provide practical recommendations for tailored therapy.
Recent findings An emerging understanding of the underlying pathophysiology that considers vestibular, postural, cognitive, and emotional aspects can enable patients to profit from vestibular rehabilitation, as well as cognitive-behavioral therapy (CBT). Crucially, approaches from CBT should inform and augment physiotherapeutic techniques, and, on the other hand, vestibular exercises or relaxation techniques can be integrated into CBT programs. Antidepressant medication might further facilitate rehabilitation, though the mechanisms are yet to be elucidated, and the level of evidence is low.
Summary In PPPD and related disorders, vestibular rehabilitation combined with CBT, and possibly supported by medication, can help patients escape a cycle of maladaptive balance control, recalibrate vestibular systems, and regain independence in everyday life.

Introduction

Persistent postural-perceptual dizziness (PPPD) is a disorder of chronic non-spinning vertigo, dizziness, or unsteadiness that is typically exacerbated by upright posture, movement, and complex visual stimulation [1••, 2•]. PPPD is a recently defined entity that will be newly introduced to the International Classification of Diseases in its 11th revision (ICD-11), but the syndrome has been identified and studied previously as chronic subjective dizziness (CSD) and phobic postural vertigo (PPV) [3, 4]. Although the diagnostic criteria are slightly different, studies on the epidemiology, pathological mechanisms, and treatment of CSD and PPV can be assumed to be highly relevant for PPPD and form the basis of current treatment strategies reviewed here.

PPPD cannot be attributed to a specific structural lesion within the vestibular system, but is rather a maladaptive dysfunction of balance control and vestibular processing. The mechanisms behind the development of PPPD are still a matter of research and are reviewed in detail elsewhere [2•, 3, 4]. Still, a summary of clinical observations and theoretical models pertaining to the pathophysiology is useful to appreciate potential treatment strategies. By definition, PPPD is triggered by an acute episode of dizziness, which is usually caused by a vestibular disorder, but may relate to a range of other types of medical events including a panic attack, head injury, or syncope. The physiological response to sudden dizziness or perceived unsteadiness is an automatic change in balance control: the gait stiffens (so-called high-risk postural control), and spatial orientation becomes reliant on visual and somatosensory rather than vestibular inputs ("visual-somatosensory dependence"). Normally, postural control systems re-adapt to normal functioning after a while. In PPPD, however, a cycle of maladaptation leads to persistent symptoms (Fig. 1) [2•]. In this model, increased self-monitoring will disturb previously automatic motor control and lead to overcompensatory movements (potentially resulting in gait disorders). At the same time, fearful expectation of dizziness and unsteadiness can disrupt and distort the processing of incoming sensory information, which is itself poorly filtered and integrated. Lastly, near-falls and subjective dizziness can promote anxiety and avoidance behavior, and maintain the abnormal postural control to complete the cycle. Figure 1 schematically shows how different treatment approaches can try to break the cycle of maladaptation that underlies PPPD.

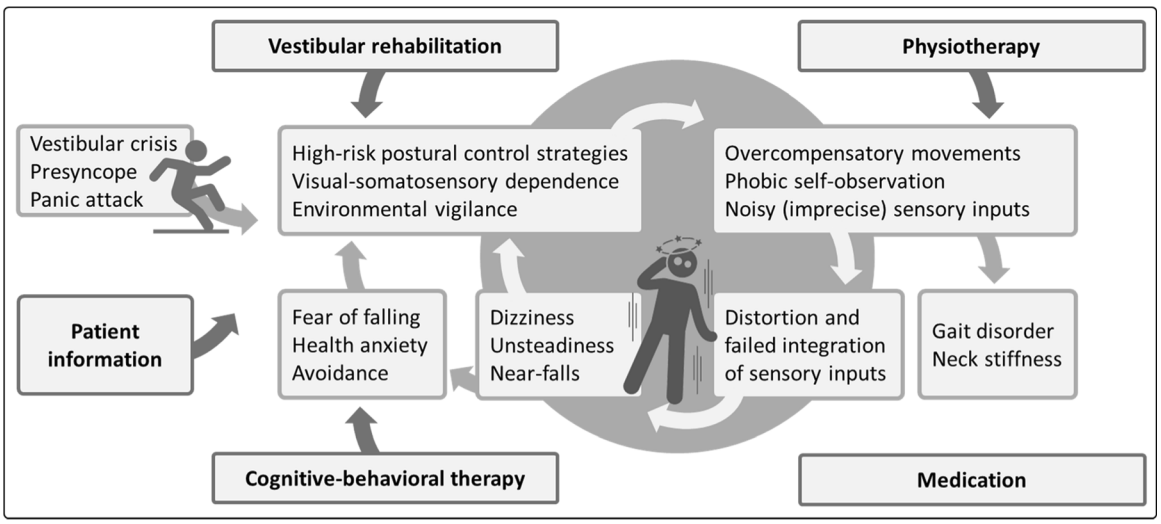

Fig. 1. Schematic overview of maladaptive pathophysiology and therapeutic options.

