Persistent Postural-Perceptual Dizziness

Jeffrey P. Staab, MD, MS[1,2]

[1] Department of Psychiatry and Psychology, Mayo Clinic, Rochester, Minnesota
[2] Department of Otorhinolaryngology – Head and Neck Surgery, Mayo Clinic, Rochester, Minnesota

Address for correspondence Jeffrey P. Staab, MD, MS, Department of Psychiatry and Psychology, Mayo Clinic, 200 1st St SW, Rochester, MN 55905 (e-mail: staab.jeffrey@mayo.edu).

Semin Neurol

Abstract

Persistent postural-perceptual dizziness (PPPD) was defined for the International Classification of Vestibular Disorders in 2017. It is a chronic vestibular disorder that manifests with waxing and waning symptoms of dizziness, unsteadiness, or non-spinning vertigo that last for 3 months or more and are exacerbated by upright posture, active or passive motion of self, and exposure to environments with complex or moving visual stimuli. Triggers of PPPD include a wide variety of conditions that may cause vestibular symptoms or disrupt balance functioning, including neuro-otologic and other medical conditions and psychological distress. The diagnosis is made by identifying key symptoms in patients' histories and conducting physical examinations and diagnostic testing of sufficient detail to establish PPPD as opposed to other illnesses. Ongoing research is providing insights into the pathophysiological mechanisms underlying PPPD and support for multimodality treatment plans incorporating specially adapted vestibular rehabilitation, serotonergic medications, and cognitive-behavior therapy.

Keywords
- chronic dizziness
- visual dependence
- postural control
- neurologic disorder

Persistent postural-perceptual dizziness (PPPD) is a recent addition to the diagnostic nomenclature.[1] It was defined for the first time in 2017 when it was added to the International Classification of Vestibular Disorders (ICVD) compiled by the Bárány Society.[2] It also was included in the upcoming revision of the International Classification of Diseases by the World Health Organization (ICD-11).[3] However, descriptions of syndromes that are phenomenologically similar to PPPD can be found in the medical literature dating back to the 1870s.[1,4] Modern developments that foreshadowed the ICVD definition of PPPD began with descriptions of phobic postural vertigo in 1986,[5] followed by accounts of space-motion discomfort,[6] visual vertigo,[7] and chronic subjective dizziness[8] from 1993 to 2004. In 2010, the Classification Committee of the Bárány Society formed an expert subcommittee that included investigators responsible for describing each of these predecessors of PPPD to advise it on the possible existence of a unifying concept. That subcommittee wrote the diagnostic criteria for PPPD that were incorporated into the ICVD and ICD-11.[1,3] PPPD has completely superseded chronic subjective dizziness. Some experts have retained phobic postural vertigo to denote a clinical condition equivalent to PPPD, but with additional phobic features.[1] Space-motion discomfort and visual vertigo (renamed visually induced dizziness in the ICVD)[9] remain as complex symptoms, not independent diagnoses. They are key features of PPPD, but, importantly, also occur in other clinical circumstances such as during attacks of vestibular migraine or as sequelae of acute peripheral or central vestibular lesions.[7] PPPD filled an important gap in neuro-otologic practice. Prior to its introduction, clinical epidemiologic studies in tertiary referral centers showed that approximately 25% of patients who sought consultations for vestibular and balance symptoms received no diagnosis.[10,11] More recent studies from centers that included PPPD in their diagnostic schema found that number was reduced to fewer than 2% of new patient evaluations.[11,12] Moreover, long-standing symptoms nonspecifically attributed to chronic vestibulopathy in the past were more properly diagnosed as PPPD.

There have not yet been any large-scale epidemiologic studies conducted on the incidence or prevalence of PPPD,

Issue Theme Neuro-Otology; Guest Editor, Terry Fife MD, FAAN, FANS.

DOI https://doi.org/10.1055/s-0039-3402736.
ISSN 0271-8235.

but estimates may be extrapolated from previous research on phobic postural vertigo and chronic subjective dizziness.[13,14] In tertiary centers, these conditions constituted the principal diagnosis in 15 to 20% of all patients presenting for evaluation of vestibular symptoms, making them the most common diagnoses among young to middle-aged adults and the second most common among all adults, trailing only benign paroxysmal positional vertigo. Based on these older data and the most recent reports from tertiary neuro-otologic practices,[11,12] PPPD would be expected to rank among the top-three diagnoses encountered in referral centers, along with benign paroxysmal positional vertigo and vestibular migraine. The incidence and prevalence of PPPD in primary care and the general population are not known. The average age of patients presenting for the evaluation of PPPD is the mid-40s, with age of onset ranging from adolescence to late adulthood.[15,16] A female preponderance has been reported.[15] The incidence of PPPD following acute or episodic vestibular disorders (e.g., acute unilateral peripheral vestibulopathy, vestibular migraine, Meniere's disease) may be estimated from prospective studies that found rates of PPPD-like chronic dizziness or persistent visually induced dizziness of approximately 25% at 3 to 12 months of follow-up, despite otherwise adequate compensation or recovery from the initial illnesses.[17–20] A study that examined the natural course of phobic postural vertigo found that only a minority of patients experienced spontaneous resolution of symptoms over an average of 8.5 years.[21] Most had a chronic, waxing, and waning course of illness, and three-quarters developed anxiety or depressive comorbidity. Retrospective investigations of patients with chronic subjective dizziness described a similar course following precipitants other than peripheral vestibular disorders (e.g., anxiety disorders, autonomic disorders, mild traumatic brain injury). Disability caused by PPPD appears to vary widely, from patients with few limitations in daily functioning to those unable to work at all.[1] These results indicate that PPPD is likely to develop in a significant proportion of patients who experience acute or episodic vestibular symptoms or disruptions of balance function, regardless of the triggering event, and that the majority of patients with PPPD are likely to remain symptomatic without treatment. It is hoped that greater recognition of PPPD will reduce this unacceptably high level of morbidity.

