

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

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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

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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 débarquement 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 co-investigators [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]. Longridge and Mallison

[50] proposed that VV was caused by an incongruity between visual and vestibular information following peripheral vestibular injury, a process that they 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 Reykjavik, 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

451 visual stimuli. On exposure to these situations, sensa-
452 tions of unsteadiness and non-spinning vertigo tend
453 to dominate the clinical picture.

454 4.1.3. Exacerbating factors

455 The exacerbating factors included in Criterion B
456 were described in all or most of the precursors of
457 PPPD (Table 1). Upright posture, active or passive
458 movement, and exposure to visually complex or mov-
459 ing stimuli aggravate the core symptoms of PPPD.
460 There are no data to identify one of these factors
461 as being consistently more troublesome than the
462 others or to determine the sensitivities and speci-
463 ficities of requiring the presence of one, two, or
464 all three factors to make a diagnosis of PPPD.
465 Therefore, the consensus definition of Criterion B
466 requires that affected individuals report at least some
467 difficulty with all three factors, while recognizing
468 that patients will not be equally sensitive to all
469 of them, and that the most troublesome of these
470 factors will vary from one patient with PPPD to
471 another.

472 Symptoms may not increase immediately on
473 standing, moving, or entering visually stimulating
474 environments. Rather, they tend to build through-
475 out continued exposure. Symptoms usually do not
476 return to baseline immediately on cessation of expo-
477 sures, but may last for hours or more thereafter.
478 This pattern differs from that experienced by patients
479 with structural vestibular deficits whose symptoms
480 increase and decrease in close temporal relationship
481 to motion. Patients with PPPD may express concerns
482 about having to endure increases in their noxious
483 symptoms on exposure to exacerbating factors, but
484 this differs from individuals with anxiety disorders
485 who focus more on fears of becoming incapacitated,
486 injuring themselves or others, or attracting unwanted
487 attention when exposed to situations that they
488 fear.

489 4.1.3.1. Upright posture. Most patients with PPPD
490 report more severe symptoms when standing or walk-
491 ing than when sitting or lying down [12, 13, 75].
492 Individuals who are particularly sensitive to postural
493 changes may experience increased symptoms when
494 sitting upright or leaning backward in a seat without
495 back or arm support. Patients may not have a complete
496 resolution of symptoms when lying down, but recum-
497 bent postures are the least troublesome. Patients may
498 minimize the adverse effects of upright posture by
499 touching fixed objects, using gait aids, or holding
500 onto other people. Patients do not have to hold tightly

501 to support their weight. Rather, a light touch is suffi-
502 cient to obtain the stabilizing effect of somatosensory
503 input.

504 4.1.3.2. Active or passive movement. Movements
505 that exacerbate symptoms may occur in any direc-
506 tion or position. Most patients find that active and
507 passive motions are troublesome in direct proportion
508 to their intensity. Speed, duration, and repetition of
509 movement appear to be more important than whether
510 motion is actively or passively induced. High velocity
511 movements that are prolonged or repeated are uni-
512 versally provocative whether encountered actively or
513 passively. Responses to less intense movements vary
514 among affected individuals. Most patients feel best
515 when still, but others report that moving at a modest
516 pace (e.g., walking or riding a bicycle) is more toler-
517 able than remaining stationary when upright [24].

518 4.1.3.3. Visual stimuli. Visual stimuli that exac-
519 erbate symptoms may be moving or stationary.
520 Environments that contain full field visual flow (e.g.,
521 passing traffic especially at high speed on a high-
522 way or motorway, large crowds of people milling
523 about), large complex patterns (e.g., an expanse of
524 busy carpeting, large store displays), or wide-open
525 spaces with distant or indistinct visuospatial refer-
526 ence points (e.g., large fields, warehouses, atria)
527 are most troublesome [15, 29, 39, 43, 50, 63, 75].
528 Exposure to full field visual stimuli may exacer-
529 bate symptoms for hours, even after brief exposures.
530 Smaller visual targets (e.g., books, mobile electronic
531 devices) may be troublesome when held at a close dis-
532 tance. Performing tasks that require precision visual
533 focus (e.g., using a computer, or watching televi-
534 sion) may exacerbate symptoms even when patients
535 are sitting still [75, 79, 81]. The increasing need
536 to view information on electronic screens in the
537 modern world is a bane for many patients with
538 PPPD.

