

A CBT MODEL OF PERSISTENT POSTURAL-PERCEPTUAL DIZZINESS

A Cognitive Behavioural Model of Persistent Postural-Perceptual Dizziness

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Acknowledgements

The authors would like to extend their thanks to the editor Dr Chu and to the two anonymous reviewers whose feedback significantly improved this article.

Whalley, M. G., & Cane, D. A. (2016). A Cognitive-Behavioral Model of Persistent Postural-Perceptual Dizziness. *Cognitive and Behavioral Practice*.

doi:10.1016/j.cbpra.2016.03.003

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Abstract

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 non-vertiginous 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 behavioural therapy is an effective treatment. A cognitive-behavioural 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: (1) unhelpful appraisals, (2) avoidance and safety behaviours, and (3) 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 behavioural therapists or vestibular clinical scientists who have received additional training in cognitive and behavioural treatment.

Introduction

Dizziness 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, 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 (lightheadedness) 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 (Furman & Jacob, 2001; Eckhardt-Henn et al., 2003; 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 et al., 1989), ‘phobic postural vertigo’ (PPV: Brandt, 1996), ‘psychogenic dizziness’ (Buljan & Ivancic, 2007), ‘chronic subjective dizziness’ (CSD: Staab & Ruckenstein, 2007), and ‘psycho-physiological dizziness’ (PPD: Edelman et al., 2012). PPPD is recognised in the draft version of ICD-11 (ICD-11 Beta Draft) 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 (for a review of the history of diagnostic terms within this field see Staab, 2012).

PPPD is characterised by three main symptom clusters:

- Persistent non-rotatory dizziness which lasts 3 or more months

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- 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 non-diagnostic findings on tests of balance function (Staab & Ruckenstein, 2007). For reference the clinical features of PPPD are given in table 1.

****Table 1 here****

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 'non-specific' where PPPD is precipitated by an episode of acute anxiety. We prefer the term 'non-specific' to 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. Peripheral vestibular disorders that are common precursors to PPPD include: unilateral or bilateral vestibulopathy (vestibular neuritis or labyrinthitis), vestibular migraine, Meniere's disease, benign paroxysmal positional vertigo (BPPV), vestibular paroxysmia, and perilymph fistula (Staab, 2012). The underlying vestibular deficit, such as an asymmetry between balance organ function, can either resolve (in which case the cause for the balance problem has resolved) or remains (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 & Balance Symptoms*'. Non-specific 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

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vestibular disorders, an 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 et al, 2008, 2009), dizziness handicap inventory (DHI: Jacobson & Newman, 1990), and the Nijmegen questionnaire (Van Doorn et al, 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 to the diagnosis. Where diagnosis is unclear an additional neurological referral and/or MRI scan of the brain and internal auditory meatus is requested – in the case of PPPD the results of these will be normal.

Symptoms of an uncompensated peripheral vestibular disorder (UVD) often overlap with PPPD and it can be hard to distinguish between them. Both sets of patients may report movement-evoked and visually-evoked symptoms of imbalance or disorientation. Many will also report fatigue, poor concentration and memory, and difficulties multi-tasking. Critically, however, patients with a compensated vestibular disorder should not report symptoms when still. Patients with UVD will often report more symptoms when multi-tasking such as when walking, and when turning their heads whilst talking, whereas for patients with PPPD the dual-task will serve as a distraction and symptoms will be reduced. Even if this distinction is unclear it is encouraging to note that similar treatment strategies including exposure to the stimuli which trigger symptoms will lead to symptom reduction in both conditions, albeit by differing mechanisms.

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Diagnosing clinicians are encouraged to carry out a simultaneous psychological screening. Adoption of concurrent medical and psychological screening has been found to reduce iatrogenic effects in conceptually similar conditions such as chronic pain (Kouyanou et al, 1998). Measures such as the health anxiety inventory (HAI: Salkovskis et al, 2002) are particularly likely to be informative.

Psychiatric Comorbidity of PPPD

Fear or anxiety is an unconditioned response to dizziness, and so it is unsurprising that anxiety features prominently in the presentation of those with PPPD. PPPD is frequently comorbid with other psychiatric conditions, although not in all cases. Kapfhammer et al (1997) conducted psychiatric assessments on a group of 42 patients with PPPD and found that although anxiety and mood disorders meeting DSM-III-R criteria were present in 2/3 of the group, they were absent in the final third. Similarly Staab & Ruckenstein (2007) found that 60% of patients with PPPD also met formal criteria for an anxiety disorder such as panic disorder and generalised anxiety disorder. Staab & Ruckenstein (2007) found that 1/3 of PPPD cases are directly precipitated by an episode of anxiety in the absence of any vestibular disorder. DSM-5 reorganised the category of 'somatic symptom and related disorders' and we would hypothesise significant overlap of PPPD with the new diagnostic category 'somatic symptom disorder'.

Chronological Development of PPPD

One of the most helpful contributions of Staab's (2012) model of PPPD is its consideration of the chronological development of the condition, particularly the identification of mechanisms operating in the acute phase of an illness that can contribute to the development of chronic dizziness. It identifies typical strategies (behavioural and attentional) which, while helpful in the acute stages, become counterproductive when maintained in the longer term. When an individual experiences acute dizziness or rotatory vertigo (spinning) sensible behavioural adaptations are adopted in order to ensure physical safety. These adaptations include holding on for support to reduce the risk of falling and minimizing head and other movement (as this will exacerbate dizziness caused by a vestibular disorder) to reduce the risk of injury. Other adaptations include keeping away from dangers such as busy roads where injury may be more likely or severe should a fall occur, and increasing vigilance towards the environment for threats to balance such as trip hazards.

Central (brain driven) adaptations are also made. There are three inputs to the balance system: the vestibular system (inner ear organ of balance), the visual system, and the proprioceptive system. Information

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from all three is integrated to ensure optimal maintenance of balance and upright posture both when still and moving. The relative importance (or weighting) of the vestibular, visual, and proprioceptive systems can change according to circumstance. When the vestibular input is damaged or unreliable the balance system relies less upon this input ('down-weighting') and relies more upon the other two reliable inputs ('up-weighting'). This will begin to occur following an attack of rotatory vertigo.

Staab's model argues that once the constant rotatory vertigo seen in the acute stage of the vestibular disorder resolves (which occurs spontaneously within a few days), longer-term recovery from the subsequent movement-evoked dizziness (which does not resolve spontaneously and requires the patient to move) is accomplished by relinquishing these acute adaptations. This gives the central balance control system the opportunity to readapt or compensate. He argues that once compensation has occurred (or function restores), PPPD results when there is failure to readapt and relinquish the acutely beneficial safety strategies. Continued up-weighting of the visual system causes the symptoms of visual vertigo. This is compounded by hypervigilance for and hypersensitivity to symptoms of dizziness, which in turn tend to lead to apparent exacerbation of symptoms – both visual and movement-evoked – and avoidance of evocative situations. Constant low-level symptoms seen in PPPD thus develop.

