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Diagnostic criteria for persistent postural-perceptual dizziness (PPPD): Consensus document of the committee for the Classification of Vestibular Disorders of the Bárány Society

Jeffrey P. Staab[a,*], Annegret Eckhardt-Henn[b], Arata Horii[c], Rolf Jacob[d], Michael Strupp[e], Thomas Brandt[e] and Adolfo Bronstein[f]

[a]*Departments of Psychiatry and Psychology and Otorhinolaryngology – Head and Neck Surgery, Mayo Clinic, Rochester, MN, USA*
[b]*Department of Psychosomatic Medicine, Klinikum Stuttgart, Stuttgart, Germany*
[c]*Department of Otorhinolaryngology, Niigata University, Niigata, Japan*
[d]*Department of Psychiatry, University of Pittsburgh, Pittsburgh, PA, USA*
[e]*Department of Neurology and German Center for Vertigo and Balance Disorders, University Hospital, LMU Munich, Germany*
[f]*Neuro-Otology Unit, Division of Brain Sciences, Imperial College London, London, UK*

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Abstract. This paper presents diagnostic criteria for persistent postural-perceptual dizziness (PPPD) to be included in the International Classification of Vestibular Disorders (ICVD). The term PPPD is new, but the disorder is not. Its diagnostic criteria were derived by expert consensus from an exhaustive review of 30 years of research on phobic postural vertigo, space-motion discomfort, visual vertigo, and chronic subjective dizziness. PPPD manifests with one or more symptoms of dizziness, unsteadiness, or non-spinning vertigo that are present on most days for three months or more and are exacerbated by upright posture, active or passive movement, and exposure to moving or complex visual stimuli. PPPD may be precipitated by conditions that disrupt balance or cause vertigo, unsteadiness, or dizziness, including peripheral or central vestibular disorders, other medical illnesses, or psychological distress. PPPD may be present alone or co-exist with other conditions. Possible subtypes await future identification and validation. The pathophysiologic processes underlying PPPD are not fully known. Emerging research suggests that it may arise from functional changes in postural control mechanisms, multi-sensory information processing, or cortical integration of spatial orientation and threat assessment. Thus, PPPD is classified as a chronic functional vestibular disorder. It is not a structural or psychiatric condition.

Keywords: Chronic subjective dizziness, phobic postural vertigo, space motion discomfort, visual vertigo, classification, Bárány Society

*Corresponding author: Jeffrey P. Staab, MD, MS, Department of Psychiatry and Psychology, Mayo Clinic, 200 1st St SW, Rochester, MN 55905 USA. Tel.: +1 507 284 4159; Fax: +1 507 284 4158; E-mail: staab.jeffrey@mayo.edu.

Abbreviations

BPPV	benign paroxysmal positional vertigo
CCBS	Committee for the Classification Vestibular Disorders of the Bárány Society
CSD	chronic subjective dizziness
fMRI	functional magnetic resonance imaging
ICD-11	International Classification of Diseases, 11th edition (beta draft)
ICVD	International Classification of Vestibular Disorders
MdDS	mal de debarquement syndrome
PIVC	parieto-insular vestibular cortex
POTS	postural orthostatic tachycardia syndrome
PPPD	persistent postural-perceptual dizziness
PPV	phobic postural vertigo
SMD	space-motion discomfort
VID	visually induced dizziness
VV	visual vertigo
VVM	visual-vestibular mismatch

1. Introduction

This paper introduces the diagnostic criteria for persistent postural-perceptual dizziness (PPPD), classified as a chronic functional vestibular disorder in the International Classification of Vestibular Disorders (ICVD) [8]. PPPD is a new term, but the core features of the disorder can be found in medical writings dating back to the 19th century [6, 21, 90]. After a brief review of this historical context, the contemporary background of PPPD is presented, followed by its diagnostic criteria with explanatory notes to guide their application. Then, the differential diagnosis is discussed in detail. Lastly, data suggesting possible pathophysiologic mechanisms are summarized.

1.1. Historical background

In the 1870 s, three German physicians described syndromes of dizziness and discomfort in motion rich environments, accompanied by autonomic arousal, anxiety, and avoidance of provocative circumstances [6, 21, 90]. Benedikt [6] emphasized a neuro-ophthalmologic process in *Platzschwindel* (vertigo in a plaza or square), whereas Cordes [21] focused on a psychological genesis in *Platzangst* (fear in a plaza or square). Westphal [47, 90] proposed that postural control, locomotion, conscious appraisal of spatial orientation, and threat assessment were "part of one process" in *Die Agoraphobie* (fear of the marketplace). Other European and American physicians added commentary [4, 48, 65], including observations that otologic diseases could precipitate Westphal's agoraphobia, especially in people with pre-existing anxiety [48], but differing views of these three syndromes and debates about whether they were predominantly neurologic or psychiatric in nature were never resolved. As otology, neurology, and psychiatry matured into separate specialties in the early 20th century, *Platzschwindel* and *Platzangst* faded from use and agoraphobia became a psychiatric disorder, losing its space and motion context [3, 95]. A century later small case series were published describing various syndromes of spatial disorientation and aberrant motion sensations, including supermarket syndrome [55], space phobia [53, 54], motorist's vestibular disorientation syndrome [60], visually induced motion symptoms [31], and physiologic height vertigo [11].

1.2. Contemporary context

Sustained investigations in larger numbers of patients began in the 1980 s. From clinical observations in their tertiary otoneurologic practice, Brandt and Dieterich [13] defined *Phobischer Attacken-Schwankschwindel* (phobic postural vertigo, PPV) in 1986 as a diagnosable clinical syndrome of postural dizziness and fluctuating unsteadiness accompanied by mild anxiety and depression in patients with obsessive compulsive personality traits. Other features of this diagnosis are listed in Table 1. Brandt, Dieterich, and their colleagues [12, 35, 36] showed that PPV was common, persistent, and distinct from other vestibular diseases and psychiatric disorders. They postulated that it arose from anxiety-related conscious awareness of discrepancies between anticipated and actual movements that occur transiently in the course of normal voluntary motion (i.e., efferent-afferent mismatch) [12], causing patients with PPV to adopt a stiffened postural control strategy [98].

Also starting in the mid-1980 s, Jacob and colleagues [38, 40, 41] conducted a series of investigations into potential links between anxiety symptoms, persistent dizziness, and vestibular dysfunction in patients from a tertiary anxiety disorders clinic. In 1989, they described [39] and subsequently validated [43] the symptom of *space-motion discomfort* (SMD) as a combination of uneasiness about spatial orientation and increased awareness of motion stimuli. Active or passive movement in visually-rich environments (e.g., walking down a

supermarket aisle, riding in a vehicle) and exposure to moving or patterned objects in the environment even when stationary (e.g., viewing passing traffic, striped curtains, or crowds of people) triggered this symptom in affected individuals. In their patients with anxiety disorders, Jacob and his coinvestigators [41] found an association between higher SMD and increased reliance on somatosensory information for controlling postural (i.e., somatosensory dependence). Features of SMD are listed in Table 1.

In 1995, Bronstein [15] described the symptom of *visual vertigo* (VV) in a portion of patients in his tertiary otoneurologic clinic. This symptom, which was identified in patients following acute peripheral or central vestibular losses, manifested with sensations of unsteadiness or dizziness on exposure to complex or moving visual stimuli. Visual vertigo often persisted despite patients seeming to recover from their acute vestibular deficits. The visual cues that triggered VV overlapped with the environmental stimuli that activated SMD [29, 63]. One hypothesis [50] suggested that VV was caused by an incongruity between visual and vestibular information following peripheral vestibular injury, a process termed visual-vestibular mismatch (VVM), but later work by Bronstein's group placed the onus on a combination of increased vigilance about vestibular symptoms and higher than normal reliance on visual cues for spatial orientation (i.e., visual dependence) [22, 23]. In 2009, the Bárány Society adopted the term visually induced dizziness or VID to replace VV in the ICVD nomenclature for vestibular symptoms [9]. Herein, the historical moniker VV is used to refer to Bronstein's original description and the subsequent body of research on that symptom. Features of VV are shown in Table 1.

Finally, in 2004, Staab and colleagues [81] described the clinical syndrome of *chronic subjective dizziness* (CSD) based on observations of patients in their tertiary balance center, and defined it more explicitly in 2007 [79]. This clinical diagnosis was similar in many ways to PPV, but focused primarily on physical not psychological symptoms.

Table 1
Features of PPV, SMD, VV, and CSD that informed the definition of PPPD

	PPV [13]	SMD [39]	VV [15]	CSD [79, 81]
Primary Symptoms (criteria A.1–3)				
Dizziness	✓✓	✓	✓✓ [22, 23]	✓✓
Unsteadiness	✓✓	✓✓	✓✓	✓✓
Non-spinning vertigo	✓✓	✓✓	✓✓	✓
Temporal profile (Criteria A.1–3)				
	Fluctuating with momentary flares	Situational (provoked)	Situational (provoked), Persistent [23]	Persistent with diurnal variability [27]
Provocative factors (Criteria B.1–3)				
Upright posture	✓✓			✓ [75]
Active or passive motion	✓	✓	✓	✓✓
Moving visual stimuli or complex patterns	✓	✓	✓✓	✓✓
Precipitants (Criterion C.1)				
Vestibular syndromes	✓	✓	✓	✓
Other medical illnesses	✓			✓
Psychological distress	✓	✓		✓
Course of illness (Criteria C.1.a-b)				
	Long-standing, waxing/waning [18]	May be long-standing	May be long-standing	Chronic
Physical exam and laboratory findings (Criterion E)				
	Normal	Somatosensory dependence on posturography [41]	Central or peripheral vestibular deficits	Abnormalities related to comorbid conditions [75]
Features not incorporated into PPPD				
Anxiety	Part of PPV	Associated with SMD [41]	Associated with prolonged VV [23]	May be comorbid with CSD [80]
Depression	Part of PPV			May be comorbid with CSD [80]
Personality traits	Obsessive-compulsive traits are part of PPV			Neurotic, introverted traits may be risk factors for CSD [76]

Included in the definition of CSD were persistent nonvertiginous dizziness or unsteadiness, heightened sensitivity to motion of self or objects in the environment, and difficulty performing tasks that required precise visual focus. Staab, et al. [74] found that this diagnostic definition was highly sensitive (>85%) and specific (>90%) for detecting CSD versus Menière's disease, vestibular migraine, or benign paroxysmal positional vertigo (BPPV), in patients with or without additional neuro-otologic comorbidity. The clinical features of CSD are listed in Table 1 to complete a side-by-side comparison of the four precursors that informed the diagnostic criteria of PPPD.