Making the Diagnosis

The key to diagnosing PPPD is patients' clinical histories, because its diagnostic criteria include symptoms alone (►**Table 1**).[1] Importantly, PPPD is not a diagnosis of exclusion.[1,13,22,23] The diagnosis of PPPD is made not by ruling out other causes of patients' symptoms but by ruling in the disorder through careful attention to its key features in patients' clinical presentations. Evidence from physical examinations, laboratory testing, or neuroimaging helps determine if PPPD is the best diagnosis for patients' symptoms, either alone or in combination with other conditions. If patients' histories include all of the diagnostic criteria of PPPD, then data indicating the existence of other diseases suggest comorbidity, unless other conditions are able to account for the entirety of patients' symptoms better than PPPD (criterion E in ►**Table 1**). A diagnosis of PPPD should not be given to patients who report only nonspecific chronic vestibular symptoms or enigmatic balance complaints that do not fulfill all of its diagnostic criteria. In such cases, prospective monitoring may provide the clinical evidence needed to verify the presence of PPPD or exclude it from consideration.[1]

Table 1 International Classification of Vestibular Disorders diagnostic criteria for PPPD

All five criteria, A–E, must be fulfilled to make the diagnosis
A. One or more symptoms of dizziness, unsteadiness, or nonspinning vertigo are present on most days for 3 mo or more
1. Symptoms last for prolonged (hours-long) periods of time but may wax and wane in severity
2. Symptoms need not be present continuously throughout the entire day
B. Persistent symptoms occur without specific provocation, but are exacerbated by three factors:
1. Upright posture
2. Active or passive motion without regard to direction or position
3. Exposure to moving visual stimuli or complex visual patterns
C. The disorder is precipitated by conditions that cause vertigo, unsteadiness, dizziness, or problems with balance including acute, episodic, or chronic vestibular syndromes; other neurologic or medical illnesses; or psychological distress
1. When the precipitant is an acute or episodic condition, symptoms settle into the pattern of criterion A as the precipitant resolves, but they may occur intermittently at first, and then consolidate into a persistent course
2. When the precipitant is a chronic syndrome, symptoms may develop slowly at first and worsen gradually
D. Symptoms cause significant distress or functional impairment
E. Symptoms are not better accounted for by another disease or disorder

Abbreviation: PPPD, persistent postural-perceptual dizziness.
Source: Reprinted with permission from Staab JP, Eckhardt-Henn A, Horii A, et al. Diagnostic criteria for persistent postural-perceptual dizziness (PPPD): consensus document of the Committee for the Classification of Vestibular Disorders of the Barany Society. J Vestib Res 2017;27(4):191–208.

