

## A Cognitive-Behavioral Model of Persistent Postural-Perceptual Dizziness

Matthew G. Whalley<sup>\*,1</sup>, *Clinical Health Psychology Service, Berkshire NHS Foundation Trust*  
Debbie A. Cane<sup>\*\*,1</sup>, *Audiology, Royal Berkshire NHS Foundation Trust*

*Persistent postural-perceptual dizziness (PPPD; previously termed “chronic subjective dizziness”) is a frequently observed disorder in patients who present with dizziness to audiology, ear, nose, and throat; or neurology clinics. The primary symptoms are persistent nonvertiginous dizziness, and hypersensitivity to motion and visual stimuli. These occur either in the absence of any active neuro-otologic illness or, where an episodic vestibular disorder exists, symptoms cannot be fully explained by the disorder alone. Diagnosis is necessarily multidisciplinary and proceeds by identification of primary symptoms and exclusion of other neurological or active medical disorders requiring treatment. Psychological processes are implicated in the development and maintenance of PPPD, with similarities to cognitive models of health anxiety and panic disorder, and there is evidence that cognitive-behavioral therapy is an effective treatment. A cognitive-behavioral model of PPPD is presented along with a case example. It is suggested that dizziness becomes persistent when it is processed as a threat, and that it is maintained by (a) unhelpful appraisals, (b) avoidance and safety behaviors, and (c) attentional strategies including selective attention to body sensations associated with dizziness. Once PPPD is identified techniques for its effective treatment fall within the skills mix of qualified cognitive-behavioral therapists or vestibular clinical scientists who have received additional training in cognitive and behavioral treatment.*

**D**IZZINESS is one of the most commonly reported symptoms in primary care (Kroenke et al., 1994), and within neurology it is the second most frequently reported symptom after headaches (Brandt, 1996). One study found the prevalence of dizziness in a community sample of working-age adults to be 40%, with half of these rating their dizziness as “handicapping” (Yardley, Owen, Nazareth, & Luxon, 1998). Chronic dizziness is often caused by dysfunction of the peripheral vestibular system, and many of these cases respond well to forms of physiotherapy such as vestibular rehabilitation (Herdman & Clendaniel, 2014). Dizziness (light-headedness) is also a symptom associated with anxiety and can occur secondary to biological preparedness for threat (fight or flight). It is one of the most frequently occurring symptoms of panic attacks, observed in up to 95% of patients with panic disorder (Barlow, 2004).

Up to 10% of patients presenting in neurology clinics have dizziness that cannot be explained by either a vestibular disorder or by another organic illness (Ödman & Maire, 2008). It is thought that a subset of dizzy patients

experience dizziness that is maintained by psychological factors such as anxiety (Eckhardt-Henn, Breuer, Thomalske, Hoffman, & Hopf, 2003; Furman & Jacob, 2001; Staab, 2012). Common neural substrates between dizziness and anxiety have been identified (Balaban & Thayer, 2001), but a truly integrated model of chronic dizziness has so far been lacking.

### What Is Persistent Postural-Perceptual Dizziness?

Persistent postural-perceptual dizziness (PPPD) is the most recent diagnostic term for what has previously been called “space motion discomfort” (Jacob, Lilienfeld, Furman, Durrant, & Turner, 1989), “phobic postural vertigo” (PPV; Brandt, 1996), “psychogenic dizziness” (Buljan & Ivančić, 2007), “chronic subjective dizziness” (CSD; Staab & Ruckenstein, 2007), and “psychophysiological dizziness” (PPD; Edelman, Mahoney, & Cremer, 2012; Edelman, n.d.). PPPD is recognized in the draft version of the International Classification of Diseases (ICD-11 Beta Draft, n.d.) and is also consistent with the presentation of somatic symptom disorder in DSM-5 (American Psychiatric Association, 2013). For simplicity the term “PPPD” will be used throughout this paper to refer to all of these conditions (see Staab, 2012, for a review of the history of diagnostic terms within this field).

PPPD is characterized by three main symptom clusters:

- Persistent nonrotatory dizziness that lasts 3 or more months

<sup>1</sup> Both authors contributed equally to this work.