539 4.1.4. Clinical course

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

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

558 Specific precipitants of PPPD cannot be identi-
 559 fied in all patients, especially those who have been
 560 symptomatic for many years and lack adequate doc-
 561 umentation of their initial clinical presentations or
 562 cannot recall the details of their earliest symptoms.
 563 Even in these situations, however, most patients
 564 describe an acute, subacute, or stuttering onset
 565 of illness. Patients with clinical histories of grad-
 566 ually worsening chronic vestibular symptoms or
 567 balance problems that have no identifiable start-
 568 ing points, particularly those without generalized
 569 anxiety or autonomic problems, are less likely to
 570 have PPPD than those with more definitive onsets
 571 of illness. They warrant prospective observation
 572 over a period of several months to verify the
 573 diagnostic picture and properly screen for other
 574 diseases, especially slowly emerging degenerative
 575 disorders.

576 4.2. Possible variants or subtypes of PPPD

577 Commonalities among PPV, SMD, VV, CSD, and
 578 the syndromes that preceded them in the 1870s
 579 formed the basis of the diagnostic criteria for PPPD.
 580 However, semiologic similarities, even those stretch-
 581 ing over more than a century, do not necessarily
 582 mean that PPPD is a single entity. The current state
 583 of scientific knowledge left two important ques-
 584 tions unanswered: (a) Is PPPD a single disorder
 585 with one principal pathophysiologic process or is
 586 it the common manifestation of multiple condi-
 587 tions that produce similar symptoms from different
 588 pathophysiologic mechanisms? (b) If PPPD is a single
 589 disorder, does it have clinically meaningful and
 590 validly distinguishable subtypes?

591 4.2.1. A single disorder or a group of related 592 conditions?

593 The precipitating factors of PPV [13], SMD
 594 [39], VV [15], and CSD [79] span a variety of
 595 neuro-otologic, other medical, and psychological
 596 conditions. It is not known if these act through
 597 one pathophysiologic process to produce a single
 598 syndrome or via separate mechanisms that share

599 enough of a final common pathway to generate
 600 symptoms and susceptibilities to exacerbating fac-
 601 tors that closely resemble one another. The former
 602 situation would be akin to posttraumatic stress
 603 disorder in which a wide variety of traumatic
 604 experiences precipitate one syndrome with four
 605 clusters of symptoms that can be exacerbated by
 606 internal and external factors [3, 95]. The latter cir-
 607 cumstance would be like hypertension in which
 608 chronically elevated blood pressure can arise from
 609 multiple diseases that produce sustained increases
 610 in intravascular pressure via different physiologic
 611 mechanisms.

612 4.2.2. Do subtypes exist?

613 Although PPV, SMD, VV, and CSD share a num-
 614 ber of features, they also have different areas of
 615 emphasis (Table 1). Postural provocation is a dis-
 616 tinguishing feature of PPV [12, 13]. It was not part
 617 of the original description of CSD [81], but was
 618 added later [75]. Difficulty with self-motion is part
 619 of PPV [12, 13], SMD [39], and CSD [75, 81]. Trou-
 620 ble with visual motion stimuli is the primary feature
 621 of VV [15, 29, 63] and is emphasized in SMD [39,
 622 41, 43] and CSD [75, 81]. Thus, PPV, SMD, VV,
 623 and CSD may reflect different perspectives on a sin-
 624 gle, multifaceted clinical entity or they may offer
 625 insights into potentially distinguishable subtypes of
 626 PPPD (e.g., posturally predominant subtype, visually
 627 predominant subtype). Mild anxiety and depressive
 628 symptoms and phobic behaviors were included in the
 629 descriptions of PPV [12, 13], but were considered
 630 comorbidities of CSD [24, 75, 80]. This raises the
 631 possibility that PPV may be either a distinct phobic
 632 subtype of PPPD or encompass PPPD plus a specific
 633 phobia of dizziness-related experiences.