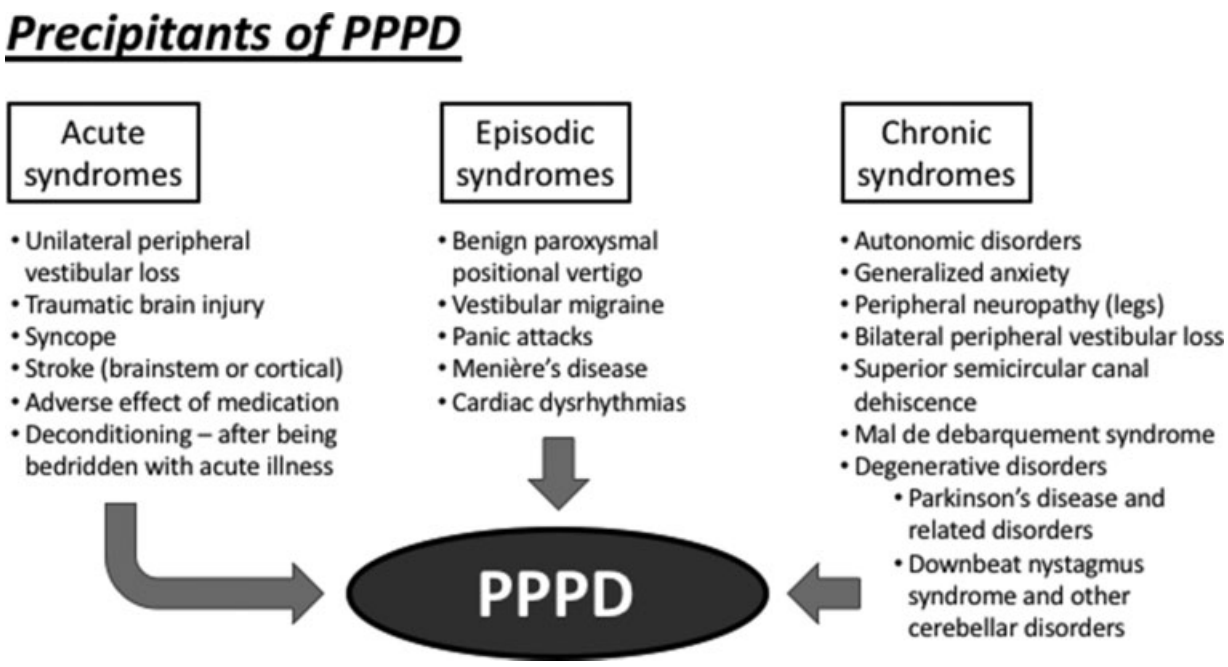

Fig. 1 Precipitants of persistent postural-perceptual dizziness (PPPD) include a wide array of acute, episodic, and chronic conditions. These triggers share an ability to cause vestibular symptoms or disrupt normal balance function.

The most common precipitants of PPPD are depicted in ►**Fig. 1**. These were derived from previous investigations of phobic postural vertigo[5,13] and chronic subjective dizziness[14] and more recent clinical experience with PPPD, itself. The key to understanding this wide range of precipitants is to recognize their unifying feature. They all share an ability to trigger vertigo, unsteadiness, or dizziness or to disrupt normal balance function. The most common triggering events are acute peripheral or central vestibular disorders (25%), panic attacks (15–20%, more common in young women), vestibular migraine (15–20%, more common in young to middle-aged women), generalized anxiety (15%, often unrecognized), mild traumatic brain injury or whiplash (10–15%, more common in young men), and dysautonomias (7%, more common in young women). Other medical events, such as cardiac dysrhythmias, adverse drug reactions, and deconditioning following major medical illnesses are encountered less often (1–2% each).

As it develops after triggering events, PPPD may progress along the three initial trajectories described in criterion C (►**Table 1**). Following monophasic acute syndromes such as an acute unilateral peripheral vestibulopathy, the symptoms of PPPD develop as the direct effects of the triggering illnesses fade, usually without an asymptomatic interval. In the setting of episodic precipitants, such as recurrent attacks of benign paroxysmal positional vertigo, vestibular migraine, or panic, the symptoms of PPPD may be intermittent at first before consolidating into a persistent course. The most difficult clinical situations are those in which patients report an indistinct onset and slowly progressive course of illness over a period of months or years. This typically occurs in the setting of insidious precipitants, which range from slowly developing generalized anxiety or dysautonomias such as postural orthostatic tachycardia syndrome in young adults to degenerative neurologic disorders such as Parkinson's disease and related conditions or cerebellar disorders in older individuals. In these cases, prospective follow-up may be needed to ensure adequate and complete diagnostic formulations.[1]

Another observation that may aid in the detection of PPPD is the nature of affected patients' responses to provocative stimuli (criterion B, ►**Table 1**). The clinical state of patients with PPPD typically reflects their cumulative responses to motion stimuli encountered over the previous hours or days, making them vulnerable to additional exacerbations of symptoms at almost any time. In contrast, patients with other vestibular disorders are affected by provoking factors when their illnesses are active (e.g., during attacks of vestibular migraine or Meniere's disease), but otherwise they are rather less susceptible to motion stimuli. Even patients with other chronic vestibular disorders like bilateral peripheral vestibulopathy can reduce symptoms quite quickly by holding still or removing themselves from motion-rich environments.[11]

►**Table 2** lists common considerations in the differential diagnosis of PPPD.[1] These may be divided into ongoing manifestations of its potential precipitants and other conditions capable of causing chronic vestibular symptoms or disruptions of balance function. The key to working through the differential diagnosis is to keep in mind that the final formulation of active diagnoses may include PPPD alone, other conditions alone, or PPPD coexisting with other diseases or disorders.[1,11,23] Following an acute syndrome, the crucial diagnostic determinations are whether ongoing symptoms suggest direct sequelae of the acute illness and whether they fulfill all of the diagnostic criteria for PPPD. A common example is vestibular symptoms that persist for longer than 3 months following an acute unilateral peripheral vestibulopathy such as vestibular neuronitis. If persistent symptoms include distinct episodes of head motion-provoked vertigo and physical examinations reveal positive responses to head thrust, headshake, or stepping tests,

Table 2 Differential diagnosis of PPPD

Chronic sequelae of acute precipitants	• Uncompensated peripheral vestibular loss
	• Residual deficits from traumatic brain injury, including postconcussive syndrome
	• Residual deficits from stroke
	• Persistent deconditioning
Coexisting manifestations of episodic syndromes (i.e., acute on chronic symptoms)	• Benign paroxysmal positional vertigo
	• Vestibular migraine
	• Panic attacks
	• Meniere's disease
	• Paroxysmal cardiac dysrhythmias
Ongoing manifestations of chronic precipitants	• Autonomic disorders
	• Generalized anxiety
	• Peripheral neuropathy
	• Bilateral peripheral vestibular loss
	• Superior semicircular canal dehiscence
	• Mal de debarquement syndrome
	• Neurodegenerative disorders
Other chronic conditions	• Adverse effects of maintenance medications (e.g., antihypertensives)
	• Agoraphobia • Fear of falling
	• Somatic symptom/bodily distress disorder
	• Functional gait disorder
	• White matter disease of the brain • Orthostatic tremor • Major depression

Abbreviation: PPPD, persistent postural-perceptual dizziness.