1.3. Current considerations for classifying vestibular diseases and disorders

The ICVD divides diseases and disorders based on duration of symptoms into acute, episodic, or chronic syndromes [8]. PPPD may last for months to years making it a chronic vestibular disorder. Vestibular diseases and disorders also are divided into structural, functional, and psychiatric conditions based on proven or presumed pathophysiologic mechanisms. Here functional conditions are considered as they were in the early 19th century as disorders "arising from a change in the mode of action of an organ" [20], unrelated to structural or cellular deficits. As revived in the modern era, most notably in gastroenterology [25], this concept of functional conditions distinguishes them from psychiatric illnesses. In this connotation, functional is not a synonym for psychogenic or psychosomatic as it was throughout most of the 20th century and, therefore, does not reflect a presumption of psychopathological abnormalities. Studies of PPV, VV, SMD, and CSD identified a number of functional alterations in vestibular and balance mechanisms [33, 37, 46, 59, 67, 68, 71, 88, 98] associated with these clinical entities. Additional investigations separated them from primary psychiatric disorders [13, 75] with which they may co-exist. These findings, reviewed in detail below, appear largely applicable to PPPD, indicating that it is a functional, not a structural or psychiatric, vestibular disorder.

2. Methods

In 2006, members of the Bárány Society created a working group to standardize nomenclature for vestibular diseases and disorders worldwide. This led to formation of the Committee for Classification Vestibular Disorders of the Bárány Society (CCBS) to oversee development of the first International Classification of Vestibular Disorders (ICVD) [8]. To date, this process has generated consensus documents defining vestibular symptoms [9], vestibular migraine [49], Menière's disease [51], BPPV [87], and vestibular paroxysmia [82]. Additional definitions are in the offing. In 2010, the CCBS chartered a Behavioral Subcommittee to identify primary and secondary psychiatric disorders that cause or amplify vestibular morbidity and review evidence about the nature of PPV, SMD, VV, and CSD. In keeping with established procedures for the classification process [5], the Behavioral Subcommittee included an otologist (A.H.), neurologist (M.S.), and members with special expertise in psychosomatic medicine (J.P.S., A.E.H.) and psychiatry (J.P.S., R.J.). Members hailed from three continents (Asia, Europe, and North America). Two senior neuro-otologists (T.B., A.B.) graciously agreed to advise subcommittee members on their deliberations.

The subcommittee met for the first time in August 2010 during the Bárány Society's biennial congress in Reykjavík, Iceland. From 2010–2012, the chair (J.P.S.) consulted with subcommittee members individually. These deliberations produced a consensus that PPV, SMD, VV, and CSD included a core set of physical symptoms that represented a distinctly definable vestibular disorder. Subcommittee members prepared a draft definition of this disorder that was updated iteratively after review by the general membership of the Bárány Society in June 2012 in Uppsala, Sweden, the CCBS in November 2013 in Mondorf-les-Bains, Luxembourg, and then again by the general membership in May 2014 in Buenos Aires, Argentina. Additional feedback was solicited from scientific societies dedicated to otorhinolaryngology, neurology, psychiatry, and psychosomatic medicine worldwide and from individual members of the Bárány Society via a posting of the draft definition to the ICVD development webpage of the *Journal of Vestibular Research.* The subcommittee used this feedback to prepare the final definition, which was approved by the CCBS.

The disorder was named persistent postural-perceptual dizziness to reflect its main diagnostic criteria of *persistent* non-vertiginous *dizziness*, unsteadiness, and non-spinning vertigo that are exacerbated by *postural* challenges and *perceptual* sensitivity to space-motion stimuli. A separate,

100-word narrative definition was prepared for the World Health Organization as part of the Bárány Society's recommendations for updates to the vestibular disorders section of the forthcoming 11th edition of the International Classification of Diseases (ICD-11) [97].

3. Criteria for the diagnosis of Persistent Postural-Perceptual Dizziness (PPPD)

PPPD is a chronic vestibular disorder defined by criteria A-E below. All five criteria must be fulfilled to make the diagnosis.

A. One or more symptoms of dizziness, unsteadiness, or non-spinning vertigo are present on most days for 3 months or more.[1–3]
   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: [4,5]
   1. Upright posture,
   2. Active or passive motion without regard to direction or position, and
   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.[6]
   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.[7]

Notes

(1) PPPD manifests with the following primary symptoms as defined previously by the CCBS [9]:
   - non-motion sensations of disturbed or impaired spatial orientation (dizziness)
   - feelings of being unstable while standing or walking (unsteadiness)
   - false or distorted sensations of swaying, rocking, bobbing, or bouncing of oneself (internal non-spinning vertigo) or similar sensations of movement of the surroundings (external non-spinning vertigo).

(2) Symptoms must be present for more than 15 of every 30 days. Most affected individuals experience symptoms every day or nearly every day. Symptoms tend to increase as the day progresses.

(3) Momentary flares of symptoms may occur spontaneously or with movement, but these transient flare-ups, lasting just seconds, are not present in all patients. Momentary flare-ups alone do not fulfill this criterion.

(4) Once the disorder is fully developed, symptoms persist without the need for ongoing exposure to precipitating conditions.

(5) The three exacerbating factors of criterion B must be discernable in the clinical history, although they do not have to be equally troublesome. Patients may try to avoid these factors to minimize noxious exacerbations of their vestibular symptoms. Such avoidance may be considered in fulfillment of this criterion.
   - Upright posture means standing or walking. Patients who are particularly sensitive to the effects of upright posture may report that sitting unsupported exacerbates their symptoms (See Section 4.1.3.1 for more details).
   - Active motion refers to a person's self-generated movements. Passive motion refers to a person being moved by conveyances or other beings (e.g., riding in a vehicle or elevator/lift, riding an animal, being jostled in a crowd) (See Section 4.1.3.2 for more details).
   - Visual stimuli may be large objects in the visual environment (e.g., passing traffic, busy patterns on floors or wall coverings, graphics displayed on large screens) or

smaller objects viewed at a close distance (e.g., books, computers, mobile electronic devices) (See Section 4.1.3.3 for more details).

(6) The most common precipitating conditions are peripheral or central vestibular disorders (25–30% of cases), attacks of vestibular migraine (15–20%), panic attacks or anxiety that manifest prominent dizziness (15% each), concussive injuries of the brain or whiplash injuries of the neck (10–15%), and autonomic disorders (7%). Other conditions that are capable of producing vertigo, unsteadiness or dizziness, or altering balance function (e.g., cardiac dysrhythmias, adverse drug reactions) precipitate the disorder less commonly (collectively ∼3%) [74, 79]. The majority of conditions that precede PPPD are acute or episodic in nature. Patients report the onset of chronic symptoms of PPPD following their acute illnesses. However, precipitants such as generalized anxiety disorder, autonomic disorders, and peripheral or central degenerative conditions may develop insidiously. In these cases, patients are less likely to report a distinct onset. It is not possible to identify a specific precipitant in every case. When a specific precipitant cannot be identified, particularly when symptoms slowly worsen, re-evaluation of the diagnosis is indicated and a period of prospective monitoring may be needed to confirm it.

(7) PPPD may co-exist with other diseases or disorders. Evidence of another active illness does not necessarily exclude a diagnosis of PPPD. Rather, clinical judgment must be exercised to determine the best attribution of the patient's vestibular symptoms to all identified illnesses [24, 75].

4. Comments

4.1. Further descriptions of diagnostic criteria

The diagnostic criteria of PPPD are explained in greater detail below, based on information derived from numerous reports on PPV, SMD, VV, and CSD. Table 1 lists the diagnostic criteria of PPPD and the characteristics of PPD, SMD, VV, and CSD that informed the definition. Citations given in the subsections below relate to the four precursors.

4.1.1. Core vestibular symptoms

The primary symptoms of PPPD are dizziness, unsteadiness, and certain types of non-spinning vertigo [13, 75]. The dizziness of PPPD is a non-motion symptom [9] that patients may describe variously as cloudiness, fuzziness, fullness, heaviness, or lightness in the head, or a feeling that their spatial orientation is not sharp or visual focus is not clear. Unsteadiness is a sensation of instability or wobbling when upright, or a feeling of veering from side to side when walking without a directional preponderance [9]. Non-spinning vertigo encompasses feelings of swaying, rocking, bouncing, or bobbing that patients may describe as motion inside their heads, involving their entire heads or bodies, or occurring in the environment. Tilting and sliding sensations are included in the Bárány Society's definition of non-spinning vertigo [9], but these are not typical symptoms of PPPD [12, 13, 24, 75]. Intermittent, momentary sensations of illusory movement that last no more than a few seconds (Note 3) may include both spinning and non-spinning vertigo [12, 13].