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

643 4.2.3. Probable (subthreshold) PPPD

644 Members of the subcommittee concluded that there
 645 were not enough published data to define a clini-
 646 cally meaningful probable or subthreshold version of
 647 PPPD. Clinical experience with PPV and CSD sug-
 648 gests caution in applying the diagnosis of PPPD to

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

651 4.3. Making a diagnosis of PPPD

652 The diagnosis of PPPD is made by gathering clinical
653 history relevant to Criteria A-D. There are no
654 findings on physical examination, laboratory testing,
655 or diagnostic imaging that are pathognomonic
656 of PPPD. Data from physical examinations and clinically
657 indicated diagnostic testing help to determine
658 if PPPD is the best diagnosis, either alone or in combination
659 with other diseases or disorders (Criterion E). In this regard,
660 an abnormal finding on physical examination or laboratory
661 testing does not exclude a diagnosis of PPPD. Rather, when
662 all criteria for PPPD are met, positive findings on examination
663 or testing indicate the ongoing presence of a precipitating
664 condition or another co-existing illness. Furthermore,
665 PPPD is not a diagnosis of exclusion [12, 24, 75].
666 It should not be given to patients who report only
667 non-specific chronic vestibular symptoms or those
668 who have enigmatic complaints that do not fulfill
669 its definition. In such cases, prospective monitoring
670 may provide the clinical evidence needed to verify or
671 exclude the diagnosis.
672

673 4.4. Differential diagnosis

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

683 4.4.1. Chronic sequelae of acute precipitants

684 Some precipitants of PPPD are acute disorders
685 that have the potential for chronic symptomatic
686 complications (e.g., vestibular neuritis or stroke leading
687 to persistent uncompensated vestibulopathies). For
688 these disorders, the diagnostic question is whether
689 patients' presenting symptoms are due to PPPD
690 alone, chronic manifestations of its precipitants, or
691 both [24]. This diagnostic dilemma is resolved by
692 careful attention to the clinical history and assessment
693 of patients' compensation status. A history of
694 persistent non-vertiginous dizziness and unsteadiness
695 provoked by upright posture, patients' own

696 movements, and exposure to visual motion stimuli
697 plus physical exam and laboratory evidence of
698 good compensation (e.g., no spontaneous nystagmus
699 or abnormal responses to head thrust, headshake,
700 or stepping tests) indicates that PPPD is the only
701 active diagnosis. In contrast, the presence of ongoing
702 episodes of head motion-provoked vertigo or
703 unsteadiness without persistent dizziness and exam
704 findings of incomplete compensation argues against
705 PPPD. A third possibility is the combination of persistent
706 dizziness and motion sensitivity plus head motion-provoked
707 symptoms and exam findings of incomplete compensation,
708 which would indicate coexisting PPPD and uncompensated
709 vestibulopathy.

710 4.4.2. Recurrent attacks of episodic precipitants

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

727 4.4.3. Ongoing manifestations of chronic precipitants

728 Some precipitants of PPPD are chronic conditions
729 themselves (e.g., anxiety and depressive disorders,
730 post-concussive syndrome, autonomic disorders, and
731 heart diseases). They may cause persistent unsteadiness
732 or dizziness with or without precipitating PPPD.
733 When these disorders are present alone, patients are
734 not as greatly affected by the motion provocations of
735 Criterion B as when PPPD exists. The strategy for
736 differential diagnosis in these cases is to determine if
737 Criteria A-D for PPPD are present and evaluate key
738 elements of clinical histories, physical examinations,
739 and laboratory testing to determine which conditions
740 explain the patients' symptoms best (i.e., PPPD alone,
741 precipitating events alone, or both) (Criterion E).
742

743 4.4.3.1. Chronic anxiety and depressive disorders.
744 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 peripheral vestibulopathy [45], neurodegenerative disorders (e.g., downbeat nystagmus syndrome and other cerebellar diseases) [61, 89], and mal de débarquement syndrome (MdDS) [19].

4.4.4.1. Bilateral peripheral vestibulopathy. Bilateral peripheral 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 to caloric stimulation. Clinical history may offer additional clues, but these are not as definitive.