then affected patients have a persistent unilateral vestibulopathy. If persistent symptoms meet criteria A to D in ►**Table 1**, then they have PPPD. If both circumstances exist, then they have PPPD coexisting with a persistent vestibulopathy. Following episodic syndromes, the crucial determinations are whether patients' clinical histories indicate complete interictal recovery or residual effects of the episodic condition (e.g., panic disorder with residual generalized anxiety or Meniere's disease with a residual peripheral deficit) and whether persistent symptoms fulfill the diagnostic criteria of PPPD. The answers will determine if the best diagnostic formulation includes only one disease or disorder that is judged to be responsible for both episodic and chronic symptoms or two conditions, PPPD plus a coexisting episodic syndrome. In the case of most chronic conditions, criterion B.3 of the definition of PPPD (►**Table 1**) holds the key to the differential diagnosis. If patients report difficulty with exposure to complex patterns or moving visual stimuli when they are well supported in a seated and stationary position, then PPPD has likely developed in conjunction with the chronic precipitant. Another important aspect of the differential diagnosis of PPPD is that it does not cause major alterations in gait or falls or near falls. Patients with PPPD may report sensations of veering from side to side when walking and may exhibit a mildly slow or cautious gait on physical examination, but any more significant alterations in ambulation indicate the presence of a structural or functional gait disorder. For a more detailed discussion of the differential diagnosis of PPPD, please refer to its defining study by the Bárány Society,[1] which is available as an open access publication at https://www.jvr-web.org/ICVD.html.

Putative Pathophysiological Mechanisms of PPPD

Research on the four predecessors of PPPD[13,19,20,24–32] provided hypotheses about its pathophysiological mechanisms that have been supported and extended by rapidly emerging data from investigations into PPPD itself.[16,33–35] In past and present studies,[13,16,19,24–26] psychological factors have been shown to play distinct roles in the development of PPPD, but importantly, these factors are not present in all patients, and their existence does not indicate that PPPD is a psychiatric disorder.[1]

►**Fig. 2** depicts a model of PPPD that was first proposed in 2013,[11] after initial drafts of its diagnostic criteria were reviewed for the first time by the general membership of the Bárány Society. Physiological, psychological, and advanced brain imaging studies published since that time have supported this conceptualization.[16,33–40] As discussed earlier and shown on the left side of ►**Fig. 2**, PPPD may be triggered by neuro-otologic disorders, other structural or metabolic conditions, and psychological distress that cause vertigo, unsteadiness, or dizziness, or disrupt balance function. With the onset of precipitating events, patients make necessary adaptations. One is a shift in the relative weighting of sensory inputs about space and motion to favor visual cues over vestibular or somatosensory/proprioceptive data. Even when triggering events do not damage vestibular or somatosensory systems, the onset of vestibular and balance symptoms appears to engender a shift to favor visual inputs (i.e., visual dependence).[34] Perhaps this occurs because visual cues are the only ones, apart from less substantive auditory inputs, to contain information about spatial orientation and movement of objects in the environment at a distance from the self, a factor that may assume greater importance in the face of vestibular and balance symptoms. A second shift is to employ high-demand postural control strategies such as moving cautiously or using supports to maintain safe and secure locomotion.[33,35] The third is to maintain a higher level of vigilance about self-motion and movement in the environment.[19] These adaptations are useful in the short run but are expected to revert to normal as the precipitant resolves or compensation occurs. For most patients, neurotologic, medical, and behavioral recovery takes place instinctively over a time course in keeping with the nature of the triggering event. In contrast, heightened anxiety or excessive body

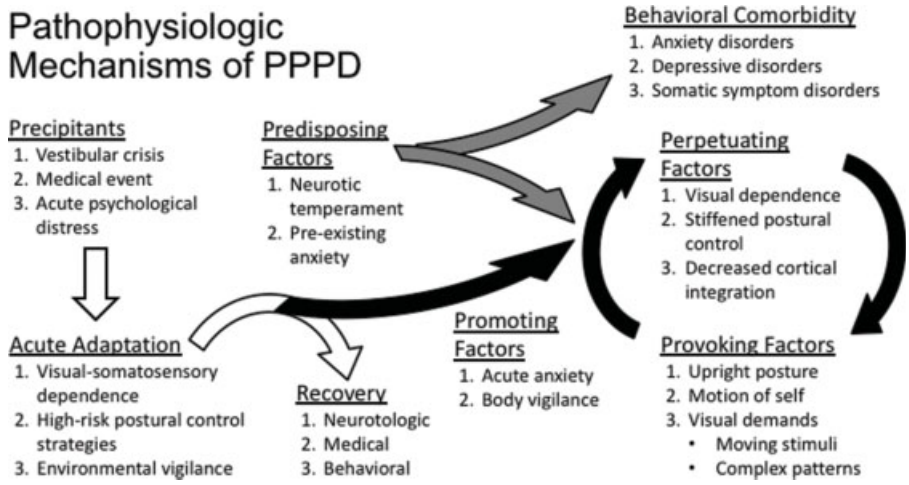

Fig. 2 Presumed pathophysiological processes leading to the development of persistent postural-perceptual dizziness (PPPD). White arrows—acute responses to events that trigger vestibular symptoms are normally adaptive and transient. Black arrows—heightened anxiety and excessive body vigilance promote ongoing reliance on visual inputs for spatial orientation and stiffening of postural control beyond what is necessary for recovery, perpetuating symptoms in the face of naturally occurring motion stimuli. Gray arrows—anxiety-related predisposing factors increase the likelihood of developing PPPD and behavioral comorbidity. (Updated with permission from Staab JP. Behavioral neuro-otology. In: Bronstein AM, ed. Oxford Textbook of Vertigo and Imbalance. Oxford, UK: Oxford University Press; 2013:333–346.)