4.1.2. Temporal pattern of symptoms

PPV was defined by fluctuating postural symptoms and momentary illusions of movement [12, 13]. SMD was described as a situational phenomenon, occurring during exposures to provocative stimuli [39, 43]. The earliest reports of VV [15] also focused on situational symptoms, though recent investigations identified a link between persistent VV and chronic dizziness [22, 23]. Definitions of CSD [75, 79] emphasized chronic symptoms lasting throughout the day and exacerbated by motion stimuli. One study found that symptoms of CSD were absent or mildest for about an hour after patients awoke in the morning, but then increased as the day progressed [27]. These observations identified the two temporal patterns incorporated into PPPD. Patients experience a background of vestibular symptoms throughout the day, nearly every day [13, 75]. Symptom-free intervals tend to be brief (minutes to hours), though a distinct minority of patients may experience symptom-free periods lasting for days to weeks. Symptoms wax and wane spontaneously, but are aggravated by the three exacerbating factors of Criterion B. When free of these circumstances, patients' symptoms may be innocuous, limited to non-motion dizziness with minimal unsteadiness or non-spinning vertigo. However, all symptoms are susceptible to exacerbation with upright posture, motion, and exposure to complex

visual stimuli. On exposure to these situations, sensations of unsteadiness and non-spinning vertigo tend to dominate the clinical picture.

4.1.3. Exacerbating factors

The exacerbating factors included in Criterion B were described in all or most of the precursors of PPPD (Table 1). Upright posture, active or passive movement, and exposure to visually complex or moving stimuli aggravate the core symptoms of PPPD. There are no data to identify one of these factors as being consistently more troublesome than the others or to determine the sensitivities and specificities of requiring the presence of one, two, or all three factors to make a diagnosis of PPPD. Therefore, the consensus definition of Criterion B requires that affected individuals report at least some difficulty with all three factors, while recognizing that patients will not be equally sensitive to all of them, and that the most troublesome of these factors will vary from one patient with PPPD to another.

Symptoms may not increase immediately on standing, moving, or entering visually stimulating environments. Rather, they tend to build throughout continued exposure. Symptoms usually do not return to baseline immediately on cessation of exposures, but may last for hours or more thereafter. This pattern differs from that experienced by patients with structural vestibular deficits whose symptoms increase and decrease in close temporal relationship to motion. Patients with PPPD may express concerns about having to endure increases in their noxious symptoms on exposure to exacerbating factors, but this differs from individuals with anxiety disorders who focus more on fears of becoming incapacitated, injuring themselves or others, or attracting unwanted attention when exposed to situations that they fear.

4.1.3.1. Upright posture. Most patients with PPPD report more severe symptoms when standing or walking than when sitting or lying down [12, 13, 75]. Individuals who are particularly sensitive to postural changes may experience increased symptoms when sitting upright or leaning backward in a seat without back or arm support. Patients may not have a complete resolution of symptoms when lying down, but recumbent postures are the least troublesome. Patients may minimize the adverse effects of upright posture by touching fixed objects, using gait aids, or holding onto other people. Patients do not have to hold tightly to support their weight. Rather, a light touch is sufficient to obtain the stabilizing effect of somatosensory input.

4.1.3.2. Active or passive movement. Movements that exacerbate symptoms may occur in any direction or position. Most patients find that active and passive motions are troublesome in direct proportion to their intensity. Speed, duration, and repetition of movement appear to be more important than whether motion is actively or passively induced. High velocity movements that are prolonged or repeated are universally provocative whether encountered actively or passively. Responses to less intense movements vary among affected individuals. Most patients feel best when still, but others report that moving at a modest pace (e.g., walking or riding a bicycle) is more tolerable than remaining stationary when upright [24].

4.1.3.3. Visual stimuli. Visual stimuli that exacerbate symptoms may be moving or stationary. Environments that contain full field visual flow (e.g., passing traffic especially at high speed on a highway or motorway, large crowds of people milling about), large complex patterns (e.g., an expanse of busy carpeting, large store displays), or wide-open spaces with distant or indistinct visuospatial reference points (e.g., large fields, warehouses, atria) are most troublesome [15, 29, 39, 43, 50, 63, 75]. Exposure to full field visual stimuli may exacerbate symptoms for hours, even after brief exposures. Smaller visual targets (e.g., books, mobile electronic devices) may be troublesome when held at a close distance. Performing tasks that require precision visual focus (e.g., using a computer, or watching television) may exacerbate symptoms even when patients are sitting still [75, 79, 81]. The increasing need to view information on electronic screens in the modern world is a bane for many patients with PPPD.

4.1.4. Clinical course

In most cases, PPPD develops as the acute symptoms of precipitating conditions remit [24]. Patients do not experience symptom-free intervals. Instead, as their acute vertiginous symptoms fade, they develop the characteristic chronic symptoms of PPPD. In other cases, PPPD has a stuttering onset in which patients may experience PPPD-like symptoms lasting days to weeks, until recurrences settle into a persistent course. This stuttering course is more likely when precipitants are short-lived, recurrent events (e.g.,

attacks of BPPV, migraine, or panic). Least often, PPPD has a gradual onset. Precipitants such as generalized anxiety, autonomic disorders, and degenerative diseases of the peripheral vestibular apparatus or cerebellum are chronic conditions that may develop slowly. With these precipitants, symptoms of PPPD may appear gradually, almost imperceptibly at first, and then worsen slowly.

Specific precipitants of PPPD cannot be identified in all patients, especially those who have been symptomatic for many years and lack adequate documentation of their initial clinical presentations or cannot recall the details of their earliest symptoms. Even in these situations, however, most patients describe an acute, subacute, or stuttering onset of illness. Patients with clinical histories of gradually worsening chronic vestibular symptoms or balance problems that have no identifiable starting points, particularly those without generalized anxiety or autonomic problems, are less likely to have PPPD than those with more definitive onsets of illness. They warrant prospective observation over a period of several months to verify the diagnostic picture and properly screen for other diseases, especially slowly emerging degenerative disorders.

4.2. Possible variants or subtypes of PPPD

Commonalities among PPV, SMD, VV, CSD, and the syndromes that preceded them in the 1870 s formed the basis of the diagnostic criteria for PPPD. However, semiologic similarities, even those stretching over more than a century, do not necessarily mean that PPPD is a single entity. The current state of scientific knowledge left two important questions unanswered: (a) Is PPPD a single disorder with one principal pathophysiologic process or is it the common manifestation of multiple conditions that produce similar symptoms from different pathophysiologic mechanisms? (b) If PPPD is a single disorder, does it have clinically meaningful and validly distinguishable subtypes?

4.2.1. A single disorder or a group of related conditions?

The precipitating factors of PPV [13], SMD [39], VV [15], and CSD [79] span a variety of neuro-otologic, other medical, and psychological conditions. It is not known if these act through one pathophysiologic process to produce a single syndrome or via separate mechanisms that share enough of a final common pathway to generate symptoms and susceptibilities to exacerbating factors that closely resemble one another. The former situation would be akin to posttraumatic stress disorder in which a wide variety of traumatic experiences precipitate one syndrome with four clusters of symptoms that can be exacerbated by internal and external factors [3, 95]. The latter circumstance would be like hypertension in which chronically elevated blood pressure can arise from multiple diseases that produce sustained increases in intravascular pressure via different physiologic mechanisms.

4.2.2. Do subtypes exist?

Although PPV, SMD, VV, and CSD share a number of features, they also have different areas of emphasis (Table 1). Postural provocation is a distinguishing feature of PPV [12, 13]. It was not part of the original description of CSD [81], but was added later [75]. Difficulty with self-motion is part of PPV [12, 13], SMD [39], and CSD [75, 81]. Trouble with visual motion stimuli is the primary feature of VV [15, 29, 63] and is emphasized in SMD [39, 41, 43] and CSD [75, 81]. Thus, PPV, SMD, VV, and CSD may reflect different perspectives on a single, multifaceted clinical entity or they may offer insights into potentially distinguishable subtypes of PPPD (e.g., posturally predominant subtype, visually predominant subtype). Mild anxiety and depressive symptoms and phobic behaviors were included in the descriptions of PPV [12, 13], but were considered comorbidities of CSD [24, 75, 80]. This raises the possibility that PPV may be either a distinct phobic subtype of PPPD or encompass PPPD plus a specific phobia of dizziness-related experiences.

In the absence of definitive scientific data on these possible subtypes, the Behavioral Subcommittee chose to define only PPPD for the ICVD, but allow for clinical variability as reflected in Criterion B. The World Health Organization permits related terms to be listed in the ICD-11. Therefore, PPV, SMD, VV, and CSD were retained as index terms for cross-referencing in the ICD-11 beta draft definition of PPPD [97].

4.2.3. Probable (subthreshold) PPPD

Members of the subcommittee concluded that there were not enough published data to define a clinically meaningful probable or subthreshold version of PPPD. Clinical experience with PPV and CSD suggests caution in applying the diagnosis of PPPD to

patients who do not fulfill all five of its diagnostic criteria.

4.3. Making a diagnosis of PPPD

The diagnosis of PPPD is made by gathering clinical history relevant to Criteria A-D. There are no findings on physical examination, laboratory testing, or diagnostic imaging that are pathognomonic of PPPD. Data from physical examinations and clinically indicated diagnostic testing help to determine if PPPD is the best diagnosis, either alone or in combination with other diseases or disorders (Criterion E). In this regard, an abnormal finding on physical examination or laboratory testing does not exclude a diagnosis of PPPD. Rather, when all criteria for PPPD are met, positive findings on examination or testing indicate the ongoing presence of a precipitating condition or another co-existing illness. Furthermore, PPPD is not a diagnosis of exclusion [12, 24, 75]. It should not be given to patients who report only non-specific chronic vestibular symptoms or those who have enigmatic complaints that do not fulfill its definition. In such cases, prospective monitoring may provide the clinical evidence needed to verify or exclude the diagnosis.