Other Formulations of PPPD

Although Staab's (2012) model is currently the most developed explanation of the mechanisms underlying the disorder there are other complementary formulations. Brandt (1996) developed an influential model of phobic postural vertigo (a term commonly used for this condition prior to PPPD). His model proposes that chronic dizziness is the result of mismatch between expected and actual sensorimotor signals from the visual, somatosensory, and vestibular inputs (recent research indicates this mismatch is broader and may encompass altered pain processing: Holle et al, 2015). According to this model if there is sensorimotor mismatch then self-generated motions can be misinterpreted as being externally-generated, causing postural vertigo to be experienced and leading to the adoption of inappropriate postural strategies. Brandt's conceptualization also included cognitive aspects but these were framed, we think unhelpfully, in terms of obsessional thinking.

Drawing upon evidence that some vestibular and anxiety symptoms are mediated by a common neuroanatomical substrate (Balaban & Thayer, 2001), Odman & Maire (2008) present a model of PPPD

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which emphasises neuroanatomical and neurochemical aspects. Their model identifies the contribution of anxiety to the maintenance of PPPD, but from a treatment perspective the model infers medical treatments at the levels of neuroanatomy and neurochemistry.

Taking a cognitive behavioural approach Edelman et al (2012) draw attention to the similarities between PPPD and panic disorder, arguing that both involve conditioned threat responses to misinterpreted and unwanted bodily sensations. They identify the counterproductive operation of safety behaviours in PPPD and hypothesise that cognitive behavioural treatment strategies similar to those used for panic disorder may be appropriate. Although frequently comorbid with anxiety disorders such as panic disorder earlier evidence indicates that not all cases of PPPD meet criteria for such diagnoses (Kapfhammer et al, 1997; Staab & Ruckenstein, 2007).

The Need For a Cognitive-Behavioural Model of PPPD

Advances in our understanding of PPPD mean that many aspects of the condition are now well explained. However, synthesis of a number of ideas is needed at this point in time. Cognitive behavioural theory (CBT) is an ideal framework within which to bring together the biopsychosocial components operating in PPPD. Our model draws heavily upon the theory of Staab (2012) which has done an excellent job of elucidating the precipitants and factors operating which result in a presentation of PPPD. It is also necessary to acknowledge the advances made by Brandt (1996) and Edelman et al (2012). Our model extends this work through detailed consideration of the psychological mechanisms which serve to maintain PPPD, with particular emphasis upon how these may predict effective treatment strategies. The resulting model shares common transdiagnostic mechanisms with cognitive behavioural formulations of health anxiety (e.g. Salkovskis & Warwick, 1986), and can be considered a dizziness-specific implementation of a health anxiety model.

A Cognitive Behavioural Model of PPPD

This paper introduces a model to explain the development and maintenance of PPPD (both otogenic and non--specific types) and to provide a framework for the cognitive-behavioural treatment of PPPD. Our model proposes that persistent dizziness is maintained by a variety of processes in the absence of a balance control system dysfunction. The model is presented in two states: 'initial/precipitating' and 'persistent' (see figure 1). The persistent state is of particular interest to the therapist working with patients diagnosed with

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PPPD. The model illustrates the mechanisms that are serving to maintain persistent dizziness and therefore identifies targets for treatment in therapy.

Insert figure 1 here

Overview

A key tenet of CBT is that appraisal shapes emotion, and emotion guides behaviour (Beck, 1976). Appraisals of particular somatic sensations as threatening can lead to feelings of anxiety and action to avoid the sensation. In many cases the action to avoid leads to a feedback cycle in which the individual fails to learn about the benign nature of the somatic sensation. This cycle has been labeled ‘interoceptive avoidance’ (Barlow, 2002) and has been identified as a transdiagnostic mechanism common to a range of conditions (Frank & Davidson, 2014). By including the mechanism of interoceptive avoidance our model shares similarities with cognitive models of panic (Clark, 1986), health anxiety (Salkovskis & Warwick, 1986, Stern & Drummond, 1991; Salkovskis, Warwick & Deale, 2003) and with the cognitive behavioural models of chronic pain (Lethem et al, 1983; Vlaeyen & Linton, 2000; Sharp, 2001). Our model can be considered to be part of the family of health anxiety formulations, although it includes additional dizziness-specific considerations. Our model is distinct from previous conceptualisations of PPPD in that treatment interventions are framed using cognitive terminology. Figure 1 summarises the key components of this model of dizziness in both initial and persistent phases, and components are explained in greater detail below.

Vulnerability

Certain personality characteristics are thought to increase vulnerability to the development of PPPD. Compared to patients with common vestibular symptoms those with PPPD score higher on neuroticism (a component of which is trait anxiety) and lower on extraversion (Staab et al., 2014). Higher than normal levels of anxiety experienced during an acute bout of vestibular symptoms may also predispose patients to developing PPPD (Best et al., 2009; Godemann et al., 2005; Heinrichs et al., 2007).

It has been established that a greater sensitivity to internal body sensations is a vulnerability factor for panic disorder (Schmidt, Lerew & Trakaowski, 1997), and that patients with panic disorder are more likely to interpret ambiguous body sensations as signs of impending catastrophe (Clark, et al., 1997). Although to the best of our knowledge this is currently untested we would make the empirical prediction that

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similar patterns would be observed in patients with PPPD compared to patients with other vestibular disorders.

A tendency to misinterpret ambiguous medical information in a negative way is a key feature of health anxiety (Warwick & Salkovskis, 1990). This tendency has been found to predict subsequent anxiety following a medical investigation at 1 week, 3 month, and 1 year latencies (Rimes & Salkovskis, 2002). Although it remains to be tested we predict that patients with PPPD engage in such misinterpretations and would score more highly on a measure of health anxiety than patients with other vestibular disorders.

Triggers For an Episode of PPPD

A wide variety of vestibular disorders can act as triggers for otogenic PPPD (Huppert et al., 1995). Conditions which cause recurrent or episodic vertigo such as migraine associated dizziness, Meniere's disease, or recurrent benign paroxysmal positional vertigo (BPPV), are particularly common triggers (Neff et al., 2012). Triggers for non-specific PPPD include episodes of anxiety, such as panic attacks, where the patient experiences dizziness or unsteadiness.