vigilance in the face of incident vestibular symptoms seems to promote the development of PPPD by sustaining visual dependence and continued use of high-demand postural control strategies, which transform previously benign stimuli (e.g., self-motion and exposure to motion-rich environments) into highly provocative situations in a perpetual loop that sustains the symptoms of PPPD (right side of ►**Fig. 2**). Patients who have an anxiety diathesis in the form of a neurotic temperament or preexisting anxiety disorders appear to be at higher risk than other individuals for developing PPPD with or without coexisting behavioral morbidity.

Neuroimaging studies have buttressed the conceptualization of PPPD depicted in ►**Fig. 2** and identified another pathophysiological process, namely, altered activity and connectivity in cortical networks in the brain that subserve spatial orientation and locomotion.[36–40] In a study using resting state functional magnetic resonance imaging (fMRI), patients with PPPD compared with healthy controls had widespread reductions in connectivity from the left hippocampus (responsible for egocentric spatial navigation) to multiple brain regions. In contrast, connectivity between regions of the frontal and occipital cortices was increased in relation to mild state anxiety, consistent with the concept of visual dependence as a key process in PPPD.[38] In a fMRI study using sound-evoked vestibular stimuli, patients with chronic subjective dizziness compared with healthy controls had decreased activity in key areas of the right (dominant) vestibular cortex, and these regions had reduced connectivity with frontal regulatory regions, visual cortex, and hippocampus.[36] In a complementary fMRI investigation using visual motion stimuli from a virtual reality roller-coaster ride, patients with PPPD failed to activate the central insular sulcus (a region that helps resolve motion trajectories in gravity) compared with healthy controls, whereas they showed activation of primary visual cortical areas in proportion to the severity of their symptoms, again pointing to visual dependence as a crucial mechanism in PPPD.[37] Neuroimaging studies using advanced anatomical analyses have identified structural changes (reduced gray matter volumes[40] and decreased cortical folding[39]) in some of the regions that showed reduced activity and connectivity in the functional imaging investigations. Given the cross-sectional design of those studies, it is not possible to know if the structural alterations were sequelae of alterations in functioning (i.e., the result of brain plasticity) or subtle, preexisting structural defects that predisposed patients to the development of PPPD. These early neuroimaging results suggest that brain areas responsible for multisensory integration in support of high-level spatial orientation and safe and secure locomotion may not be as active or well-connected in patients with PPPD as in normal individuals, potentially leaving these tasks under the control of lower level brain networks. These imaging findings await replication and extension in larger prospective investigations that can correlate them with clinical and physiological data and track their responses to effective treatment.

Treatment

No large-scale, randomized, controlled trials of any treatments for PPPD have been done. However, several uncontrolled medication trials[8,41–45] and a few modest-sized controlled investigations of vestibular rehabilitation and psychotherapy[46–49] were completed on patients with chronic subjective dizziness and phobic postural vertigo, followed by one prospective study of medication versus medication plus psychotherapy[50] and one retrospective study of vestibular rehabilitation[51] for PPPD. Regarding medications, from 2002 to 2007 one large case series ($N = 60$)[8] and four open-label prospective trials ($N = 5$–60)[8,41–43] were published describing the use of selective serotonin reuptake inhibitors (SSRIs) in patients with chronic subjective dizziness[8] or PPPD-like chronic dizziness.[41–43] All six SSRIs that are commercially available in the United States were

included in at least one of those studies. Taken together, the following general conclusions may be drawn: (1) SSRIs were tolerated as well by patients with vestibular symptoms as by any other group of patients treated with these medications. (2) Approximately 65% of all patients who began treatment and 85% of patients who completed at least 8 to 12 weeks of treatment experienced a reduction in symptoms from moderately severe (partially impairing) to mild (not impairing). (3) Vestibular symptoms improved in concert with anxiety and depression in patients with psychiatric comorbidity, but the improvement in vestibular symptoms was not predicated on a reduction in psychiatric symptoms. (4) Mean and modal doses of SSRIs were in the lower half of the approved dose ranges for these medications. (6) No SSRI appeared to be more or less effective or better tolerated than the others.

Two studies were published showing the results of treatment with serotonin norepinephrine reuptake inhibitors (SNRIs).[44,45] Results of a study of 40 patients treated with milnacipran for chronic PPPD-like dizziness were comparable to that of the SSRI trials.[44] A case series of 32 patients with chronic subjective dizziness and vestibular migraine suggested that patients with coexisting anxiety disorders were more likely to experience improvements in all symptoms (dizziness, headache, anxiety) than those without anxiety disorders.[45]

Since the publication of the diagnostic criteria for PPPD, one randomized, prospective, but unblinded study compared treatment with sertraline (a SSRI) alone ($N = 45$) versus sertraline plus cognitive behavior psychotherapy ($N = 46$).[50] Patients who received combination therapy had significantly greater improvement after 4 and 8 weeks of treatment despite using a lower dose of medication than those who received sertraline alone. Patients in the sertraline-only arm achieved results that were comparable (slightly better) to those in a smaller ($N = 20$) prospective, unblinded study of sertraline for chronic subjective dizziness that was completed 14 years earlier.[8] These two clinical trials provide the most germane evidence to guide medication treatment of PPPD, though they provide only Level II evidence. Dosing strategies for sertraline, two other SSRIs, two SNRIs, and one alternative are listed in ►**Table 3** based on these limited open trial data and the author's clinical experience over the last two decades.