Treatment strategies

A close look at the pathophysiological model presented above reveals how different treatment strategies can help patients escape the cycle of maladaptive postural control and dizziness. Ideally, the clinician formulates the patient's case individually recognizing which elements of the presentation are "core" PPPD and assessing the extent to which other common comorbidities such as ongoing vestibular disorders, anxiety, functional gait disorder, or neck pain are complicating the picture.

First, patients need to be made aware that their condition is potentially reversible in principle, that it is not "just stress" or "all in the head." Once the diagnosis and basic underlying mechanisms have been communicated, patients may profit from vestibular rehabilitation that comprises exercises which can help recalibrate maladaptive postural strategies and re-habituate the vestibular, motor, and oculomotor systems to normal functioning. Other forms of physical therapy can aid this re-adaptation and also help with secondary problems such as functional gait disorder. Elements from cognitive-behavioral therapy (CBT) should be utilized in this treatment modality. In cases where psychological factors or psychiatric comorbidities are major driving factors, extended CBT can be useful. Finally, open-label trials have shown some benefit from selective serotonin and/or noradrenalin reuptake inhibitors (SSRI/SNRI). Electrical neural stimulation represents a novel treatment approach that remains to be tested clinically.

Communicating the diagnosis

In functional disorders such as PPPD, an informed understanding of the underlying mechanisms is crucial, more so than for most structural disorders [5•, 6, 7]. Persisting apprehensions about missed disease ("Could it be a brain tumor?") or catastrophic consequences ("What if I fall and injure myself?") can stand in the way of recovery. On the other hand, insisting that it is "all just stress/anxiety" or "in the mind" ignores the demonstrable changes in motor control and sensory integration, and will in all likelihood alienate patients who have no conscious control over their symptoms, and are often no more stressed, anxious, or depressed than patients with structural vestibular disorders [7]. Thus, several points should be made explicitly when communicating the diagnosis:

- PPPD is a common diagnosis made on the basis of characteristic positive signs and criteria, not one made solely by exclusion on the basis of negative scans and normal test results.
- While an incipient event, for example, a bout of vestibular neuritis, is often to blame for the development of PPPD, it is not residual damage that causes the symptoms, but an initially adaptive, and then *mal*adaptive response to the trigger that leads to dizziness. Even when residual vestibular damage is detected, the reversibility of PPPD should be emphasized.
- Psychological factors do play a role, but physicians should be aware that some patients can be sensitive to excessive "psychologizations," and rightly so. All too often, patients' symptoms will have been dismissed previously

as somehow less real, exaggerated, or even malingered when tests and scans came back normal. It is usually helpful to explain cognitive and emotional effects as one cogwheel in the machinery of the brain that is also turning, instead of the main driver of dysfunction.

- Metaphors and anecdotal explanations can be useful. Most patients will know the weird sensation of imbalance shortly after taking off ice skates or getting off a boat, or when standing at dizzying heights. These examples can be used to explain dizziness as an adaptive brain process that can get stuck; an "alert system" that has remained switched on by mistake, but can also be shut down gradually through therapy. This is, in simple terms, a software problem in the brain rather than a hardware one, and the treatment aims to "retrain the brain."
- Where possible, clinical signs, such as the reduction of body sway through distraction or the normalization of gait during forced, backwards, distracted, or running gait, can be demonstrated and explained to patients not just as positive diagnostic signs, but as evidence of reversibility, as proof of cognitive influences and as starting points for treatment [6, 8].
- PPPD is potentially treatable, but considering the modest prognosis for full recovery, and the duration of treatment required even in successful cases, overly optimistic predictions can be misunderstood as trivialization of a serious condition, and reduce confidence in the diagnosis should initial treatment fail.

Once patients show understanding and confidence in the diagnosis, further treatment strategies should be explained and initiated (see Stone et al. 2016 [5•] for a general guide on explanation of functional neurological disorders and Popkirov et al. 2018 [2•] for explaining PPPD).