4.4. Differential diagnosis

The differential diagnosis of PPPD includes chronic sequelae of acute precipitants, recurrent attacks of episodic precipitants, ongoing manifestations of chronic precipitants, other chronic vestibular syndromes, medical or psychiatric disorders that produce persistent unsteadiness or dizziness, and adverse effects of regularly consumed prescription or non-prescription medications [13, 15, 24, 79], as discussed in turn below.

4.4.1. Chronic sequelae of acute precipitants

Some precipitants of PPPD are acute disorders that have the potential for chronic symptomatic complications (e.g., vestibular neuritis or stroke leading to persistent uncompensated vestibulopathies). For these disorders, the diagnostic question is whether patients' presenting symptoms are due to PPPD alone, chronic manifestations of its precipitants, or both [24]. This diagnostic dilemma is resolved by careful attention to the clinical history and assessment of patients' compensation status. A history of persistent non-vertiginous dizziness and unsteadiness provoked by upright posture, patients' own movements, and exposure to visual motion stimuli plus physical exam and laboratory evidence of good compensation (e.g., no spontaneous nystagmus or abnormal responses to head thrust, headshake, or stepping tests) indicates that PPPD is the only active diagnosis. In contrast, the presence of ongoing episodes of head motion-provoked vertigo or unsteadiness without persistent dizziness and exam findings of incomplete compensation argues against PPPD. A third possibility is the combination of persistent dizziness and motion sensitivity plus head motion-provoked symptoms and exam findings of incomplete compensation, which would indicate coexisting PPPD and uncompensated vestibulopathy.

4.4.2. Recurrent attacks of episodic precipitants

PPPD may be preceded by episodic vestibular disorders such as vestibular migraine [49], Menière's disease [51], and BPPV [87] that cause distinct bouts of vestibular symptoms in contrast to the persistent, waxing and waning dizziness, unsteadiness and non-spinning vertigo that are hallmarks of PPPD. When PPPD co-exists with these disorders, proper diagnosis rest on identifying the characteristic symptoms of each active disorder. Episodic disorders add distinctive vestibular symptoms to the background of PPPD [58], such acute attacks of vertigo plus cephalalgia, photophobia and phonophobia, with or without visual aura for vestibular migraine [49], short-lived positional vertigo for BPPV [87], or attacks of vertigo, tinnitus, and fluctuating hearing for Menière's disease [51].

4.4.3. Ongoing manifestations of chronic precipitants

Some precipitants of PPPD are chronic conditions themselves (e.g., anxiety and depressive disorders, post-concussive syndrome, autonomic disorders, and heart diseases). They may cause persistent unsteadiness or dizziness with or without precipitating PPPD. When these disorders are present alone, patients are not as greatly affected by the motion provocations of Criterion B as when PPPD exists. The strategy for differential diagnosis in these cases is to determine if Criteria A-D for PPPD are present and evaluate key elements of clinical histories, physical examinations, and laboratory testing to determine which conditions explain the patients' symptoms best (i.e., PPPD alone, precipitating events alone, or both) (Criterion E).

4.4.3.1. Chronic anxiety and depressive disorders. Chronic anxiety due to generalized anxiety disorder,

agoraphobia, social phobia, obsessive compulsive disorders, and traumatic stress disorders may manifest with persistent dizziness [3, 95]. Depressive disorders also may cause dizziness [57]. These disorders are diagnosed according to the latest versions of the International Classification of Diseases [95] or Diagnostic and Statistical Manual of Mental Disorders [3]. However, in neurologic, otologic, and primary care settings where patients with vestibular symptoms are most commonly encountered, simple self-report questionnaires offer a valid and efficient means of detecting psychiatric morbidity. The 7-item Generalized Anxiety Disorders Scale (GAD-7) may be used to screen for pathological anxiety [73]. The 9-item Patient Health Questionnaire (PHQ-9) may be used to screen for depression [72]. The 14-item Hospital Anxiety and Depression Scale (HADS) covers both anxiety and depressive symptoms [102]. Positive results indicate that an anxiety or depressive disorder is likely, either as the sole cause of vestibular symptoms or co-existing with PPPD [12, 58, 75, 79]. As described in Note 5, patients may avoid circumstances that exacerbate PPPD because of the noxious nature of persistent vestibular symptoms. This may be considered a maladaptive means of managing the disorder and does not, in and of itself, merit another diagnosis. However, moderate to severe avoidance that is associated with ruminative worries about making the condition worse, becoming incapacitated, or drawing unwanted attention to oneself may be manifestations of a phobic disorder if they repeatedly disrupt desired activities. The diagnosis of PPPD alone, a phobic disorder alone (e.g., agoraphobia, social phobia, specific phobia of dizziness) or both depends on the extent to which patients' symptoms fulfill criteria A-D for PPPD and criteria for the phobic disorder [3, 95].

A history of troubling life circumstances or recent stressful events cannot be used as evidence for or against the presence of functional or psychiatric diagnoses, including PPPD [24]. A recent study found that childhood and adulthood adversity were equally prevalent in patients with structural versus functional or psychiatric causes of vestibular symptoms [69]. Furthermore, panic disorder and generalized anxiety disorder frequently occur in the absence of identifiable stressors [3, 95]. Therefore, a diagnosis of PPPD rests on fulfillment of its diagnostic criteria, regardless of patients' histories of adversity.

4.4.3.2. Postconcussive syndrome. Patients with postconcussive syndrome following a traumatic brain injury or whiplash often experience chronic dizziness in addition to headache, insomnia, cognitive symptoms, and mood lability [32]. Patients who fulfill all of the diagnostic criteria for PPPD after a traumatic brain injury or whiplash should receive the diagnosis. The presence or absence of other sequelae of injury will determine if additional diagnoses are warranted. On the other hand, patients who complain of chronic dizziness after head injury should not be given a diagnosis of PPPD if they do not manifest Criteria A-D.

4.4.3.3. Autonomic disorders. Autonomic disorders frequently cause dizziness. The autonomic disorders most likely to precipitate PPPD, based on research from CSD [77], are postural orthostatic tachycardia syndrome (POTS) and type 1 neurocardiogenic (vasovagal) syncope, which are most often encountered in adolescents and young adults. Orthostatic intolerance with or without hypotension from neurologic and cardiovascular illnesses (e.g., autonomic neuropathy) is more common in older adults, in whom it may be part of multi-factorial dizziness. Patients with POTS, neurocardiogenic syncope, and other autonomic disorders tend to have more pronounced orthostatic and exertional dizziness than those with PPPD [77]. However, symptoms overlap considerably, so the differential diagnosis depends on examination of autonomic integrity. PPPD does not cause abnormal changes in heart rate or blood pressure. Autonomic disorders do not create difficulties with complex or moving visual stimuli in patients who are sitting still. Thus, vital signs during autonomic challenges and sensitivity to visual stimuli when seated at rest best distinguish PPPD from autonomic disorders, recognizing the potential for the two problems to co-exist.

4.4.4. Other chronic vestibular syndromes

Chronic vestibular syndromes in the differential diagnosis of PPPD include bilateral vestibulopathy [45], neurodegenerative disorders (e.g., downbeat nystagmus syndrome and other cerebellar diseases) [61, 89], and mal de debarquement syndrome (MdDS) [19].

4.4.4.1. Bilateral vestibulopathy. Bilateral vestibulopathy is best distinguished from PPPD by its characteristic findings on physical examination and laboratory testing [45], such as bilaterally positive head thrusts and diminished responses on head impulse testing, caloric irrigation, or sinusoidal stimulation in a rotary chair. Clinical history may offer

additional clues, but these are not as definitive. For example, PPPD does not cause oscillopsia, but oscillopsia is present in only 30–40% of patients with bilateral vestibulopathy. Individuals with PPPD are susceptible to symptom exacerbations when exposed to complex visual stimuli even when sitting still, whereas patients with bilateral vestibulopathy have minimal symptoms when seated and stationary.

4.4.4.2. Chronic neurologic disorders. Neurodegenerative disorders that affect posture and gait such as Parkinson's disease, cerebellar degeneration [61], including the downbeat nystagmus syndrome [89], and small vessel white matter disease [2, 16, 66] may manifest with dizziness or unsteadiness when standing or walking before motor signs can be detected on physical examination. The gradual onset of these complaints in the relative absence of difficulties with complex or moving visual stimuli should arouse suspicion that PPPD is not the correct diagnosis. The best approach in this situation is a period of prospective monitoring (typically 6–12 months) with a symptom log kept by the patient and serial examinations performed by the clinician before making a definitive diagnosis.

Bilateral peripheral neuropathy and orthostatic tremor [16, 99, 101] may cause or contribute to dizziness and unsteadiness when patients are upright, though neither one causes trouble with complex or moving visual stimuli. Peripheral neuropathy is much more likely to manifest with sensory loss and pain, or present as one part of a multi-factorial picture, than to be the sole cause of dizziness. Orthostatic tremor typically causes discomfort when standing still (e.g., standing in line at a supermarket). Symptoms are relieved by walking or sitting down. Orthostatic tremor is diagnosed by identifying its characteristic 13–18 Hz tremor in the lower legs on electromyographic or posturographic testing [16, 99, 101].