**Keywords:** chronic subjective dizziness; psycho-physiological dizziness; phobic postural vertigo; cognitive behavioral therapy; formulation

- Hypersensitivity to motion stimuli (including the patient's own movement) and hypersensitivity to visual stimuli including the movement of objects in a busy visual environment
- Difficulty with precision visual tasks such as reading or using computers

These symptoms occur in the absence of an active physical neuro-otologic illness or medication causing dizziness, or where an active episodic medical or vestibular disorder exists, symptoms cannot be fully explained by the disorder alone (Staab, 2012). They occur in the presence of normal brain radiology findings, and nondiagnostic findings on tests of balance function (Staab & Ruckenstein, 2007). For reference, the clinical features of PPPD are given in Table 1.

PPPD can be precipitated by a medical condition (normally otogenic), or by acute episodes of anxiety. In this article we use the term “otogenic” where there is a pathophysiological precipitant and “nonspecific” where PPPD is precipitated by an episode of acute anxiety. We prefer the term “nonspecific” to the previous labels of “psychogenic” and “interactive” (Staab & Ruckenstein, 2003, 2007). Where a vestibular disorder is identified as a precipitant for PPPD (otogenic PPPD) it typically results in acute symptoms of rotatory vertigo (spinning) or other symptoms of dizziness including loss of balance. Peripheral vestibular disorders that are common precursors to PPPD include unilateral or bilateral vestibulopathy (vestibular neuritis or labyrinthitis), vestibular migraine, Ménière's disease, benign paroxysmal positional vertigo (BPPV), vestibular paroxysmia, and perilymph fistula (Staab, 2012). The underlying vestibular deficit, such as an asymmetry between balance and organ function, can either resolve (in which case the cause for the balance problem has resolved) or remain (in which case the patient's balance system “compensates” for the deficit with time and there is no remaining medical cause for persistent dizziness). For more information about vestibular compensation see the later section “Balance Control System Dysfunction and Balance Symptoms.” Nonspecific PPPD

can be precipitated by acute episodes of anxiety such as panic attacks.

### Diagnosis of PPPD

As serious central causes of dizziness (e.g., neurodegenerative disorders, space-occupying lesions, and vestibular schwannoma) can present in a similar way to PPPD (i.e., constant low-level dizziness), it is important that an accurate differential diagnosis is made by an appropriate professional such as a specialist in vestibular disorders; an ear, nose, and throat (ENT) specialist; or a neurologist. In this respect, PPPD can be considered similar to other health conditions, such as pain or chronic fatigue syndrome, where good practice guidelines within clinical health psychology recommend the identification of positive symptoms and the exclusion of other disease processes potentially responsible for the clinical presentation (Seime, Clark, & Whiteside, 2003).

A specialist balance assessment will consist of taking a history of past and current symptoms, balance function testing (e.g., videonystagmography, head impulse test, positioning/positional tests, calorics, and vestibular-evoked myogenic potentials), and administration of psychometric tests of patient's symptoms and perceived disabilities. Useful psychometric tests include the Vestibular Rehabilitation Benefit Questionnaire (VRBQ; Morris, Lutman, & Yardley, 2008, 2009), Dizziness Handicap Inventory (DHI; Jacobson & Newman, 1990), and the Nijmegen Questionnaire (Van Doorn, Colla, & Folgering, 1983). In clients with PPPD, test results should either be normal or, if significant findings are demonstrated (such as a significant canal paresis on caloric testing), either the patient's history and other tests of central compensation such as directional preponderance will suggest that compensation is complete. In the case of an active episodic vestibular disorder, the patient's symptoms cannot be explained by the disorder (or recorded physiological deficit) alone. Limitations of testing, such as the fact that a normal test result does not always preclude a vestibular disorder, often mean that the history and current reported symptoms are fundamental

Table 1  
Symptoms of (PPPD) Described by Staab and Ruckenstein (2007) and Staab (2012)

#### Clinical Features of Persistent Postural-Perceptual Dizziness (PPPD)

- Persistent sensations of unsteadiness and/or nonvertiginous dizziness lasting 3 or more months
- Symptoms are present on more days than not (at least 15 of last 30 days)
- Symptoms worsen with upright posture, head or body motion, exposure to complex or motion-rich environments
- Symptoms lessen or are absent in a reclined or resting posture
- Absence of currently active medical or neurological condition, or use of medication, that may cause dizziness
- Results from radiographic imaging exclude significant anatomical lesions
- Findings from balance function tests are within normal limits, reveal deficits not believed to be currently active, or cannot fully explain all of the patient's symptoms

Download English Version:

<https://daneshyari.com/en/article/5038586>

Download Persian Version:

<https://daneshyari.com/article/5038586>

[Daneshyari.com](https://daneshyari.com)