Vestibular rehabilitation and physiotherapy

Vestibular rehabilitation is an umbrella term for a range of physical treatments that aim to compensate or retune impaired balance control in various vestibular and neurological disorders [9, 10•]. In PPPD and related disorders, the guiding principle is to desensitize a balance control system that is stuck on "high alert" by use of habituation exercises and relaxation techniques [9, 10•, 11]. Although vestibular rehabilitation was initially developed as a purely physical therapy, it has evolved to incorporate various cognitive-behavioral principles such as graded exposure and cognitive reframing, and often CBT and vestibular exercises are combined into one hybrid therapy (see also the next section) [10•].

A wide range of exercises can be used in vestibular rehabilitation: from more general and natural exercises, such as walking programs (gradual build-up of brisk walk of up to 30 min), to more specific dizziness-provoking head movements, which should be performed relatively slowly (see references [11, 12] for specific examples). Patients should be encouraged to become aware of any dizziness, but at the same time remain calm about it and persist with the exercises. To further promote the desensitization of the dizziness-alert-anxiety response, relaxation exercises such as diaphragmatic breathing or autogenic training can be used [13–15]. To help with the desensitization of visual vertigo or motion sensitivity, simulator-based exercises using head-mounted displays or wall projections can be used [11]. A simpler alternative is to let patients buy a large umbrella with stripes and spin it while seated and later while standing for

up to 2 min [13]. PPPD can sometimes lead to a functional gait disorder, which may respond to specialized physiotherapy [16]. In treating functional gait disorders, an explanation of a cognitive-behavioral framework is again essential; exercises will include distraction (dual task) techniques, as well as alternative or exaggerated gaits (e.g., backwards, running, sliding) that are then used to gradually build-up to a normal gait [17]. For all of the above, it is advisable to combine guided treatment by a physical therapist with unsupervised daily exercises at home.

Since most large trials of vestibular rehabilitation have been done on subjects with long-standing chronic vertigo, it is likely that study populations included, at least in part, patients that would now be diagnosed with PPPD [10•]. A few studies specifically on patients with PPPD have been published more recently. Thompson and colleagues conducted a retrospective review with telephone follow-up of 26 patients with PPPD who had undergone at least one session of vestibular and balance rehabilitation treatment [13]. Fourteen patients found treatment beneficial, and most of those reported partial or complete relief of symptoms. In another retrospective study, Morisod and colleagues looked at objectifiable treatment effects of vestibular rehabilitation in CSD patients who had benefited from treatment (42 out of 53 patients with reported treatment effects) [18]. While baseline posturography was abnormal in 79% of cases, this proportion dropped to 33% post-treatment. Interestingly, no CSD-specific pattern of posturographic abnormality could be identified in this study, suggesting different phenotypic subtypes of a complex pathophysiology. In a small prospective study on seven patients with CSD who had not responded to SSRI treatment, Goto and colleagues tested the usefulness of autogenic training [15]. Improvements in trait anxiety and dizziness were seen on self-report questionnaires, but the study's significance is limited by the small sample size. In summary, supervised and unsupervised physical exercises (especially habituation and relaxation techniques) are likely to be helpful for many PPPD patients, especially in combination with patient education and/or CBT.

Cognitive-behavioral therapy

The close psychosomatic interplay in PPPD lends itself to cognitive-behavioral conceptualization and treatment (Fig. 1) [19]. Early therapeutic strategies at vertigo treatment centers utilized core principles of CBT such as basic patient information combined with encouragement for self-controlled desensitization through exposure and moderate physical activity [20, 21]. CBT programs of various lengths, in individual or group settings, personally tailored or strictly manualized, were later developed and tested. The following components are included in most published treatment programs (see [19, 22, 23•, 24, 25] for individual summaries):

- Patient education, often referred to as "psychoeducation," figures in all CBT programs as an introductory session that conveys important aspects of pathophysiology. Brochures, information fliers, or internet websites can be of additional value to patients. Furthermore, in-session behavioral experiments and activities can be used to demonstrate and explain features such as distractibility and reversibility. This foundational part of CBT is crucial and should be started as soon as the diagnosis is established by the treating physician (see the "Communicating the diagnosis" section).