845 For example, PPPD does not cause oscillopsia, but
846 oscillopsia is present in only 30–40% of patients with
847 bilateral peripheral vestibulopathy. Individuals with
848 bilateral vestibulopathy typically have less trouble
849 with complex visual stimuli when sitting still than
850 patients with PPPD, but this symptom varies among
851 patients with both disorders.

852 *4.4.4.2. Chronic neurologic disorders.* Neurode-
853 generative disorders that affect posture and gait such
854 as Parkinson's disease, cerebellar degeneration [61],
855 including the downbeat nystagmus syndrome [89],
856 and small vessel white matter disease [16, 66] may
857 manifest with dizziness or unsteadiness when stand-
858 ing or walking before motor signs can be detected
859 on physical examination. The gradual onset of these
860 complaints in the relative absence of difficulties with
861 complex or moving visual stimuli should arouse sus-
862 picion that PPPD is not the correct diagnosis. The best
863 approach in this situation is a period of prospective
864 monitoring (typically 6–12 months) with a symptom
865 log kept by the patient and serial examinations per-
866 formed by the clinician before making a definitive
867 diagnosis.

868 Bilateral peripheral neuropathy and orthostatic
869 tremor [16, 99, 101] may cause or contribute to
870 dizziness and unsteadiness when patients are upright,
871 though neither one causes trouble with complex or
872 moving visual stimuli. Peripheral neuropathy is much
873 more likely to manifest with sensory loss and pain, or
874 present as one part of a multi-factorial picture, than
875 to be the sole cause of dizziness. Orthostatic tremor
876 typically causes discomfort when standing still (e.g.,
877 standing in line at a supermarket). Symptoms are
878 relieved by walking or sitting down. Orthostatic
879 tremor is diagnosed by identifying its characteristic
880 13–18 Hz tremor in the lower legs on electromyo-
881 graphic or posturographic testing [16, 99, 101].

882 *4.4.4.3. Mal de débarquement syndrome.* Mal de
883 débarquement syndrome (MdDS) [19] is a condition
884 of persistent unsteadiness triggered by traveling on
885 boats, aircraft, or automobiles, usually for at least
886 a few hours. Symptoms characteristically decrease
887 during passive motion (e.g., riding in a car) and then
888 increase again when motion ceases. That is opposite
889 the pattern found in most patients with PPPD, though
890 a minority of individuals with PPPD experience tem-
891 porary decreases in symptoms during modest motion,
892 such as walking at a medium pace or riding a bicycle
893 on a smooth path. A "spontaneous onset" version of
894 MdDS has been described, though most patients in

895 those reports had migraine or anxiety disorders [19],
896 which are known precipitants of PPPD. A major dif-
897 ference between MdDS and PPPD is the extent of
898 treatment response. MdDS generally has a limited
899 response to vestibular habituation exercises or medi-
900 cations, whereas treatment studies of PPV [86], SMD
901 [42, 92], VV [62, 64], CSD [81], and PPPD, itself
902 [84], showed significant improvements with vestibular
903 habituation or serotonergic antidepressants.

904 *4.4.5. Adverse effects of medications*

905 Prescription medications, over the counter prepa-
906 rations, and dietary supplements may cause dizziness,
907 unsteadiness, or vertigo. Vestibular symptoms caused
908 by newly administered medications or changed doses
909 of existing medications may precipitate PPPD.

910 *4.4.6. Other functional forms of vestibular 911 symptoms*

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

934 *4.4.7. Gait disorders, falls, and near falls*

935 Patients with PPPD may report sensations of veer-
936 ing from side-to-side when walking. On exam, they
937 may exhibit a mildly slow or cautious gait. One study
938 of walking mechanics found that patients with PPV
939 walked slower, had reduced stride length, and spent a
940 greater fraction of time with both feet on the ground
941 than healthy subjects [71]. These changes correlated
942 with reduced balance confidence. A case study dis-
943 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

1141 results suggest that brain areas responsible for high
 1142 level spatial orientation, multi-sensory integration,
 1143 and threat assessment may not be as active or well
 1144 connected in patients with PPPD as in normal people,
 1145 potentially leaving lower level posture and gaze control
 1146 mechanisms poorly integrated with one another.
 1147 These findings await confirmation in larger studies
 1148 with sufficient power to control for potential confounds
 1149 and measure associations with clinical aspects
 1150 of PPPD.