4.4.4.3. Mal de debarquement syndrome. Mal de debarquement syndrome (MdDS) [19] is a condition of persistent unsteadiness triggered by traveling on boats, aircraft, or automobiles, usually for at least a few hours. Symptoms characteristically decrease during passive motion (e.g., riding in a car) and then increase again when motion ceases. That is opposite the pattern found in most patients with PPPD, though a minority of individuals with PPPD experience temporary decreases in symptoms during modest motion, such as walking at a medium pace or riding a bicycle on a smooth path. A "spontaneous onset" version of MdDS has been described, though most patients in those reports had migraine or anxiety disorders [19], which are known precipitants of PPPD. A major difference between MdDS and PPPD is the extent of treatment response. MdDS generally has a limited response to vestibular habituation exercises or medications, whereas treatment studies of PPV [86], SMD [42, 92], VV [62, 64], CSD [81], and PPPD, itself [84], showed significant improvements with vestibular habituation or serotonergic antidepressants.

4.4.5. Adverse effects of medications

Prescription medications, over the counter preparations, and dietary supplements may cause dizziness, unsteadiness, or vertigo. Vestibular symptoms caused by newly administered medications or changed doses of existing medications may precipitate PPPD.

4.4.6. Other functional forms of vestibular symptoms

Clinicians may encounter patients who describe persistent vestibular symptoms that do not fit the diagnostic criteria of either PPPD or other well-defined chronic vestibular syndromes [24]. Examples include constant, invariant vertigo, unsteadiness, or dizziness, complex body motions in multiple directions simultaneously, and kaleidoscopic swirling movements of large portions of the visual field. Patients with these complaints often report a lack of provoking or mitigating factors. These clinical presentations have not been studied systematically, but their continuous nature, unwavering intensity, and abnormal complexity distinguish them from the episodic or fluctuating symptoms reported by patients with structural deficits, PPPD, and anxiety or depressive disorders that cause vestibular symptoms [24]. In many patients, these functional forms of vestibular symptoms are accompanied by other chronic physical complaints such as fatigue and pain, raising the possibility that they are but one manifestation of a broader somatic symptom disorder [3] or bodily distress disorder [96].

4.4.7. Gait disorders, falls, and near falls

Patients with PPPD may report sensations of veering from side-to-side when walking. On exam, they may exhibit a mildly slow or cautious gait. One study of walking mechanics found that patients with PPV walked slower, had reduced stride length, and spent a greater fraction of time with both feet on the ground than healthy subjects [71]. These changes correlated with reduced balance confidence. A case study dis-

tinguished gait and posture symptoms of CSD from those of functional gait disorders [34]. Falls and near falls have never been a part of PPV or CSD [24]. Therefore, clinical evidence of significant changes in gait or recurrent falls or near falls indicates the presence of a structural or functional gait disorder. PPPD may co-exist with these disorders.

4.5. Epidemiology

No epidemiologic studies are available for PPPD, but its prevalence and incidence may be estimated from research done on patients with PPV, VV, CSD, and chronic dizziness following acute vestibular syndromes [7, 12, 23, 28, 30, 79].

4.5.1. Estimates of the prevalence of PPPD

Clinical epidemiologic data from tertiary care centers with special interest in PPV [13] and CSD [79] showed their prevalence to be 15–20% among all patients presenting for evaluation of vestibular symptoms, making them the most common diagnoses among young adults and the second most common among all adults, trailing only BPPV. The average duration of illness at the time of tertiary consultation was 4.5 years with some patients experiencing symptoms for decades [13, 79]. Disability varied widely from individuals who had few limitations in daily functioning to those who were severely impaired and unable to work. The average age of patients presenting for evaluation of PPV, CSD, and PPPD is the mid-40 s, with a range from adolescence to late adulthood [10, 79, 100]. A female predominance has been reported in the first clinical reports on PPPD [10, 100].

4.5.2. Estimates of the incidence of PPPD

The incidence of PPPD following neuro-otologic precipitants may be estimated from studies that followed patients prospectively after bouts of acute or episodic vestibular disorders (e.g., vestibular neuritis, BPPV, vestibular migraine, Menière's disease) [7, 23, 28, 30]. These investigations found PPPD-like chronic dizziness [7, 28, 30] or persistent VV [23] in about 25% of patients after 3–12 months of follow-up, despite otherwise adequate compensation or recovery from the initial illnesses. These results indicate that PPPD is likely to develop in a significant proportion of patients afflicted with acute or episodic vestibular syndromes. Similar prospective studies of clinical outcomes following other medical and psychological precipitants of PPPD have not been conducted. However, retrospective investigations found that the course [80] and treatment response [78] of patients with CSD precipitated by anxiety disorders mirrored that of patients with CSD precipitated by acute vestibular conditions, suggesting that the clinical course of PPPD may be similar regardless of precipitant. A long-term follow-up study of patients with PPV found that only a minority experienced spontaneous resolution of symptoms [36]. Most had a chronic waxing and waning course and three-quarters developed anxiety or depressive comorbidity. Thus, the majority of patients with PPPD are likely to remain symptomatic without treatment, regardless of initial precipitant.

The incidence and prevalence of PPPD in primary care practices and the general population are not known as detailed epidemiologic studies of PPV, SMD, VV, and CSD have not been conducted in those settings.

4.6. Possible pathophysiologic processes underlying PPPD

Investigators studying PPV, CSD, SMD, and VV have identified pathophysiologic processes that may be applicable to PPPD, including anxiety-related personality traits as a possible risk factor [86, 88, 89] and high levels of anxiety and vigilance about acute symptoms during precipitating events as initial pathologic responses [7, 23, 28, 30]. Alterations in postural control strategies [33, 46, 59, 67, 68, 71], shifts in multi-sensory integration [23], and reduced cortical integration of spatial orientation and threat assessment networks [37, 88] may be sustaining mechanisms. All of these will have to be studied in greater detail in patients meeting the specific diagnostic criteria for PPPD.

4.6.1. Possible risk factors

In their original paper, Brandt and Dieterich made the clinical observation that patients with PPV were likely to have obsessive compulsive personality traits [13]. Subsequent studies using formal psychometric measures found that individuals with the anxiety-related personality traits of neuroticism and introversion [76] had an increased risk for CSD. High neuroticism also was identified in a report on PPPD [100]. In contrast, persons demonstrating resilience, optimism, and beliefs that life is meaningful and manageable had a reduced risk of persistent dizziness after acute vestibular events [85]. Patients with family or personal histories of anxiety disorders pre-dating the

onset of vestibular symptoms had an increased risk of developing persistent dizziness [7] or CSD [80] after precipitating events. In patients with anxiety disorders, a history of previous vestibular deficits was associated with SMD [38, 41]. These studies suggest that anxiety-related personality traits or a personal or family history of anxiety disorders may be risk factors for developing PPPD following relevant precipitants.

4.6.2. Initial reactions

Three prospective studies found that high anxiety about dizziness during and after bouts of acute vestibular neuritis or BPPV predicted continued dizziness three [30], six [23], and twelve [28] months later. These initial psychological responses had far greater effects on long-term outcomes than the initial or subsequent states of patients' peripheral vestibular functioning or vestibulo-ocular reflexes. Furthermore, patients with emerging symptoms of CSD who were treated with three sessions of cognitive behavioral therapy started within 8 weeks of precipitating events had marked reductions in dizziness and avoidance of provocative circumstances [26], benefits that endured at follow-up six months later [52]. Collectively, these data raise the possibility that a highly anxious response to precipitating events may be the pivotal initial pathophysiologic process in the development of PPPD, and that early symptom-specific interventions might counter this effect.

4.6.3. Possible alterations in postural control

Several investigations showed that patients with PPV manifested an alteration in postural control characterized by high frequency, low amplitude postural sway related to co-contraction of lower leg muscles when standing at rest [46, 98]. One study of patients with CSD demonstrated similar results [59]. Investigations conducted in normal, healthy people found that they used this high demand postural control strategy only in challenging balance situations such as standing at heights [1, 17, 18]. Patients with PPV adopted this strategy during less demanding tasks than normal individuals [33, 67, 71], possibly related to a lower threshold for engaging closed loop feedback mechanisms to adjust posture [98]. Brandt et al. [14] reported the case of a patient who was followed prospectively from a bout of acute vestibular neuritis to the development of PPV. The transition to chronic symptoms coincided with emergence of the high frequency, low amplitude sway pattern of PPV. Future studies will have to measure the prevalence of this postural control strategy among patients with PPPD and determine its association with clinical characteristics of the disorder, particularly postural symptoms.

4.6.4. Possible association with visual dependence

Bronstein and colleagues [15, 29, 63] showed that people with VV manifested visual dependence, a trait-like tendency to rely on visual information for spatial orientation. In a prospective study, Cousins et al., [23] found that patients who had persistent dizziness for at least six months following bouts of acute vestibular neuritis had greater visual dependence than those who recovered without chronic symptoms. Redfern et al. [70] found that patients with anxiety disorders and SMD had greater body sway in response to moving visual surrounds than patients with anxiety disorders but no SMD and also than normal controls. Future studies will have to measure the prevalence and severity of visual dependence in patients with PPPD and determine its association with clinical features of the disorder, particularly visual symptoms.