Two randomized controlled trials of cognitive behavioral therapy were published prior to PPPD being defined, one for phobic postural vertigo ($N = 31$)[46,47] and the other for chronic subjective dizziness ($N = 44$).[48,49] In the study of phobic postural vertigo, patients were randomized to self-directed exposure exercises versus 12 weeks of cognitive behavioral therapy. Patients who received cognitive behavioral therapy had greater symptom improvements at the end of active treatment,[46] but benefits were lost at 12-month follow-up.[47] In the study of chronic subjective dizziness, patients were randomized to active treatment with just three sessions of cognitive behavior therapy or a wait list control condition. Patients who received active treatment had large reductions in dizziness and dizziness-related handicaps that were retained at 6-month follow-up.[48,49] The most important difference between these two studies was that patients with phobic postural vertigo had chronic illness, whereas those with chronic subjective dizziness were enrolled within 8 months of illness onset, which suggests that cognitive behavior therapy might be effective as an early intervention strategy for PPPD.

Table 3 Dosing recommendations for selected medications for PPPD[a]

	Initial dose	Therapeutic dose range[b]
Selective serotonin reuptake inhibitors		
Sertraline	25 mg daily	50–150 mg daily
Escitalopram	5 mg daily	10–20 mg daily
Fluoxetine	10 mg daily	20–60 mg daily
Serotonin norepinephrine reuptake inhibitors		
Venlafaxine extended release	37.5 mg daily	75–225 mg daily
Duloxetine	20–30 mg daily	40–60 mg daily
Alternative		
Mirtazapine	7.5 mg nightly	15–30 mg nightly

Abbreviation: PPPD, persistent postural-perceptual dizziness.
[a]These are the medications most commonly used by the author. Dosing recommendations for additional medications may be found in Staab JP. Chronic subjective dizziness. Continuum (Minneapolis, MN). 2012;18 (5 Neuro-otology):1118–1141.
[b]Dose ranges apply to uncomplicated cases of PPPD. Patients with coexisting anxiety or depressive disorders may need higher doses for their psychiatric illnesses.

Vestibular rehabilitation was developed in the early 1990s to treat patients with chronic vestibular symptoms of any kind.[52,53] As the predecessors of PPPD were described, various forms of vestibular rehabilitation were applied to them, in the clinic and at home with self-directed or therapist-designed habituation plans.[54] The most sophisticated plans included stimulation in virtual reality and were developed to treat patients with visually induced dizziness that complicated structural vestibular lesions.[55,56] As for PPPD specifically, one retrospective study reported that most patients with PPPD found a physical therapy consultation to be helpful, and a majority experienced clinically significant improvements in symptoms with home-based exercises, though benefits for symptoms related to self-motion were greater than for those related to visual motion in the environment.[51] ►**Table 4** lists several physical therapy and cognitive-behavioral therapy strategies that may be used to treat specific symptoms of PPPD and its behavioral complications.

Conclusion

The definition of PPPD was established in 2017, but descriptions of patients struggling with similar symptoms date back at least one and a half centuries. In tertiary neurology and otology centers that routinely recognize PPPD, it is the most common cause of chronic dizziness and one of the top-three diagnoses

Table 4 Recommendations regarding physical therapy and psychotherapy for PPPD

Symptom/behavior	Vestibular rehabilitation	Cognitive behavior therapy
Stiffened stance and gait (including coexisting functional gait disorder)	• Normalize stance (relaxed posture, normal weight distribution) • Normalize gait (relaxed gait, natural stride, eliminate unneeded gait aids)	• Reduce excessive body vigilance • Counter fears of dizziness or falling
Sensitivity to own movements	• Habituation exercises (progressive increase in head, eye, and body movements)	• Reduce excessive body vigilance • Desensitization strategies to complement physical therapy
Visual dependence	• Habituation exercises (progressive exposure to increasingly complex patterns and moving visual stimuli)	• Reduce excessive body vigilance • Desensitization strategies to complement physical therapy
Avoidance of provocative environments	• Gradual exposure	• Counter anticipatory anxiety • Promote gradual exposure

Abbreviation: PPPD, persistent postural-perceptual dizziness.

identified in adults seeking consultation for vestibular symptoms. Data emerging from physiological, psychological, and advanced neuroimaging investigations are converging in support of a pathophysiological model of PPPD that includes three interconnected mechanisms of illness: (1) shifts in the functioning of spatial orientation systems to favor visual stimuli over vestibular and somatosensory/proprioceptive inputs (i.e., visual dependence), (2) alterations in locomotor control to stiffen stance and gait, and (3) reduced activity and connectivity in cortical networks that support these processes. Recommended treatment strategies are based on interventions described in uncontrolled and small randomized clinical trials of the four precursors of PPPD and the first therapeutic trials of PPPD, itself. These indicate that specifically modified vestibular rehabilitation and treatment with SSRIs/SNRIs, supplemented with cognitive-behavioral therapy, are able to substantially reduce the morbidity of PPPD for most patients.