- Guided self-observation on physical, emotional, and psychosocial levels is the first step towards breaking out of maladaptive cognitive-behavioral cycles. Patients are taught to recognize abnormal postural control (stiffened stance, visual dependence) as well as overreaction to misinterpreted normal postural behavior (e.g., physiological spontaneous body sway). Desensitizing exercises can be used to increase tolerance of perceived disequilibrium and reduce automatic "high-risk" postural strategies (see previous section).
- Identification and appraisal of emotional and cognitive responses to dizziness can help reduce fear arousal and catastrophizing thoughts. Patients may be trained to respond to dizziness with calm detachment. Various relaxation techniques can be used to facilitate this (see previous section).
- Psychosocial factors such as fear of falling and social embarrassment, avoidance, and safety behaviors are identified and assessed. To address these, exposure therapy in various forms (with therapist, in group sessions, self-guided) and degrees (from general increase in physical activity to exposure to specific triggers) can be utilized.
- Treatment aims should be shifted in favor of reduction of handicap and normalization of everyday life, instead of exclusively on symptom reduction. A certain degree of dizziness and risk of falling needs to be accepted in order to prevent re-entry into cycles of anxiety and avoidance.

Several treatment trials have so far evaluated the benefits of CBT in treatment of PPPD and related disorders. Holmerg and colleagues tested the effects of 8–12 sessions of individualized CBT in addition to vestibular self-treatment (daily desensitization exercises) in 16 patients with PPV [22]. Compared to the exercises-only group, CBT-treated patients had significantly greater improvements in experienced handicap, anxiety, and depression. Unfortunately, treatment effects had disappeared on 1-year follow-up [26]. A waiting-list-controlled study of three weekly sessions of CBT for CSD on 41 patients demonstrated improvements in dizziness, disability, and safety behaviors [23•]. Treatment gains were still evident on 1- and 3-month follow-up [27]. In a waiting-list-controlled study on 24 patients with "somatoform vertigo and dizziness," 14 patients received group CBT in 10 weekly sessions of 100 min [28]. The study population showed significantly improved understanding of their illness and felt more in control of it; an effect that was largely sustained at 12-month follow-up. But although about two-thirds of patients described the program as "very helpful," no significant long-term reductions in dizziness and related handicap could be measured. In a follow-up study by the same group, 13 patients with somatoform vertigo and dizziness completed group CBT (6–8 participants per group; 10 weekly sessions á 90 min) [24]. Patients underwent exhaustive posturographic measurements before and after the psychotherapeutic intervention which demonstrated partial normalization of phobic postural control. A pre- and post-intervention questionnaire did not measure any significant corresponding improvements in dizziness-related handicap. A recently published study on 91 patients with PPPD and moderate depression tested the effect of CBT (16 twice weekly 60-min sessions) in addition to sertraline treatment compared to medication only [25]. The CBT group reported significantly larger improvements in handicap, anxiety, and depression scores than the medication-only group. Furthermore, the CBT group needed less

Table 1. Studies of SSRI or SNRI for chronic subjective dizziness and related disorders

	Year	Control group	Diagnosis	*n*[a]	Outcome measure	Medication	Completed treatment	Responders (intention-to-treat)
[31]	2002	None	Mixed[b]	60	CGI-I	Sertraline, fluoxetine, paroxetine, citalopram	45 (75%)	38 (63%)
[32]	2004	None	CSD	20	DHI, BSI-53, STAI, CES-D, HAM-A, HAM-D, CGI-I	Sertraline	15 (75%)	11 (55%)
[33]	2004	"Organic" vestibular disorder	Dizziness complaints with no evidence of organic disease	18	Modified DHI; self-rating depression scale	Paroxetine	n/a	n/a; improvement in depression and social activity in depressed individuals (6/18; 33%)
[34•]	2005	None	CSD and clinically significant anxiety	88	CGI-I	Sertraline, fluoxetine, paroxetine, citalopram, escitalopram	72 (82%)	59 (67%)
[35]	2007	"Organic" vestibular disorder	Dizziness complaints with no evidence of organic disease	30	HADS; modified DHI	Fluvoxamine	22 (73%)	n/a; reduction in handicap scores
[36]	2011	None	CSD and migraine	32	Not stated	Venlafaxine	24 (75%)	17 (53%)
[37]	2016	None	CSD[c]	12	HADS; modified DHI	Milnaciplan	n/a	n/a[c]
[25]	2018	+CBT[d]	PPPD + HAM-D≥17[e]	91[d]	DHI, HAM-A, HAM-D	Sertraline	91 (100%); 37% adverse effects	n/a; improvement in all outcome measures; better in +CBT group