1151 5. Additional commentary

1152 The consensus process for decision-making seeks
 1153 a result that has broad support. It does not
 1154 require unanimity, but demands more than a simple
 1155 majority [83]. Were the latter sufficient for
 1156 developing diagnostic criteria or writing treatment
 1157 guidelines, then informative aspects of the
 1158 minority opinion could be lost to practicing clinicians,
 1159 the scientific community, and general public.
 1160 Though not all agree [44], arguments for publishing
 1161 minority opinions in such endeavors have been
 1162 made [56].

1163 Members of the Behavioral Subcommittee and
 1164 their advisors reached consensus about the diagnostic
 1165 criteria for PPPD and its relationship to SMD,
 1166 VV, and CSD, but not PPV. SMD and VV were
 1167 seen as complex symptoms, not stand alone diagnostic
 1168 entities. They informed criterion B of PPPD,
 1169 but are known to occur in other situations as well
 1170 [15, 39]. The definition of PPPD paralleled work in
 1171 other areas of medicine where functional disorders
 1172 (e.g., the functional gastrointestinal disorders [25, 91]
 1173 and fibromyalgia [93, 94]) were defined by their core
 1174 physical symptoms and not associated psychological
 1175 features. CSD was a step in that direction based in part
 1176 on the physical features of PPV [79]. However, the
 1177 definition of PPPD is better supported; hence, PPPD
 1178 now fully supplants CSD.

1179 Members of the subcommittee and their advisors
 1180 carefully considered a proposal to include
 1181 PPV as a subtype of PPPD. Arguments in favor
 1182 of this idea focused on the richness of the definition
 1183 of PPV, which includes personality factors and
 1184 psychological symptoms, particularly phobic
 1185 symptoms, that were shown to affect clinical presentation
 1186 [12, 71], as well as 30 years of research that
 1187 described its clinical course [12, 35, 36] and identified
 1188 potential pathophysiologic mechanisms [33, 46,
 1189 67, 68, 71, 98]. Arguments against the idea included

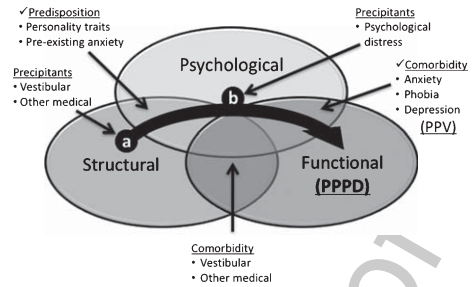


Fig. 1. Putative mechanisms of PPPD. PPPD is thought to develop via a dynamic process (arrow). In about 70% of patients, a structural vestibular syndrome (e.g., vestibular neuritis, BPPV) or other medical condition precipitates PPPD (black dot, a) [79]. Individuals who respond to the precipitating event 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.

1190 concerns that the mix of physical and psychologi-
 1191 cal symptoms and personality traits that constitute
 1192 PPV is difficult to operationalize in clinical practice
 1193 [75] and may have to be updated based on
 1194 newer research [76, 85], and that including PPV as a
 1195 subtype of PPPD without validating their relationship
 1196 could perplex clinicians and investigators. In
 1197 the end, a simple majority of subcommittee members
 1198 voted against including PPV as a subtype of
 1199 PPPD.

1200 The debate about PPV highlighted the fact that
 1201 PPPD is a dynamic condition as illustrated in Fig. 1.
 1202 It is a functional disorder, although structural and psy-
 1203 chological factors affect its development. Clinicians
 1204 who apply Criteria A and B as simple checklists of
 1205 symptoms without capturing the dynamic features of
 1206 their patients' histories (Criterion C) assume a reductionist
 1207 view of the diagnosis, potentially missing important
 1208 aspects of patients' morbidity (Criterion D) and nuances
 1209 of the differential diagnosis (Criterion E). Scientists
 1210 who fail to consider the dynamics of PPPD risk
 1211 muddling rather than illuminating the nature of the
 1212 disorder and its pathophysiologic mechanisms.
 1213

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