Conflict of Interest
None declared.

References

1 Staab JP, Eckhardt-Henn A, Horii A, et al. 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(04):191–208
2 Bisdorff AR, Staab JP, Newman-Toker DE. Overview of the International Classification of Vestibular Disorders. Neurol Clin 2015; 33(03):541–550, vii
3 World Health Organization. ICD-11 for Mortality and Morbidity Statistics (Version 04/2019). AB32.0 Persistent Postural-Perceptual Dizziness. Available at: https://icd.who.int/browse11/l-m/en#/http%3a%2f%2fid.who.int%2ficd%2fentity%2f2005792829. Accessed July 14, 2019
4 Balaban CD, Jacob RG. Background and history of the interface between anxiety and vertigo. J Anxiety Disord 2001;15(1-2):27–51
5 Brandt T, Dieterich M. Phobischer Attacken Schwankschwindel, ein neues Syndrom? Munch Med Wochenschr 1986;128:247–250
6 Jacob RG, Lilienfeld SO, Furman JMR, Durrant JD, Turner SM. Panic disorder with vestibular dysfunction: further clinical observation and description of space and motion phobic stimuli. J Anxiety Disord 1989;3:117–130
7 Bronstein AM. Visual vertigo syndrome: clinical and posturography findings. J Neurol Neurosurg Psychiatry 1995;59(05):472–476
8 Staab JP, Ruckenstein MJ, Amsterdam JD. A prospective trial of sertraline for chronic subjective dizziness. Laryngoscope 2004; 114(09):1637–1641
9 Bisdorff A, Von Brevern M, Lempert T, Newman-Toker DE. Classification of vestibular symptoms: towards an International Classification of Vestibular Disorders. J Vestib Res 2009;19(1-2):1–13
10 Muelleman T, Shew M, Subbarayan R, et al. Epidemiology of dizzy patient population in a neurotology clinic and predictors of peripheral etiology. Otol Neurotol 2017;38(06):870–875
11 Staab JP. Behavioural neuro-otology. In: Bronstein AM, ed. Oxford Textbook of Vertigo and Imbalance. Oxford, UK: Oxford University Press; 2013:333–346
12 Staibano P, Lelli D, Tse D. A retrospective analysis of two tertiary care dizziness clinics: a multidisciplinary chronic dizziness clinic and an acute dizziness clinic. J Otolaryngol Head Neck Surg 2019; 48(01):11
13 Brandt T. Phobic postural vertigo. Neurology 1996;46(06):1515–1519
14 Staab JP, Ruckenstein MJ. Expanding the differential diagnosis of chronic dizziness. Arch Otolaryngol Head Neck Surg 2007;133 (02):170–176
15 Bittar RSM, Lins EM. Clinical characteristics of patients with persistent postural-perceptual dizziness. Rev Bras Otorrinolaringol (Engl Ed) 2015;81(03):276–282
16 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(01):33–37
17 Godemann F, Siefert K, Hantschke-Brüggemann M, Neu P, Seidl R, Ströhle A. What accounts for vertigo one year after neuritis vestibularis - anxiety or a dysfunctional vestibular organ? J Psychiatr Res 2005;39(05):529–534
18 Heinrichs N, Edler C, Eskens S, Mielczarek MM, Moschner C. Predicting continued dizziness after an acute peripheral vestibular disorder. Psychosom Med 2007;69(07):700–707
19 Cousins S, Kaski D, Cutfield N, et al. Predictors of clinical recovery from vestibular neuritis: a prospective study. Ann Clin Transl Neurol 2017;4(05):340–346
20 Best C, Tschan R, Eckhardt-Henn A, Dieterich M. Who is at risk for ongoing dizziness and psychological strain after a vestibular disorder? Neuroscience 2009;164(04):1579–1587