4.6.5. Possible changes in activity and connectivity of crucial brain regions

The first neuroimaging studies of patients with CSD [37] and PPPD [88] were completed recently. The first study [37] measured the activity and connectivity of vestibular, visual, and anxiety-related regions of the brain using functional magnetic resonance imaging (fMRI) in response to sound-evoked vestibular stimulation in patients with CSD versus normal control subjects matched for anxiety-related personality traits. Patients with CSD showed reduced stimulus-related activity in the parieto-insular vestibular cortex (PIVC), anterior insula, inferior frontal gyrus, hippocampus, and anterior cingulate cortex compared to normal individuals. They also had more negative connectivity between the PIVC and the anterior insula, anterior cingulate cortex, and hippocampus, as well as between the anterior insula and middle occipital cortex. The second study [88] compared women with PPPD to women who had recovered without sequelae from illnesses that caused acute vestibular symptoms (i.e., typical precipitants of PPPD). Using fMRI, the authors found that women with PPPD had less activation of the anterior cingulate cortex, which was related to state anxiety, and less activation of the precuneus, which remained after controlling for psychological variables, in response to a non-motion stimulus of standardized pictures

designed to elicit negative emotions. These early results suggest that brain areas responsible for high level spatial orientation, multi-sensory integration, and threat assessment may not be as active or well connected in patients with PPPD as in normal people, potentially leaving lower level posture and gaze control mechanisms poorly integrated with one another. These findings await confirmation in larger studies with sufficient power to control for potential confounds and measure associations with clinical aspects of PPPD.

5. Additional commentary

The consensus process for decision-making seeks a result that has broad support. It does not require unanimity, but demands more than a simple majority [83]. Were the latter sufficient for developing diagnostic criteria or writing treatment guidelines, then informative aspects of the minority opinion could be lost to practicing clinicians, the scientific community, and general public. Though not all agree [44], arguments for publishing minority opinions in such endeavors have been made [56].

Members of the Behavioral Subcommittee and their advisors reached consensus about the diagnostic criteria for PPPD and its relationship to SMD, VV, and CSD, but not PPV. SMD and VV were seen as complex symptoms, not stand alone diagnostic entities. They informed criterion B of PPPD, but are known to occur in other situations as well [15, 39]. The definition of PPPD paralleled work in other areas of medicine where functional disorders (e.g., the functional gastrointestinal disorders [25, 91] and fibromyalgia [93, 94]) were defined by their core physical symptoms and not associated psychological features. CSD was a step in that direction based in part on the physical features of PPV [79]. However, the definition of PPPD is better supported; hence, PPPD now fully supplants CSD.

Members of the subcommittee and their advisors carefully considered a proposal to include PPV as a subtype of PPPD. Arguments in favor of this idea focused on the richness of the definition of PPV, which includes personality factors and psychological symptoms, particularly phobic symptoms, that were shown to affect clinical presentation [12, 71], as well as 30 years of research that described its clinical course [12, 35, 36] and identified potential pathophysiologic mechanisms [33, 46, 67, 68, 71, 98]. Arguments against the idea included concerns that the mix of physical and psychological symptoms and personality traits that constitute PPV is difficult to operationalize in clinical practice [75] and may have to be updated based on newer research [76, 85], and that including PPV as a subtype of PPPD without validating their relationship could perplex clinicians and investigators. In the end, a simple majority of subcommittee members voted against including PPV as a subtype of PPPD.

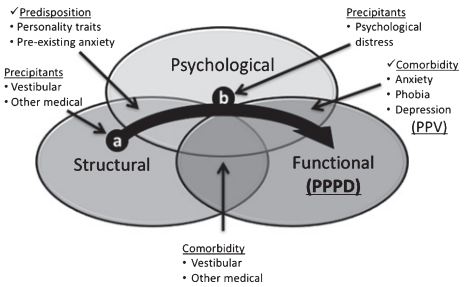

Fig. 1. Putative mechanisms of PPPD. PPPD is thought to develop via a dynamic process (arrow). In about 70% of patients, a structural vestibular syndrome (e.g., vestibular neuritis, BPPV) or other medical condition precipitates PPPD (black dot, a) [79]. Individuals who respond to the precipitating event with a high level of anxiety and body vigilance appear likely to progress to PPPD (i.e., to traverse the arrow from the initial structural event through this transient psychological stage to the chronic functional disorder) [23, 28, 30]. Anxiety-related personality traits or pre-existing anxiety disorders appear to increase the risk of developing PPPD [7, 76, 80]. In about 30% of patients, PPPD begins with acute psychological distress (black dot, b) and then progresses to the functional disorder [79]. PPPD may co-exist with structural or psychological illnesses [24], placing patients in the intersections of the functional and psychological or structural ellipses. Anxiety-related personality traits and psychological symptoms (check marks, ✓) are incorporated into PPV [12, 13], whereas they are considered predisposing factors and comorbid symptoms, respectively, in PPPD. PPPD = Persistent postural-perceptual dizziness. PPV = Phobic postural vertigo.

The debate about PPV highlighted the fact that PPPD is a dynamic condition as illustrated in Fig. 1. It is a functional disorder, although structural and psychological factors affect its development. Clinicians who apply Criteria A and B as simple checklists of symptoms without capturing the dynamic features of their patients' histories (Criterion C) assume a reductionist view of the diagnosis, potentially missing important aspects of patients' morbidity (Criterion D) and nuances of the differential diagnosis (Criterion E). Scientists who fail to consider the dynamics of PPPD risk muddling rather than illuminating the nature of the disorder and its pathophysiologic mechanisms.

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References

[1] A.L. Adkin, J.S. Frank, M.G. Carpenter and G.W. Peysar, Postural control is scaled to level of postural threat, *Gait Posture* **12** (2000), 87–93.

[2] H. Ahmad, N. Cerchiai, M. Mancuso, A.P. Casani and A.M. Bronstein, Are white matter abnormalities associated with "unexplained dizziness"? *J Neurol Sci* **15** (2015), 428–431.

[3] American Psychiatric Association, The Diagnostic and Statistical Manual of Mental Disorders, 5th Edition (DSM-5), American Psychiatric Association: Washington, DC, 2013.

[4] C.D. Balaban and R.G. Jacob, Background and history of the interface between anxiety and vertigo, *J Anxiety Disorders* **15** (2001), 27–51.

[5] Bárány Society initiative for the establishment of the International Classification of Vestibular Disorders (ICVD), http://www.jvr-web.org/images/Instructions for ICVD subcommittees_as_of_19Oct2014.pdf. Accessed 07 July 2017.

[6] M. Benedikt, Über "Platzschwindel," Allgemeine *Wien Medizin Zeitschrift* **15** (1870), 488–490.

[7] C. Best, R. Tschan, A. Eckhardt-Henn and M. Dieterich, Who is at risk for ongoing dizziness and psychological strain after a vestibular disorder? *Neuroscience* **164** (2009), 1579–1587.

[8] A.R. Bisdorff, J.P. Staab and D.E. Newman-Toker, Overview of the International Classification of Vestibular Disorders, *Neurol Clin* **33** (2015), 541–550.

[9] A. Bisdorff, M. von Brevern, T. Lempert and D.E. Newman-Toker, Classification of vestibular symptoms: Towards an international classification of vestibular disorders, *J Vestib Res* **19** (2009), 1–13.

[10] R.S.M. Bittar and E.M.D. von Söhsten Lins, Clinical characteristics of patients with persistent postural-perceptual dizziness, *Braz J Otorhinolaryngology* **81** (2015), 276–282.

[11] W. Bles, T.S. Kapteyn, T. Brandt and F. Arnold, The mechanism of physiological height vertigo. II. Posturography, *Acta Otolaryngol* **89** (1980), 534–540.

[12] T. Brandt, Phobic postural vertigo, *Neurology* **46** (1996), 1515–1519.

[13] T. Brandt and M. Dieterich, Phobischer Attacken Schwankschwindel, ein neues Syndrom? *Munch Med Wschr* **28** (1986), 247–250.

[14] T. Brandt, M. Strupp, S. Novozhilov and S. Krafczyk, Artificial neural network posturography detects the transition of vestibular neuritis to phobic postural vertigo, *J Neurol* **259** (2012), 182–184.

[15] A.M. Bronstein, Visual vertigo syndrome: Clinical and posturography findings, *J Neurol Neurosurg Psychiatry* **59** (1995), 472–476.

[16] A.M. Bronstein and M. Guerraz, Visual-vestibular control of posture and gait: Physiological mechanisms and disorders, *Curr Opin Neurol* **12** (1999), 5–11.

[17] L.A. Brown and J.S. Frank, Postural compensations to the potential consequences of instability: Kinematics, *Gait Posture* **6** (1997), 89–97.

[18] M.G. Carpenter, J.S. Frank and C.P. Silcher, Surface height effects on postural control: A hypothesis for a stiffness strategy for stance, *J Vestib Res* **9** (1999), 277–286.

[19] Y.H. Cha, Mal de debarquement, *Semin Neurol* **29** (2009), 520–527.

[20] A. Combe, Observations on mental derangement: Being an application of the principles of phrenology to the elucidation of the causes, symptoms, nature, and treatment of insanity, John Anderson: Edinburgh, 1831.

[21] E. Cordes, Die Platzangst (Agoraphobie), Symptom einer Erschöpfungsparese, Zeitschrift für Psychiatrie, *Berlin* **3** (1872), 521–574.

[22] S. Cousins, N.J. Cutfield, D. Kaski, A. Palla, B.M. Seemungal, J.F. Golding, J.P. Staab and A.M. Bronstein, Visual dependency and dizziness after vestibular neuritis, *PLoS One* **9** (2014), e105426.

[23] S. Cousins, D. Kaski, N. Cutfield, Q. Arshad, H. Ahmad, M.A. Gresty, B.M. Seemungal, J. Golding and A.M. Bronstein, Predictors of clinical recovery from vestibular neuritis: A prospective study, *Ann Clin Transl Neurol* **4** (2017), 340–346.