Balance Control System Dysfunction & Balance Symptoms

Persistent dizziness is often precipitated by an acute vestibular insult. In this phase there is usually dysfunction of one or both of the balance organs, their associated nerves, or central processing systems. The balance control system can rest in a number of states, depending upon whether the peripheral 'cause' for the balance episode has been 'compensated for' centrally. A reduction of function in one or both balance organs will initially cause acute vertigo (unilateral loss of function), or severe imbalance (bilateral loss of function) – even when the individual is still. Symptoms while stationary will gradually diminish over 24-48 hours as static compensation occurs centrally for an asymmetry in function (or as function restores in Meneire's, or as the altered neurological processes in migraine-associated-dizziness resolve). Where balance organ function remains reduced, the patient will then experience movement-evoked dizziness/imbalance, and this is often associated with visual vertigo (symptoms of nausea, imbalance and disorientation elicited by busy visual environments). Dynamic compensation will only occur with exposure to the symptoms. This is achieved by having the patient practice a tailored set of vestibular rehabilitation exercises, of increasing difficulty, which evoke the symptoms mildly 2-5 times per day over a number of weeks (generally 6-8 depending on compliance). Once the patient is symptom-free upon movement and in busy environments compensation is

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said to have occurred, even if the balance organ function remains impaired. It should be noted that compensation and complete alleviation of symptoms should be possible in patients with unilateral dysfunction. Recovery in patients with bilateral hypofunction will take longer and patients will often be symptomatic during some activities or situations (for example where both vision and proprioception is compromised, or in sports requiring running or quick changes of direction). Decompensation can occur at times of stress, anxiety, tiredness or illness. During decompensation there is no further change in balance organ function, but the compensation mechanisms are dysfunctional. Exercises to promote compensation can then be re-administered. (Curthoys & Halmagyi, 2007).

These states of the balance control system relevant to PPPD are outlined below:

- 1) Balance organ functioning normally → Patient symptom-free
- 2) Balance organ functioning normally + panic attack → Balance symptoms (lightheadedness) experienced
- 3) Dysfunction in balance organ (reduction of function in one or both balance organs) → patient experiences acute vertigo (unilateral drop in function) or severe imbalance and oscillopsia (bilateral drop in function. This will initially occur even when still, for up to 48 hours, and then be movement-evoked
- 4) Dysfunction in balance organ → Balance system is uncompensated → Balance problem experienced
- 5) Dysfunction in balance organ → Central compensation occurs → No balance problem on movement experienced
- 6) Balance organ function restores → No balance problem experienced (no compensation required)

In the persistent stage of PPPD the dysfunction resolves. This may be at the level of the balance organ (if function restores), at the level of the 'balance control system' through central compensation for dysfunction in the balance organ, or through recovery from the acute experience of panic. Our model proposes that persistent dizziness is maintained by a variety of processes in the absence of a balance control system dysfunction.

Balance Symptoms

Symptoms experienced prior to the development PPPD will depend upon the precise nature of the triggering event. In the case where vestibular neuritis is the trigger, symptoms experienced would include acute rotatory vertigo (even when still) and episodes of vomiting for 24-48 hours exacerbated by movement. This is then followed by dizziness evoked by movement. In the case of a triggering panic attack physical

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symptoms might include lightheadedness and imbalance, and both may be accompanied by nausea and an emotional response of fear. Balance symptoms in the persistent phase of PPPD involve sensations of rocking or swaying, unsteadiness, or non-vertiginous dizziness, and sensitivity to motion and visual stimuli. In PPPD these occur in the absence of any active medical or neurological condition which would cause balance symptoms.

Appraisal Of The Balance Symptoms / Bodily Sensations

In cognitive therapy the appraisal of a stimulus determines the response. If balance sensations are appraised as non-threatening (e.g. *“My dizziness is understandable as I have had a few drinks”, “I’m just hot and I’ll feel better when I get some fresh air”*) then we would hypothesise that individuals will not develop a persistent balance problem. However, if balance symptoms are appraised as threatening (e.g. *“I’m having a stroke and may die”*) then a strong emotional response is initiated, often followed by a behavioural response – the consequences of which can lead to persistent dizziness. Unhelpful appraisals have been linked to the chronicity of dizziness following vestibular dysfunction (Godemann et al., 2005; Heinrichs et al., 2007, Yardley, 1994).

The magnitude of perceived threat has been linked to four types of appraisals: perceived likelihood of threat, perceived consequences of threat, perceived ability to cope, and perceived rescue factors (Salkovskis, 1996). This framework is helpful in understanding balance appraisals, several types of which can result in a sense of threat. Catastrophic appraisals regarding the consequences of symptoms can increase anxiety (*“I’m having a stroke”, “I’m in real danger”, “I could fall”, “I’ll never get better”*). Over-general interpretation of normal sensations of dizziness or imbalance can lead patients to predict an acute attack as likely (*“I’m feeling dizzy, this means I’ll have another severe attack”*). Misinterpretations of (normal) movement-evoked symptoms experienced during the uncompensated phase of a peripheral vestibular insult are an example of this. Catastrophic appraisals regarding coping can further increase anxiety (*“I can’t cope with being ill”, “If I fall no-one will help”*). Negative appraisals of others can trigger a perception of social threat (*“Other people will think I’m drunk”*) leading to feelings of embarrassment or humiliation.

Emotional Response to Appraisal

As described above, the idiosyncratic nature of the appraisal will determine the precise emotional response. The most common emotional responses in PPPD are anxiety and fear, but other responses such as

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shame can also be present. For example *“People will think I’m drunk”* (embarrassment), *“I’m letting my family down”* (guilt/shame).

Behavioural Adaptation Strategies

In the acute stage of a balance problem there is a direct threat to physical safety due to the risk of falling. When this threat is perceived the individual takes action to minimise the consequences of the dysfunction. As already discussed, Staab (2012) identifies these as ‘acute behavioural adaptation strategies’. In the short term these acute behavioural strategies are effective at keeping the individual safe from harm. In individuals who experience an acute balance problem but do not go on to develop PPPD compensation occurs in tandem with these strategies being relinquished and the symptoms resolve.

In individuals who develop persistent dizziness these acute behavioural strategies persist, and often extend or generalise. Patients presenting at the clinic may have stopped exercising, avoid busy places, and retreat to ‘safe’ places such as home whenever dizziness starts. They may avoid situations in which they fear that the consequences of a further attack of dizziness may be serious, and are often not keen to go back to places where they had an attack of dizziness for fear of a further attack. The persistence of such strategies can lengthen the time to recovery by preventing the full compensation of the balance control system, and may also lead to PPPD. When acute behavioural strategies continue to be used beyond their original usefulness they have the role of preventing the updating of unhelpful balance related appraisals (e.g. *“If I move my head I’ll get dizzy and fall”*, *“I’m not safe unless I hold on”*). Such appraisals lead to threat perception and further preparation for action.

Acute Attentional Strategies

As well as behavioural strategies Staab (2012) also identifies attentional strategies as operating in the acute stage of a balance disorder, which in PPPD the patient has failed to relinquish. These attentional strategies vary in terms of how automatic or deliberate they are. One attentional strategy in PPPD is increased vigilance to environmental factors with the aim of reducing risk. Such a strategy would include looking out for things that might result in injury (e.g. slip, trip hazards) or factors that would increase the severity or consequences should an accident occur (e.g. where in a pavement it would be safest to fall, such as away from a road). However, when used longer-term, increased vigilance (hypervigilance) for danger

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increases the likelihood of environmental dangers being identified, with a consequential prolonging of- or increase in threat appraisal and threat perception.