[a]Number of patients in treatment group with functional dizziness disorder
[b]Psychogenic dizziness; dizziness due to neurotological condition as well as psychiatric symptoms; idiopathic dizziness
[c]Study population included various disorders, 12/29 patients had CSD; results were only reported for overall study group, and treatment effects for CSD were not reported separately
[d]Study compared sertraline + CBT vs. sertraline only; table reports results for all patients
[e]A score of 17 or more points on the Hamilton Depression Rating Scale indicates moderate, severe, or very severe depression
BSI-53, Brief Symptom Inventory-53; *CBT*; cognitive-behavioral therapy; *CES-D*, Centers for Epidemiological Studies-Depression Scale; *CGI-I*, Clinical Global Impressions-Improvement; *CSD*, chronic subjective dizziness; *DHI*, Dizziness Handicap Inventory; *HADS*, hospital anxiety and depression scale; *HAM-A*, Hamilton Anxiety Rating Scale; *HAM-D*, Hamilton Depression Rating Scale; *n/a*, not applicable due to study design or reporting; *STAI*, State-Trait Anxiety Index

sertraline and reported half as many adverse effects. An important limitation of this study is the lack of follow-up beyond the 8 weeks of twice weekly CBT to test for durability of treatment effects.

CBT appears to be a useful intervention for PPPD although its long-term benefits have yet to be established. It seems most effective when combined with in-session exercises, be they cognitive techniques [23•], vestibular rehabilitation exercises [24, 29], or relaxation techniques [22].

Medication

Selective serotonin reuptake inhibitors (SSRI) and serotonin-norepinephrine reuptake inhibitors (SNRI) are commonly recommended for chronic functional dizziness with and without psychiatric comorbidity [30•] (see Staab 2012 [30•] for recommended dosing strategies). It should be noted, however, that the evidence level of the commonly cited studies is low (Table 1) [25, 31–33, 34•, 35–37]. The existing studies are all open-label non-randomized studies, which is problematic for two reasons. First, in functional disorders, symptom expectations are highly relevant and significant placebo responses are to be expected. Second, only subjective investigator-rated and/or patient self-report outcome measures were used as study endpoints, potentially introducing various biases. Different agents have been studied, with two studies evaluating more than one SSRI [31, 34•]. Diagnoses at recruitment vary between studies, with some studies recruiting mixed populations with chronic dizziness [31, 37], and others only patients with dual diagnoses (CSD and anxiety [34•], PPPD and depression [25], CSD and migraine [36]). Usually, 18–27% of patients will discontinue SSRI, typically due to adverse effects or perceived ineffectiveness [31, 32, 34•, 35, 36]. From an intention-to-treat perspective, about half to two-thirds of patients can be expected to respond well to treatment with SSRI. Taken together, these studies suggest that SSRI (or SNRI) can be a useful treatment option in patients with PPPD. Randomized controlled and blinded trials are needed, however, to provide more robust evidence for this common and potentially useful clinical practice. Since rehabilitation from PPPD relies on "re-adaptation" of vestibular and balance control systems, vestibular suppressant drugs such as antihistamines and benzodiazepines can be expected to delay rather than help rehabilitation, and should be avoided if possible [38].

Electrical stimulation

Recent studies have provided evidence of altered brain activation and structure in PPPD [39, 40], so it is not surprising that experimental treatments using non-invasive neural stimulation are starting to emerge. In a recent pilot study, transcranial direct current stimulation (tDCS) to the left dorsolateral prefrontal cortex in eight PPPD patients (5 days of daily stimulation for 30 min) resulted in a transient reduction in subjective dizziness-related handicap during the days of stimulation but not on follow-up [41]. Another recent study looked at the effect of non-invasive vagus nerve stimulation (nVNS) [42]. A total of 16 patients with refractory PPPD received nVNS (3× 90-s stimulation during dizziness exacerbation and twice daily between attacks) which led to improvements in quality of life and depression and dizziness attack severity as assessed by self-report questionnaires, as well as improvement on measures of body sway during a postural challenge (standing on foam rubber). A "standard of

care" control group did not improve, but the study was not controlled through sham stimulation to estimate the placebo effect of self-applied neural stimulation. These are innovative approaches to functional dizziness, but further studies are needed to establish electrical stimulations as effective and safe treatment options in routine clinical practice.

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Compliance with Ethical Standards

Conflict of Interest
Stoyan Popkirov and Dagny Holle-Lee each declare no conflicts of interest. Jon Stone reports independent expert testimony work for personal injury and medical negligence claims and royalties from UpToDate for articles on functional neurological disorder, and runs a free non-profit self-help website, www.neurosymptoms.org, that discusses PPPD.

Human and Animal Rights and Informed Consent
This article does not contain any studies with human or animal subjects performed by any of the authors.

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- •• Of major importance

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