[24] M. Dieterich, J.P. Staab and T. Brandt, Functional (psychogenic) dizziness, *Handb Clin Neurol* **139** (2016), 447–468.

[25] D.A. Drossman (Ed), Rome III: The Functional Gastrointestinal Disorders, 3rd edition. Degnon Associates, Inc., McLean, VA (2006).

[26] S. Edelman, A.E. Mahoney and P.D. Cremer, Cognitive behavior therapy for chronic subjective dizziness: A randomized, controlled trial, *Am J Otolaryngol* **33** (2012), 395–401.

[27] R. Feuerecker, M. Habs, M. Dieterich and M. Strupp, Chronic subjective dizziness: Fewer symptoms in the early morning – a comparison with bilateral vestibulopathy and downbeat nystagmus syndrome, *J Vestib Res* **25** (2015), 67–72.

[28] F. Godemann, K. Siefert, M. Hantschke-Bruggemann, P. Neu, R. Seidl and A. Ströhle, What accounts for vertigo one year after neuritis vestibularis—anxiety or a dysfunctional vestibular organ? *J Psychiatr Res* **39** (2005), 529–534.

[29] M. Guerraz, L. Yardley, P. Bertholon, L. Pollak, P. Rudge, M.A. Gresty and A.M. Bronstein, Visual vertigo: Symptom assessment, spatial orientation and postural control, *Brain* **124** (2001), 1646–1656.

[30] N. Heinrichs, C. Edler, S. Eskens, M.M. Mielczarek and C. Moschner, Predicting continued dizziness after an acute

peripheral vestibular disorder, *Psychosom Med* **69** (2007), 700–707.
[31] R.A. Hoffman and K.H. Brookler, Underrated neurotological symptoms, *Laryngoscope* **88** (1978), 1127–1138.
[32] L. Holm, J.D. Cassidy, L.J. Carroll, J. Borg and the Neurotrauma Task Force on Mild Traumatic Brain Injury of the WHO Collaborating Centre, Summary of the WHO Collaborating Centre for Neurotrauma Task Force on Mild Traumatic Brain Injury, *J Rehabil Med* **37** (2005), 137–141.
[33] J. Holmberg, F. Tjernström, M. Karlberg, P.A. Fransson and M. Magnusson, Reduced postural differences between phobic postural vertigo patients and healthy subjects during a postural threat, *J Neurol* **256** (2009), 1258–1262.
[34] J.A. Honaker, J.M. Gilbert and J.P. Staab, Chronic subjective dizziness versus conversion disorder: Discussion of clinical findings and rehabilitation, *Am J Audiol* **19** (2010), 3–8.
[35] D. Huppert, T. Kunihiro and T. Brandt T, Phobic postural vertigo (154 patients): Its association with vestibular disorders, *J Audiol* **4** (1995), 97–103.
[36] D. Huppert, M. Strupp, N. Rettinger, J. Hecht and T. Brandt, Phobic postural vertigo – a long-term follow-up (5 to 15 years) of 106 patients, *J Neurol* **252** (2005), 564–569.
[37] I. Indovina, R. Riccelli, G. Chiarella, C. Petrolo, A. Augimeri, L. Giofré, F. Lacquaniti, J.P. Staab and L. Passamonti, Role of the insula and vestibular system in patients with chronic subjective dizziness: An fMRI study using sound-evoked vestibular stimulation, *Front Behav Neurosci* **9** (2015), 334. doi: 10.3389/fnbeh.2015.00334
[38] R.G. Jacob, J.M. Furman, J.D. Durrant and S.M. Turner, Panic, agoraphobia, and vestibular dysfunction, *Am J Psychiatry* **153** (1996), 503–512.
[39] R.G. Jacob, S.O. Lilienfeld, J.M.R. Furman, J.D. Durrant and S.M. Turner, Panic disorder with vestibular dysfunction: Further clinical observation and description of space and motion phobic stimuli, *J Anxiety Disorders* **3** (1989), 117–130.
[40] R.G. Jacob, M.B. Møller, S.M. Turner and C Wall 3rd, Otoneurological examination in panic disorder and agoraphobia with panic attacks: A pilot study, *Am J Psychiatry* **142** (1985), 715–720.
[41] R.G. Jacob, M.S. Redfern and J.M. Furman, Space and motion discomfort and abnormal balance control in patients with anxiety disorders, *J Neurol Neurosurg Psychiatry* **80** (2009), 74–78.
[42] R.G. Jacob, S.L. Whitney, G. Detweiler-Shostak and J.M. Furman, Vestibular rehabilitation for patients with agoraphobia and vestibular dysfunction: A pilot study, *J Anxiety Disord* **15** (2001), 131–146.
[43] R.G. Jacob, S.R. Woody, D.B. Clark, S.O. Lilienfield, B.E. Hirsch, G.D. Kucera, J.M. Furman and J.D. Durrant, Discomfort with space and motion: A possible marker of vestibular dysfunction assessed by the Situational Characteristics Questionnaire, *J Psychopathol Behav Assess* **15** (1993), 299–324.
[44] R.M. Jacobson, Clinical practice guidelines and recommendations: Room for dissent? *Mayo Clinic Proceedings* **91** (2016), 551–553.
[45] M. Strupp, J.-S. Kim, T. Murofushi, D. Straumann, J. C. Jen, S. M. Rosengran, C. C. Della Santina and H. Kingma, Bilateral vestibulopathy: Diagnostic criteria - Consensus document of the Classification Committee of the Bárány Society, *J Vestib Res* **27** (2017), 177–189.
[46] S. Krafczyk, S. Tietze, W. Swoboda, P. Valkovič and T. Brandt, Artificial neural network: A new diagnostic posturographic tool for disorders of stance, *Clin Neurophysiol* **117** (2006), 1692–1698.
[47] K. Kuch and R.P. Swinson, Agoraphobia: What Westphal really said, *Can J Psychiatry* **37** (1992), 133–136.
[48] M. Lannois and C. Tournier, Les lésions auriculaires sont une cause déterminante fréquente de l'agoraphobie, Annales des Maladies de l'Oreille, du Larynx, *du Nez et du Pharynx* **24** (1898), 286–301.
[49] T. Lempert, J. Olesen, J. Furman, J. Waterston, B. Seemungal, J. Carey, A. Bisdorff, M. Versino, S. Eversi and D. Newman-Toker, Vestibular migraine: Diagnostic criteria. Consensus document of the Bárány Society and the International Headache Society, *J Vestib Res* **22** (2012), 167–172.
[50] N.S. Longridge, A.I. Mallinson and A. Denton A, Visual vestibular mismatch in patients treated with intratympanic gentamicin for Meniere's disease, *J Otolaryngol* **31** (2002), 5–8.
[51] J. A. Lopez-Escamez, J. Carey, W.-H. Chung, J.A. Goebel, M. Magnusson, M. Mandala, D.E. Newman-Toker, M. Strupp, M. Suzuki, F. Trabalzini and A. Bisdorff, Diagnostic criteria for Meniére's Disease. Consensus document of the Bárány Society, The Japan Society for Equilibrium Research, the European Academy of Otology and Neurotology (EAONO), the American Academy of Otolaryngology-Head and Neck Surgery (AAO-HNS) and the Korean Balance Society, *J Vestib Res* **25** (2015), 1–7.
[52] A.E.J. Mahoney, S. Edelman and P.D. Cremer, Cognitive behavior therapy for chronic subjective dizziness: Longer-term gains and predictors of disability, *Am J Otolaryngol* **34** (2013), 115–120.
[53] I. Marks, Space "phobia": A pseudo-agoraphobic syndrome, *J Neurol Neurosurg Psychiatry* **44** (1981), 387–391.
[54] I.M. Marks and P. Bebbington, Space phobia: Syndrome or agoraphobic variant? *Br Med J* **2** (1976), 345–347.
[55] B.F. McCabe, Diseases of the end organ and vestibular nerve. In: R.F. Naunton, ed. The Vestibular System, Academic Press: New York, 1975, 299–302.
[56] D.M. Musher, Should committees that write guidelines and recommendations publish dissenting opinions? *Mayo Clinic Proceedings* **91** (2016), 634–639.
[57] M. Nakao and E. Yano, Prediction of major depression in Japanese adults: Somatic manifestation of depression in annual health examinations, *J Affective Disord* **90** (2006), 29–35.
[58] B.A. Neff, J.P. Staab, S.D. Eggers, M.L. Carlson, W.R. Schmitt, K.M. Van Abel, D.K. Worthington, C.W. Beatty, C.L. Driscoll and N.T. Shepard, Auditory and vestibular symptoms and chronic subjective dizziness in patients with Menière's disease, vestibular migraine, and Menière's disease with concomitant vestibular migraine, *Otol Neurotol* **33** (2012), 1235–1244.
[59] M. Ödman and R. Maire, Chronic subjective dizziness, *Acta Otolaryngologica* **128** (2008), 1085–1088.
[60] N.G. Page and M.A. Gresty, Motorist's vestibular disorientation syndrome, *J Neurol Neurosurg Psychiatry* **48** (1985), 729–735.
[61] M. Pandolfo, M. Manto, Cerebellar and afferent ataxias, *Continuum: Lifelong Learning in Neurology (Minneap Minn)* **19** (2013), 1312–1343.
[62] M. Pavlou, A.M. Bronstein and R.A. Davies, Randomized trial of supervised versus unsupervised optokinetic