Another key attentional strategy, albeit one out of control of the patient, is an alteration in importance given to (or ‘attention’ paid) to sensory inputs to maintain balance at the expense of others by the balance control mechanisms in the brain. Successful balance relies upon combined input from the visual, somatosensory, and vestibular systems. In a situation where one of these systems is unreliable, such as during an acute vestibular disorder, up-weighting of visual and somatosensory input (i.e. the brain relies more upon these inputs for balance) and down-weighting of vestibular input (i.e. the brain relies less upon this input for balance) occurs automatically. This strategy is useful acutely following a vestibular insult, but up-weighting of visual information in the long term can cause symptoms of visual vertigo (imbalance or disorientation in a busy visual environment, or difficulties with reading or computer work). This symptom is a feature of some uncompensated peripheral vestibular disorders but also a key feature of PPPD. Patients find it an uncomfortable symptom and if it is appraised as threatening it can lead to the adoption of further unhelpful behavioural strategies.

A final attentional strategy is the checking and monitoring of the bodily sensations related to dizziness. In an acute phase such monitoring allows the rapid identification of a threat (in signal detection theory a ‘hit’). However, once the balance control system is operating normally mental checking for balance sensation increases the likelihood of balance sensation perception, which can trigger the loop of faulty appraisal (false alarms), threat perception, and further vigilance. In this way patients can become hypersensitive to symptoms of dizziness. We hypothesise that the additional cognitive load of checking and monitoring also affects patient’s concentration.

Treatment Implications

Cognitive behavioural treatment for PPPD progresses through a number of stages. Our model suggests that recovery from PPPD requires change in three areas: alterations in appraisals related to threat and health, reduction in unhelpful behavioural adaptation strategies, and reduction in unhelpful attentional strategies. Previous RCT and case study literature supports the use of cognitive behavioural techniques to promote symptom change in PPPD (e.g. Edelman et al., 2012; Holmberg et al., 2006). Below we describe the procedures that we have found particularly helpful in work in our clinic, informed by our model.

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Psychological Assessment Including Functional Analysis

A psychological assessment of PPPD should begin with a functional analysis:

- The triggering event(s) for the patient's PPPD, or their experience of other significant episodes of dizziness – as well as gathering details of where and when, it is informative to explore a patient's understanding of the symptoms at the time as well as later.
- Presenting problems – how often and for how long does the patient experience their dizziness? In PPPD rotatory vertigo is not typically experienced and patients will report a wide range of non-vertiginous symptoms such as unsteadiness, lightheadedness, or rocking.
- Modifying factors – at which times, in which locations or situations, during which activities, or with which company are the symptoms *most* and *least* bothersome?
- Appraisals regarding symptoms and the triggering events:
 - o What did (does) the patient believe was (is) happening?
 - o What was the patient predicting would happen?
 - o How do they currently explain what they are feeling?
 - o (Therapist to attend particularly to threat and health appraisals).
- Behavioural adaptations:
 - o What is the patient doing or not doing in order to cope with how they are feeling?
 - o Why are they acting (or not acting) in these ways? (Clarify patient's beliefs and rationale)
 - o Assess the presence of avoidance or safety behaviours (e.g. holding on for support, changes in walking behaviour)
- Attentional strategies:
 - o What is the patient paying more (or less) attention to? (E.g. trip hazards, flickering patterns, inwards towards body sensations).
 - o Does the patient mentally 'check on' any symptoms or body sensations?

The following areas should also be investigated:

- Past mental health history – pay particular attention to the experience of anxiety and panic attacks
- Psychosocial factors which may provide context for the current presentation, or which may influence threat appraisals (e.g. family and work stresses, significant life events).

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- Biological factors – anything which temporally coincided with the dizziness onset (e.g. cold or flu which may have temporarily affects the vestibular system).

Measures such as the vestibular rehabilitation benefit questionnaire (VRBQ), dizziness handicap inventory (DHI), and the Nijmegen questionnaire are helpful measures of physiological symptoms. Measures such as the health anxiety inventory (HAI: Salkovskis et al, 2002) can provide information regarding health appraisals. Measures of anxiety and depression can also be informative.

Formulation And Rationale For Treatment

Our model is biopsychosocial in nature, incorporating (typically) biological triggering events that have led to the adoption of behavioural or psychological strategies, which are in turn serving to maintain the experience of dizziness. A case conceptualization synthesises information from the assessment together with theory to aid a patient's understanding of a problem and the mechanisms maintaining it. This guides selection of treatment approaches. Our approach to conceptualization of PPPD with patients typically includes:

- Psychoeducation regarding the balance control system – its integration of vestibular, visual, and somatosensory inputs, and typical outcomes (symptoms, subjective experiences) when inputs become unreliable.
- Psychoeducation about the cognitive behavioural model – the links between thoughts (appraisals) and emotion, how emotions guide behavior, and how actions sometimes have unintended consequences.
- A summary of the patient's appraisals regarding their dizziness, their emotional responses, and a summary of their behavioural and attentional coping strategies.
- An exploration of the potential consequences (intended and unintended) of the strategies adopted, and consideration of alternative coping strategies.

Explore & Update Symptom Origin Appraisals

Patients typically present with purely biological or medical appraisals for their symptoms. In PPPD medical appraisals for current symptoms will normally be unhelpful because they prompt maladaptive coping strategies (such as passive waiting for symptom improvement or suppression of balance symptoms with medication). In addition to a biopsychosocial case formulation and psychoeducation about CBT and

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PPPD, behavioural experiments (e.g. hypothesis testing or an interoceptive exposure exercise) can be used to generate evidence for a behavioural contribution to an experience of a somatic symptom. Helpful outcomes of this stage include new appraisals that the patient's actions and focus of attention can influence their perception of symptoms. In-session behavioural experiments for manipulating attention can be used to reinforce this point by, for example, attending to symptoms vs. dis-attending from them, or testing the magnitude of lightheadedness when standing near a support vs. standing in the middle of the room. If the symptom origin is fixed and organic these conditions should all feel the same. Patient's understanding of the results of these experiments should be gently explored, and summaries drawn.

Explore & Update Threat Appraisals

Patients with PPPD typically present with threat appraisals drawn from a limited number of categories. It is clinically useful to make explicit the individual's idiosyncratic threat appraisals. Table 2 illustrates a selection of typical threat appraisals and a selection of useful questions to elicit such beliefs and assumptions.

****Table 2 here****

To update threat appraisals behavioural experiments can be conducted to test their validity. A similar approach is used in patients with chronic pain maintained by fear and avoidance, where behavioural experiments have been found to be superior to graded exposure in reducing threat appraisals (Leeuw et al, 2008). Graded exposure to feared stimuli and situations can also act to increase self-efficacy and to reduce threat appraisals. For PPPD examples of common predictions and experiments are given in table 3.