21 Huppert D, Strupp M, Rettinger N, Hecht J, Brandt T. Phobic postural vertigo–a long-term follow-up (5 to 15 years) of 106 patients. J Neurol 2005;252(05):564–569
22 Staab JP. Chronic subjective dizziness. Continuum (Minneap Minn) 2012;18(5 Neuro-otology):1118–1141
23 Dieterich M, Staab JP, Brandt T. Functional (psychogenic) dizziness. Handb Clin Neurol 2016;139:447–468
24 Staab JP, Rohe DE, Eggers SD, Shepard NT. Anxious, introverted personality traits in patients with chronic subjective dizziness. J Psychosom Res 2014;76(01):80–83
25 Chiarella G, Petrolo C, Riccelli R, et al. Chronic subjective dizziness: analysis of underlying personality factors. J Vestib Res 2016; 26(04):403–408
26 Tschan R, Best C, Beutel ME, et al. Patients' psychological well-being and resilient coping protect from secondary somatoform vertigo and dizziness (SVD) 1 year after vestibular disease. J Neurol 2011; 258(01):104–112
27 Krafczyk S, Tietze S, Swoboda W, Valkovic P, Brandt T. Artificial neural network: a new diagnostic posturographic tool for disorders of stance. Clin Neurophysiol 2006;117(08):1692–1698
28 Wuehr M, Pradhan C, Novozhilov S, et al. Inadequate interaction between open- and closed-loop postural control in phobic postural vertigo. J Neurol 2013;260(05):1314–1323
29 Holmberg J, Tjernström F, Karlberg M, Fransson PA, Magnusson M. Reduced postural differences between phobic postural vertigo patients and healthy subjects during a postural threat. J Neurol 2009;256(08):1258–1262
30 Querner V, Krafczyk S, Dieterich M, Brandt T. Patients with somatoform phobic postural vertigo: the more difficult the balance task, the better the balance performance. Neurosci Lett 2000;285(01):21–24
31 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(04):738–746
32 Ödman M, Maire R. Chronic subjective dizziness. Acta Otolaryngol 2008;128(10):1085–1088
33 Söhsten E, Bittar RS, Staab JP. Posturographic profile of patients with persistent postural-perceptual dizziness on the sensory organization test. J Vestib Res 2016;26(03):319–326
34 Staab JP, Wheeler LV, Shepard NT, Bronstein AM. Effects of clinical state and visual background on visual dependence measured by a modified rod and frame/disk test. Presented at the 29th meeting of the Bárány Society, Seoul, Korea. J Vestib Res 2016;26(1–2):48
35 Shepard N, McCaslin D, Staab J, Eggers S. Sensory Organization Test profile for patients with persistent postural-perceptual dizziness. Presented at the 30th meeting of the Bárány Society, Uppsala, Sweden. J Vestib Res 2018;28(1–2):185
36 Indovina I, Riccelli R, Chiarella G, et al. Role of the insula and vestibular system in patients with chronic subjective dizziness: an fMRI study using sound-evoked vestibular stimulation. Front Behav Neurosci 2015;9:334
37 Riccelli R, Passamonti L, Toschi N, et al. Altered insular and occipital responses to simulated vertical self-motion in patients with persistent postural-perceptual dizziness. Front Neurol 2017;8:529
38 Lee JO, Lee ES, Kim JS, et al. Altered brain function in persistent postural perceptual dizziness: a study on resting state functional connectivity. Hum Brain Mapp 2018;39(08):3340–3353
39 Nigro S, Indovina I, Riccelli R, et al. Reduced cortical folding in multi-modal vestibular regions in persistent postural perceptual dizziness. Brain Imaging Behav 2019;13(03):798–809
40 Wurthmann S, Naegel S, Schulte Steinberg B, et al. Cerebral gray matter changes in persistent postural perceptual dizziness. J Psychosom Res 2017;103:95–101
41 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(04):536–543
42 Simon NM, Parker SW, Wernick-Robinson M, et al. Fluoxetine for vestibular dysfunction and anxiety: a prospective pilot study. Psychosomatics 2005;46(04):334–339
43 Horii A, Uno A, Kitahara T, et al. Effects of fluvoxamine on anxiety, depression, and subjective handicaps of chronic dizziness patients with or without neuro-otologic diseases. J Vestib Res 2007;17(01):1–8
44 Staab JP. Clinical clues to a dizzying headache. J Vestib Res 2011;21 (06):331–340
45 Horii A, Imai T, Kitahara T, et al. Psychiatric comorbidities and use of milnacipran in patients with chronic dizziness. J Vestib Res 2016;26(03):335–340
46 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(04):500–506
47 Holmberg J, Karlberg M, Harlacher U, Magnusson M. One-year follow-up of cognitive behavioral therapy for phobic postural vertigo. J Neurol 2007;254(09):1189–1192
48 Edelman S, Mahoney AE, Cremer PD. Cognitive behavior therapy for chronic subjective dizziness: a randomized, controlled trial. Am J Otolaryngol 2012;33(04):395–401
49 E J Mahoney A, Edelman S, D Cremer P. . Cognitive behavior therapy for chronic subjective dizziness: longer-term gains and predictors of disability. Am J Otolaryngol 2013;34(02):115–120
50 Yu Y-C, Xue H, Zhang Y-X, Zhou J. Cognitive behavior therapy as augmentation for sertraline in treating patients with persistent postural-perceptual dizziness. BioMed Res Int 2018;2018:8518631
51 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: a pilot study. J Vestib Res 2015;25(02):97–103, quiz 103–104
52 Shepard NT, Telian SA. Programmatic vestibular rehabilitation. Otolaryngol Head Neck Surg 1995;112(01):173–182
53 Whitney SL, Rossi MM. Efficacy of vestibular rehabilitation. Otolaryngol Clin North Am 2000;33(03):659–672
54 Staab JP. Behavioral aspects of vestibular rehabilitation. Neuro-Rehabilitation 2011;29(02):179–183
55 Pavlou M, Kanegaonkar RG, Swapp D, Bamiou DE, Slater M, Luxon LM. The effect of virtual reality on visual vertigo symptoms in patients with peripheral vestibular dysfunction: a pilot study. J Vestib Res 2012;22(5-6):273–281
56 Alahmari KA, Sparto PJ, Marchetti GF, Redfern MS, Furman JM, Whitney SL. Comparison of virtual reality based therapy with customized vestibular physical therapy for the treatment of vestibular disorders. IEEE Trans Neural Syst Rehabil Eng 2014; 22(02):389–399