exercise in persons with peripheral vestibular disorders, *Neurorehabil Neural Repair* **27** (2013), 208–218.
[63] M. Pavlou, R.A. Davies and A.M. Bronstein, The assessment of increased sensitivity to visual stimuli in patients with chronic dizziness, *J Vestib Res* **16** (2006), 223–231.
[64] M. Pavlou, R.G. Kanegaonkar, D. Swapp, D.E. Bamiou, M. Slater and L.M. Luxon, The effect of virtual reality on visual vertigo symptoms in patients with peripheral vestibular dysfunction: A pilot study, *J Vestib Res* **22** (2012), 273–281.
[65] M. Perroud, Note sur l'agoraphobie, *Annales de la Societe de Medecine de Lyon* **21** (1873), 82–93.
[66] D. Pinter, S.J. Ritchie, F. Doubal, T. Gattringer, Z. Morris, M.E. Bastin, M. Del C Valdés Hernández, N.A. Royle, J. Corley, S. Muñoz Maniega, A. Pattie, D.A. Dickie, J. Staals, A.J. Gow, J.M. Starr, I.J. Deary, C. Enzinger, F. Fazekas and J. Wardlaw, Impact of small vessel disease in the brain on gait and balance, *Sci Rep* **7** (2017), 41637.
[67] V. Querner, S. Krafczyk, M. Dieterich and T. Brandt, Patients with somatoform phobic postural vertigo: The more difficult the balance task, the better the balance performance, *Neurosci Lett* **285** (2000), 21–24.
[68] V. Querner, S. Krafczyk, M. Dieterich and T. Brandt, Phobic postural vertigo. Body sway during visually induced roll vection, *Exp Brain Res* **143** (2002), 269–275.
[69] K. Radziej, G. Schmid, A. Dinkel, A. Zwergal and C. Lahmann, Psychological traumatization and adverse life events in patients with organic and functional vestibular symptoms, *J Psychosom Res* **79** (2015), 123–129.
[70] M.S. Redfern, J.M. Furman and R.G. Jacob, Visually induced postural sway in anxiety disorders, *J Anxiety Dis* **21** (2007), 704–716.
[71] R. Schniepp, M. Wuehr, S. Huth, C. Pradhan, T. Brandt and K. Jahn, Gait characteristics of patients with phobic postural vertigo: Effects of fear of falling, attention, and visual input, *J Neurol* **261** (2014), 738–746.
[72] R.L. Spitzer, K. Kroenke and J.B.W. Williams, Validation and utility of a self-report version of PRIME- MD - The PHQ primary care study, *JAMA* **282** (1999), 1737–1744
[73] R.L. Spitzer, K. Kroenke, J.B.W. Williams and B. Lowe, A brief measure for assessing generalized anxiety disorder - The GAD-7, *Arch Intern Med* **166** (2006), 1092–1097.
[74] J. Staab, S. Eggers, B. Neff and N. Shepard, Validation of a clinical syndrome of persistent dizziness and unsteadiness. Abstracts from the XXVI Bárány Society Meeting, Reykjavik, Iceland, August 18-21, 2010, *J Vest Res* **20** (2010), 172–173.
[75] J.P. Staab, Chronic subjective dizziness, *Continuum: Lifelong Learning in Neurology (Minneap Minn)* **18** (2012), 1118–1141.
[76] J.P. Staab, D.E. Rohe, S.D. Eggers and N.T. Shepard, Anxious, introverted personality traits in patients with chronic subjective dizziness, *J Psychosom Res* **76** (2014), 80–83.
[77] J.P. Staab and M.J. Ruckenstein, Autonomic nervous system function in chronic dizziness, *Otol Neurotol* **28** (2007), 854–859.
[78] J.P. Staab and M.J. Ruckenstein, Chronic dizziness and anxiety: Effect of course of illness on treatment outcome, *Arch Otolaryngol Head Neck Surg* **131** (2005), 675–679.
[79] J.P. Staab and M.J. Ruckenstein, Expanding the differential diagnosis of dizziness, *Arch Otolaryngol Head Neck Surg* **133** (2007), 170–176.
[80] J.P. Staab and M.J. Ruckenstein, Which comes first? Psychogenic dizziness versus otogenic anxiety, *Laryngoscope* **113** (2003), 1714–1718.
[81] J.P. Staab, M.J. Ruckenstein and J.D. Amsterdam, A prospective trial of sertraline for chronic subjective dizziness, *Laryngoscope* **114** (2004), 1637–1641.
[82] M. Strupp, J.A. Lopez-Escamez, J. Kim, D. Straumann, J. Jen, J. Carey, A. Bisdorff and T. Brandt, Vestibular paroxysmia: Diagnostic criteria. Consensus document of the Committee for the Classification of Vestibular Disorders of the Bárány Society, *J Vestib Res* **26** (2016), 409–415.
[83] The MIT-Harvard Public Disputes Program, A short guide to consensus building, http://web.mit.edu/publicdisputes/practice/cbh_ch1.html, accessed 10 June 2016.
[84] K.J. Thompson, J.C. Goetting, J.P. Staab and N.T. Shepard, Retrospective review and telephone follow-up to evaluate a physical therapy protocol for treating persistent postural-perceptual dizziness: A pilot study, *J Vestib Res* **25** (2015), 97–104.
[85] R. Tschan, C. Best, M.E. Beutel, A. Knebel, J. Wiltink, M. Dieterich and A. Eckhardt-Henn, Patients' psychological well-being and resilient coping protect from secondary somatoform vertigo and dizziness (SVD) 1 year after vestibular disease, *J Neurol* **258** (2011), 104–112.
[86] R. Tschan, A. Eckhardt-Henn, V. Scheurich, C. Best, M. Dieterich and M. Beutel, Steadfast-effectiveness of a cognitive behavioral self-management program of patients with somatoform vertigo and dizziness, *Psychother Psychosom Med Psychol* **62** (2012), 111–119.
[87] M. von Brevern, P. Bertholon, T. Brandt, T. Fife, T. Imai, D. Nuti and D. Newman-Toker, Benign paroxysmal positional vertigo: Diagnostic criteria. Consensus document of the Committee for the Classification of Vestibular Disorders of the Bárány Society, *J Vestib Res* **25** (2015), 105–117.
[88] E.M. von Söhsten Lins, Functional resonance in women with persistent postural-perceptual dizziness, Doctor of Philosophy dissertation, Department of Otorhinolaryngology, University of São Paulo, São Paulo, Brazil, 2015.
[89] J.N. Wagner, M. Glaser, T. Brandt and M. Strupp, Downbeat nystagmus: Aetiology and comorbidity in 117 patients, *J Neurol Neurosurg Psychiatry* **79** (2008), 672–677.
[90] C. Westphal, Die Agoraphobie, eine neuropathische Erscheinung, Zeitschrift für Psychiatrie, *Berlin* **3** (1871), 138–161.
[91] W.E. Whitehead, O. Palsson, K.R. Jones, Systematic review of the comorbidity of irritable bowel syndrome with other disorders: What are the causes and implications? *Gastroenterology* **122** (2002), 1140–1156.
[92] S.L. Whitney, R.G. Jacob, P.J. Sparto, E.F. Olshansky, G. Detweiler-Shostak, E.L. Brown and J.M. Furman, Acrophobia and pathological height vertigo: Indications for vestibular physical therapy? *Phys Ther* **85** (2005), 443–458.
[93] F. Wolfe, D.J. Clauw, M.-A. Fitzcharles, D.L. Goldenberg, R.S. Katz, P. Mease, A.S. Russell, I.J. Russell, J.B. Winfield and M.B. Yunus, The American College of Rheumatology preliminary diagnostic criteria for fibromyalgia and measurement of symptom severity, *Arthritis Care Res* **62** (2010), 600–610.
[94] F. Wolfe, H.A. Smythe, M.B. Yunus, R.M. Bennett, C. Bombardier, D.L. Goldenberg, P. Tugwell, S.M. Campbell, M. Abeles, P. Clark, A.G. Fam, S.J. Farber, J.J.

Fiechtner, C.M. Franklin, R.A. Gatter, D. Hamaty, J. Lessard, A.S. Lichtbroun, A.T. Masi, G.A. McCain, W.J. Reynolds, T.J. Romano, I.J. Russell and R.P. Sheon, The American College of Rheumatology 1990 criteria for the classification of fibromyalgia. Report of the multicenter criteria committee, *Arthritis Rheum* **33** (1990), 160–172.

[95] World Health Organization, International Classification of Diseases, 10th edition, http://apps.who.int/classifications/icd10/browse/2015, Accessed 20 June 2015.

[96] World Health Organization, International Classification of Diseases, 11th edition beta draft version (ICD-11 beta), definition of bodily distress disorder, http://id.who.int/icd/entity/767044268. Accessed 20 June 2015.

[97] World Health Organization, International Classification of Diseases, 11th edition beta draft version (ICD-11 beta), definition of persistent postural-perceptual dizziness, http://id.who.int/icd/entity/2005792829. Accessed 20 June 2015.

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

[99] T.C. Yaltho and W.G. Ondo, Orthostatic tremor: A review of 45 cases, *Parkinsonism & Related Disorders* **20** (2014), 723–725.

[100] Z. Yan, L. Cui, T. Yu, H. Liang, Y. Wang and C. Chen C, Analysis of the characteristics of persistent postural-perceptual dizziness: A clinical-based study in China, *Int J Audiol* **56** (2016), 1–5. [Epub ahead of print]

[101] K. Yarrow, P. Brown, M.A. Gresty and A. Bronstein, Force platform recordings in the diagnosis of primary orthostatic tremor, *Gait Posture* **13** (2001), 26–34.

[102] A.S. Zigmond and R.P. Snaith, The Hospital Anxiety and Depression Scale, *Acta Psychiatr Scand* **67** (1983), 361–370.