****Table 3 here****

A selection of typical avoidance and safety behaviours common in PPPD are given in table 4 along with helpful techniques to address these:

****Table 4 here****

Teach Relaxation Techniques

Dizziness and lightheadedness can be induced by hyperventilation. Diaphragmatic breathing can be used to counteract the effects of hyperventilation-induced dizziness. If relaxed breathing leads to a reduction in dizziness it can further reinforce the new appraisal that such symptoms are anxiety-mediated. There has been debate within the field of panic treatment regarding the efficacy of teaching breathing retraining: a

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number of studies have found it helpful, but a dismantling study found that it did not add anything over and above cognitive restructuring and interoceptive exposure (e.g. Schmidt et al., 2000). In our experience patients find it helpful, and it may help to facilitate patient approach toward feared situations (Rachman, Radomsky & Shafran, 2008). PPPD often occurs in patients with high trait anxiety (Staab et al., 2014) and we hypothesise that relaxation may also reduce symptoms via this route.

Interoceptive Exposure

Interoceptive exposure exercises are designed to facilitate approach towards feared body sensations, and are a key component in the treatment of panic (Barlow, 2004). Interoceptive exposure exercises, such as deliberate hyperventilation to induce lightheadedness, are an effective way of changing appraisals about the origin of bodily symptoms, and about the threat implied by the experiencing of such symptoms. When taught in combination with relaxed breathing (i.e. the patient learns that they have bidirectional control over the symptom) such exercises can be a powerful way to shift threat cognitions. In the treatment of panic a number of interoceptive exposure exercises, such as hyperventilation, spinning in a chair, or vigorously shaking one's head, are used to induce a feeling of dizziness. In the case of PPPD, as full compensation has occurred then exercises including hyperventilation, gentle movements such as slow/moderate head turns (causing symptoms secondary to increased motion sensitivity), computer screen work (causing symptoms secondary to visual sensitivity), and standing with eyes closed can be used as a means to safely induce dizziness/imbalance. Care should be taken with those patients with bilateral hypofunction as, despite compensation, the patient will always have impaired balance - notably with eyes closed on unstable surfaces.

Reduce Avoidance and Safety Behaviours

Patient's behavioural adaptations, which include avoidance and safety behaviours, are driven by their threat appraisals. Patients are reminded that their balance symptoms are maintained through reduction in activity, and are encouraged to reduce their avoidance and increase their level of activity. Such exposure exercises may need to be carried out in a graded fashion, eventually leading to the dropping of safety behaviours. Some patients may find it helpful to be encouraged to identify their emotion and then to act in opposition to the behaviour it encourages ('opposite action': Linehan, 1993).

Exercises To Reduce Visual Vertigo and Motion Sensitivity

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Visual vertigo is caused by an automatic up-weighting of visual input to the balance control system following an acute vestibular event. This is a helpful acute strategy but often persists unhelpfully in patients with PPPD. Even when compensation has occurred many patients report an increase in symptoms on head/body movements and when walking. Vestibular rehabilitation plans often include recommendations for patients to practise head and body movements whilst standing and walking, balance exercises, and gaze stabilization exercises. With completion of these exercises down-weighting of the visual system often results. In those for whom visual symptoms persist visual desensitization exercises can be given. Essentially these are sets of activities, tailored to the individual, that cause mild symptoms of visual vertigo. The exercises typically need to be done for a number of minutes up to 5 times per day for 8-12 weeks, gradually increasing in difficulty as the patient's symptoms subside. Examples in increasing order of stimulation include:

- Looking at a busy visual background e.g. spotty or striped wallpaper
- Head turn or nod looking at a letter of the alphabet superimposed upon a busy visual background
- Head turn or nod looking at busy visual background
- Looking at a moving busy background (e.g. scrolling up and down a computer screen, watching or playing action computer games)
- Head movements with a moving busy background (e.g. moving screen saver)
- Looking at a revolving striped umbrella

Graded exposure to real world busy environments can then be added to further promote visual desensitization. Patients with PPPD often report that they avoid shopping due to visual vertigo symptoms provoked by supermarket aisles – a graded exposure programme to such environments can therefore be an effective exercise to reduce visual vertigo. It is important to ensure that the patient is not a migraineur, and to ensure the exercises do not prematurely overwhelm the visual system, which can sometimes lead to decompensation.

Sophisticated virtual reality systems have demonstrated efficacy in reducing visual sensitivity (Pavlou et al., 2004). Recent research indicates that use of the computer game accessory 'Wii fit' as a rehabilitation tool indicates may be an inexpensive alternative (Verdecchia et al., 2014). Other visual desensitization options include DVD-based visual/optokinetic stimuli (Pavlou, 2010).

Mindfulness and Acceptance Techniques

Complementing the use of traditional CBT approaches are mindfulness and acceptance interventions. Practitioners of these third-wave CBT approaches argue that an overarching goal for this work is to act in the service of valued goals (e.g. living a fulfilling life in spite of a symptom) rather than focusing on symptom reduction (Bach & Moran, 2008). Instead of cognitive restructuring of unhelpful appraisals patients might be taught the use of mindfulness or cognitive defusion techniques. Cognitive defusion strategies might include distancing (*“I am noticing that I am having a worry about falling”*). Regular mindfulness practice can be used to cultivate non-judging acceptance and there is reasonable evidence that these approaches are helpful in the treatment of other health conditions such as chronic pain and tinnitus (Ost, 2014). Many of our patients with PPPD have found them helpful and another study of PPPD included mindfulness and acceptance approaches as part of a successful treatment programme (Edelman et al, 2012).

Case Example

A fictitious case example is presented below showing how the new model of treatment for PPPD may be applied in practice.

Referral

Ms B was a single woman in her mid-30's with three young children. Her initial diagnosis had been one of vestibular neuritis, but when she was seen in the Audiology Balance Clinic no signs of active pathology were demonstrated – meaning that her balance organ function had restored or compensation had occurred. Despite this, Ms B continued to report symptoms of persistent dizziness that were present even when stationary. She described these as a rocking or light-headed sensation, and reported that they were made worse when she moved or when out in a busy visual environment. A diagnosis of otogenic PPPD was made and Ms B was referred to a psychologist.

Psychological assessment

At assessment with the psychologist a functional assessment was completed. Ms B reported that her first episode of vertigo had occurred a year previously when she had woken feeling a little nauseous and generally unwell. She described how she had been bending down to dress her children as normal when she suddenly experienced a spinning sensation (acute rotatory vertigo) which caused her to vomit and to feel very frightened. She was unable to walk and had to crawl back to bed. The vertigo continued for a number of

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hours during which time she reported feeling that she had to ‘hold on to the bed’ for fear she would fall even though she was lying down. She described having worried that there was something seriously medically wrong with her (health appraisal), and having to call her mother to come and take care of her children. Within 48 hours she stopped experiencing vertigo while she stayed still but she continued to experience short lived dizziness and imbalance whenever she moved. She continued to worry about the cause of the dizziness, tried to avoid movements and situations that evoked it, and admitted to close internal monitoring of her symptoms. After a few weeks she began to notice dizziness not only when moving but also at a low level when still. Ms B reported that she had not experienced any further attacks of acute vertigo but that for the past year in addition to her constant low level symptoms she had experienced exacerbations of what she described as ‘dizzy attacks’ when out in public, where she had felt the need to sit down. She expressed considerable concern about what would happen if she experienced a severe attack in public and worried what would happen to her children (threat appraisal).

Ms B did not report any previous history of mental health problems such as anxiety or depression. She did not have a significant physical health history. When discussing her family she reported that her father’s health had always been poor and that this had affected his ability to be a good parent – she was concerned about this pattern repeating itself in her own life. Behaviourally Ms B had reduced the amount of physical activity that she would attempt, particularly when on her own or when she was in the sole care of her children. She tried to keep more still, particularly reducing her head movements, would climb stairs very carefully, and had made sure to try to reduce trip hazards at home. Her most regular physical activity was walking her children to school but she would often avoid this by asking a neighbour to take them if she felt at all unwell, and if she did go herself she would attempt to minimise her anxiety by taking a stroller/buggy for balance despite her children being too old to need it. She would avoid routes involving busy roads or uneven surfaces. She would also try to avoid busy locations such as shops, which she found disorienting. She reported being very mentally aware of her balance and would frequently ‘check in’ to assess how she was feeling.

Ms B reported significant anxiety and feeling very sure that she would fall unless she took precautions to prevent this from happening. When her psychologist explored her anxious predictions Ms B was able to describe a mental image of seeing herself collapsed on a pavement unable to get up, and that in

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the image her young children were very upset and did not know what to do. Ms B found this image very upsetting, and reported that she would typically try to mentally avoid it.

Case conceptualisation

The psychologist hypothesised that a number of mechanisms might be operating to maintain Ms B's experience of dizziness and heightened anxiety:

- Unhelpful cognitive appraisals regarding the nature and consequences of symptoms – including her beliefs regarding the uncontrollability of the events, her inability to cope, and her concern that she was a bad mother – leading to feelings of anxiety and shame.
- Classical conditioning – the fear Ms B felt during the original episode may have led her to associate previously neutral physiological stimuli with fear (interoceptive conditioning).
- Behavioural avoidance & safety behaviours – leaving her with fewer opportunities to disconfirm anxious predictions about the likelihood of catastrophe, reinforcing the idea that she was a bad mother, and possibly leading to unintentionally greater perception of balance symptoms.
- Thought suppression of the unpleasant imagined image may have led to 'rebound' and increased perception of this unwanted cognition (Wenzlaff & Wegner, 2000).
- Attentional strategies including attending to the minutiae of her interoceptive experience meant that she was more prone to notice bodily experiences, then to mis-appraise them and experience anxiety.
- Central up-weighting of visual information by her balance control system (an automatic process) could have been contributing to experiences of visual vertigo.

A case conceptualization was developed collaboratively with Ms B during which the psychologist was able to provide information about the balance control system (with reiteration that her balance system was functioning normally after a transient disruption) and psychoeducation regarding the CBT model. The psychologist summarised Ms B's threat appraisals and anxious predictions, and together they explored how these can lead to feelings of anxiety. Her feelings informed discussion of her behavioural (e.g. safety behaviours), cognitive (e.g. worrying, asking 'what if ...' questions), and attentional (e.g. symptom monitoring, central up-weighting) coping strategies – the intended and unintended consequences of each of these actions were explored.

Treatment & Challenges

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An agreed treatment plan was developed from the case conceptualization. A provisional plan was to first attend to Ms B's unhelpful health and threat appraisals, teach relaxation techniques to manage anxiety, work to reduce her use of avoidance and safety behaviours, and then work to reduce her use of unhelpful attentional strategies. Three treatment sessions were initially negotiated, and a further three agreed before treatment was completed. Ms B attended all sessions, and the main block of treatment was completed within two months.

Behavioural experiments and guided discovery were used to test the effects of distraction and of directing attention towards and away from balance symptoms. Short experiments were conducted in-session while sitting and standing. In these Ms B was encouraged to direct her attention towards how she felt (interoceptive attention), or to describe topics such as her favourite holiday or the surroundings in the room (distraction / external attention). These exercises, followed by socratic questioning, led Ms B to generate new health appraisals "*If I pay less attention to it I'm less bothered by it*" and "*If this was a permanent disorder of my inner ear then distraction would not make it better*". Cognitive restructuring and soothing self-talk were practised to increase coping self-efficacy. Time was spent examining the likelihood of catastrophic consequences and the likelihood of not being able to summon help should a catastrophe occur, and Ms B became more adept at 'decatastrophising'. She and the therapist developed a 'worst case' plan to increase her sense of coping efficacy. Despite cognitive work leaving her feeling stronger Ms B reported that she continued to experience an intrusive image of a worst-case scenario in which her children were distressed, and so an imagery rescripting technique was used to address the distress associated with Ms B's intrusive image of helplessness.

Interoceptive exposure exercises (hyperventilating) were used to expose Ms B to uncomfortable body sensations. She was able to do these in the therapy room but initially avoided doing the agreed exercises as homework. Similarly, behavioural experiments were conducted near the office to test the belief "*I will fall*" and a graded exposure hierarchy was developed, but Ms B avoided doing this work at home. She reported finding these exercises too threatening and she and the therapist struggled to generate suitable intermediate steps for a graded exposure hierarchy that would enable her to initiate the practice. To address Ms B's difficulty in initiating self-practice a second set of experiments were conducted in a local park. These tasks including walking and running on smooth and rough surfaces, hopping on and off curb stones, and

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running in straight lines. The therapist had to use a combination of hopeful enthusiasm and genuine curiosity to encourage Ms B to explore the effects of movement upon her symptoms, and the limits of her abilities. Initial success was an opportunity for more encouragement and further experimentation. These experiments were made more challenging by combining them with altered body sensations produced by deliberate (initially mild) hyperventilation. New appraisals were generated and tested: *“Even if I am feeling unbalanced I am able to make quite extreme movements without falling”*. Increased feelings of self-efficacy allowed her to access more positive appraisals. Some of these were contained within imagery which allowed her to quickly access summary versions of helpful appraisals (e.g. image of a time she had previously coped with adversity to encapsulate the idea *“Even if the worst does happen I will be able to cope”*). Challenges involved in this session included requiring a longer session, maintaining a curious stance/attitude, and containing the client’s anxiety in the face of experiences she found threatening. Reflecting later upon the therapy Ms B reported that this out-of-office session had been the most confidence-building.

Ms B was encouraged to practice the in-session work at home with the agreement that to begin with self-practice would be less extreme than the exercises she had already completed successfully in therapy sessions. Exposure exercises were designed to encourage more physical activity, building up to a return to her pre-illness levels. Ms B was able to engage with these with the support of her mother, after in-session rehearsal, and with repeated gentle encouragement. Over time she was also encouraged to reduce her use of safety strategies. Homework tasks included walking her children to school without a stroller/buggy for support, testing and measuring the effects of keeping her head less still, and re-engaging in light exercise.

Review And Follow-Up

At one-month follow-up Ms B reported that she had resumed activities such as taking her children to school and had begun attending a yoga class. She reported that her anxiety had reduced significantly. She described how she still intermittently experienced symptoms of visual vertigo, and that although they were not accompanied by significant threat appraisals they were still unwelcome. She was reassured that these experiences were a consequence of heightened visual sensitivity (visual ‘up-weighting’) and that they were not an indication of any underlying abnormality. She was informed that they would typically resolve with exposure to normal movement and that they were often the last symptom in PPPD to full remit. She was given some written psychoeducational material, a set of visual desensitization exercises, and was taught a

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simple mindful breathing technique to practice non-judgmental acceptance of her somatic and cognitive experience.

Summary and Empirical Support

A cognitive behavioural model of persistent postural-perceptual dizziness is presented together with suggested treatment targets. In PPPD an acute dysfunction of the peripheral vestibular system (or an anxiety reaction) causes rotatory vertigo or severe loss of balance, with heightened threat perception and subsequent adoption of behavioral strategies (automatic and voluntary) to keep the patient safe. Once the acute vestibular disorder (or anxiety reaction) has resolved or compensated patients with PPPD report persistent non-vertiginous dizziness, heightened threat perception, and have motion and visual sensitivity. This is due in part to failure to dispose of acute behavioral strategies and results in increased awareness of balance symptoms that continue to be interpreted as threatening. Safety strategies and avoidance serve to maintain unhelpful appraisals that further maintain the perception of balance symptoms as a threat. The proposed model is consistent with the main clinical features of PPPD, and provides a framework for treatment by identifying three key targets for change: unhelpful appraisals, avoidance and safety behaviours, and unhelpful attentional strategies. Components of the present model are present in other disorder models including health anxiety (Salkovskis & Warwick, 1986, Stern & Drummond, 1991; Salkovskis, Warwick & Deale, 2003), panic (Clark, 1986), and cognitive behavioural models of chronic pain (Lethem et al, 1983; Vlaeyen & Linton, 2000; Sharp, 2001). The present formulation may best be considered a dizziness-specific implementation of a health anxiety model.

The Efficacy of CBT in the Treatment of PPPD

The treatment approaches suggested by this model already have demonstrated efficacy for PPPD. A cognitive-behavioural treatment protocol with similarities to that identified here, implemented in 3 sessions, was used in a randomised trial of CBT for PPPD (Edelman et al., 2012). It used techniques which targeted patient's unhelpful appraisals, and encouraged patients to reduce unhelpful behavioural and attentional strategies. Significant reductions in dizziness were demonstrated post treatment, with 75% of patients reporting clinically significant reductions in handicap associated with dizziness. Significant positive behavioural changes were also reported post-treatment. These improvements were maintained at one-year follow-up (Mahoney et al., 2013).

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A long-term follow-up of PPPD patients in a neurology clinic who were given short-term interventions targeting psychoeducation, decatastrophising, and exposure to vertigo-inducing situations found that 75% of patients showed improvements in their symptoms (Huppert et al., 2005). A non-randomised trial comparing vestibular rehabilitation exercises to individualised CBT in patients self-managing their CSD found that although both groups led to improvements in vertigo severity, the addition of CBT led to additional improvements in vertigo handicap, depression and anxiety (Holmberg et al., 2006) although in this case improvements were not maintained at one-year follow-up (Holmberg et al., 2007).

Case report data have also suggested that cognitive-behavioural techniques may be efficacious in the treatment of PPPD. For example, Sardinha (2009) presented a case study of a patient who responded to a combination of medication, psychoeducation, experiments to test beliefs around the effects of attending to symptoms, and graded exposure to previous daily activities. Symptom relief was reported to occur within 9 weeks. There is also some evidence that CBT and associated techniques are helpful in treating dizziness more broadly than just PPPD. Naber et al (2011) offered a group treatment to dizzy patients in a neurology clinic which included cognitive behavioural, mindfulness and vestibular rehabilitation exercises. They found improvements in patient symptoms, and a decrease in patient utilisation of clinic services.

Clinical Recommendations And Guidelines

Techniques for the treatment of PPPD fall within the skills mix of professionals qualified in delivering cognitive behavioural therapy, or vestibular clinical scientists who have received additional training in cognitive and behavioural treatment. Our service operates a stepped-care approach. Patients have a diagnosis of PPPD confirmed at an outpatient balance clinic. Those with simpler presentations receive psychoeducation and guidance for self-directed exercises in sessions in this service. Patients with more complex presentations or comorbidities are stepped up to interventions with a clinical psychologist in an outpatient psychology clinic, or to a balance specialist with an interest in CBT – where they receive more traditional CBT interventions with individualised case formulations. Edelman et al (2012) report effective treatment for PPPD in 3 sessions with an experienced clinical psychologist. In our experience although many patients do respond this quickly, others with more entrenched difficulties or comorbidities require a longer and more case-formulation-driven approach. Honacker et al (2013) report a case example of a client with

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otogenic PPPD, pre-existing generalized anxiety and hypochondriasis-type health anxiety who required a longer-term (1 year) treatment intervention.

A common clinical question regards the decision about which maintaining mechanism to address first. There is little evidence to support a particular course and so therapists must rely upon their clinical judgement. Our experience of working with this patient group would prompt us to make the following recommendations: (1) the component of treatment from which patients typically make the largest therapeutic gains is in tackling behavioural avoidance, (2) patients with higher levels of anxiety and poorer self-soothing abilities may benefit from a longer initial period of cognitive and emotional regulation work (e.g. cognitive restructuring, decatastrophising, relaxation exercises) before they are able to engage in behaviourally focused tasks, (3) symptoms of visual vertigo sometimes remain after otherwise successful treatment – reassurance and visual desensitization exercises have helped many of our patients, (4) in patients with comorbid anxiety disorders problem prioritization and a case formulation are necessary to negotiate a treatment plan (Persons, 2012), (5) where symptoms do not remit and where it has been difficult to exclude a diagnosis of an uncompensated vestibular problem re-assessment of vestibular function can be helpful – the patients may need more formal and intensive vestibular rehabilitation.

Preventing PPPD

We believe that many cases of PPPD could be prevented by timely education about the true causes of dizziness-related symptoms combined with early opportunities for vestibular rehabilitation. The following recommendations have been developed from a combination of our respective clinical practices as a balance specialist and clinical psychologist, as predictions from cognitive behavioural models of health anxiety, and from research concerning iatrogenic effects in conditions such as chronic pain. Research formally examining preventative strategies for PPPD should be considered a priority.

Early identification of those who may be at increased risk of developing PPPD followed by prioritization for testing and individualised rehabilitation would seem prudent. Even where a specialist balance assessment is required to rule out organic pathology primary care practitioners are in a good position to identify psychosocial contributors to a patient's distress and to normalize psychological reactions to unpleasant symptoms. This can be accomplished through psychometric measures of health appraisals / health anxiety (e.g. the HAI: Salkovskis et al., 2002), measures of anxiety or depression, or simple questions about

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life circumstances. Given the central role of negative (health) appraisals in health anxiety models such as ours early delivery of accurate information about the operation of the balance system and likely symptoms (e.g. Yardley, 2015) may reduce the likelihood of a patient going on to develop PPPD.

Although commonly prescribed vestibular suppressants (e.g. Prochlorperazine maleate) are useful in abating symptoms in the acute phase of a vestibular disorder such as vestibular neuritis or labyrinthitis their longer-term administration will only serve to prevent compensation and cause continuation of movement-evoked dizziness (Rascol et al, 1995). This in turn may consolidate avoidance of evocative movements and situations, continued symptoms, and repeated return visits to the primary care physician. If symptoms and fears about their cause are not addressed then persistent dizziness may result. Medical recommendation is that when patients present in primary care with a known acute vestibular disorder, such as vestibular neuritis then vestibular suppressants should be given for a few days only. There should be no need to prescribe such medication for BPPV. Medication where given should always be accompanied by education (verbal and written) regarding the likely cause of the dizziness symptoms and how to obtain resolution (e.g. Yardley, 2015).

Resolution of an acute vestibular disorder will commonly either require vestibular rehabilitation (exercises to gently evoke symptoms of dizziness and to promote compensation in cases of vestibular neuritis) or particle repositioning maneuvers such as the Epley maneuver for BPPV. If it is possible then treatment of a precipitating vestibular condition should be given (or at least initiated) by the primary care physician. Research has demonstrated that vestibular rehabilitation exercises can be successfully administered by nurses in primary care settings (Yardley et al, 2004). In cases where the diagnosis is unclear, in patients with suspected Meniere's disease or migraine, or in patients at higher risk of developing PPPD (such as those with a significant history of anxiety disorders), a timely referral should be made for balance function testing and appropriate rehabilitation should be made.

Empirical Support And Testable Predictions

There is some experimental research supporting propositions in the present model. The central role of appraisals in the development of PPPD-like conditions is supported by several lines of evidence. In a piece of prospective research Godemann et al (2006) assessed the cognitions of patients presenting at hospital clinics for the first time with vestibular neuritis and symptoms of vertigo and nausea before

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following these patients for two years to monitor the development of panic or somatoform disorders (relatively good proxy diagnoses for PPPD). They found that while initial vertigo severity was not correlated with the later development of a disorder, patients who exhibited intense preoccupation with the causes and consequences of their vertigo went on to experience significantly more difficulty. Similarly, Heinrichs et al (2007) found that an interaction between ‘fear of bodily sensations’ and the severity of an attack of vestibular neuritis predicted an experience of chronic dizziness 3 months later, and Yardley (1994) found that in a population of patients suffering from vestibular disorder the factors ‘fear of losing control’ and ‘increased autonomic symptoms’ predicted later handicap in these patients.

A number of key questions exist regarding PPPD. For example, given the demonstrated anxiolytic effect of SSRI’s (Hoffman & Mathew, 2008), the central role of threat perception and anxiety in our model may explain why such pharmacological interventions have demonstrated efficacy in PPPD (Staab et al., 2002; 2004). However, the role of anxiety in PPPD needs further research given the mixed pattern of anxiety outcomes in earlier treatment studies (Holmberg et al., 2006; Edelman et al., 2012). Our model would predict heightened anxiety specifically with respect to balance and balance symptoms. We would predict that patients with PPPD, like those with health anxiety, will be more likely to appraise ambiguous body sensations or medical information in a catastrophic manner – and will score more highly on a measure of health anxiety than other vestibular patients. We would also predict that, similar to patients with panic, those with PPPD will be more sensitive to internal bodily sensations than control patients with other vestibular disorders.

Finally, and in agreement with Staab (2011), our model predicts that ‘vestibular-rehabilitation-like’ exercises will be helpful in cases of PPPD even where there is no active vestibular disorder, due to the heightened sensitivity of PPPD patients to motion. Completion of such exercises will act to reduce behavioural avoidance and provide exposure to threatening body sensations (interoceptive exposure), thereby promoting habituation to the symptoms. This will lead to or be moderate by changes in appraisals related to balance. Dismantling studies are recommended to assess the relative contributions of the three key targets for change identified by our model (cognitive appraisals, behavioural adaptations, attentional adaptations). We plan to conduct small-N multiple baseline designs in our clinic to assess change in these factors.

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Clinical features of PPPD
Persistent sensations of unsteadiness and/or non-vertiginous 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

Table 1: Symptoms of (PPPD) described by Staab & Ruckenstein (2007) and Staab (2012)

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Typical threat appraisals	Useful questions to elicit specific threat appraisals
<ul style="list-style-type: none"> • Concern about short-term implications of the symptoms (e.g. falling) • Concern about long-term implications of the symptoms (e.g. unable to return to work) • Concern about embarrassment (e.g. having a further vertigo attack and vomiting in public) • Concern about not being helped, or being unable to get help should a catastrophe (e.g. further vertigo attack) arise 	<ul style="list-style-type: none"> • <i>“If you get dizzy do you think it will last for a long time, perhaps like your original vertigo attack, or will it pass quickly?”</i> • <i>“What are the consequences for you if you get dizzy?”</i> (Variations, when you are: alone, in company, at home, outside) • <i>“What anxious predictions do you make when you’re worried?”</i>

Table 2: Typical threat appraisals in PPPD and useful questions to elicit these

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Prediction	Behavioural experiment
Unless I hold on for support I will fall (catastrophe)	Relinquish acute balance control strategies
If I walk along a rough, bumpy, path I will fall and get hurt (catastrophe)	Walk along a variety of surfaces to test the prediction of falling
If I walk in a visually busy environment I will get overwhelmed and fall (catastrophe)	Walk in a variety of environments to test the prediction of falling
If I'm feeling unsteady everyone will notice, will think I'm drunk, and will ostracise me (social threat)	Therapist and patient to walk in public in normal and exaggerated ways. Patient to be given opportunities to observe the reactions of other people
I can't walk in a straight line and people will think I'm crazy (social threat)	Record the straightness of the patient and therapist's walks. Monitor the reactions of strangers during exaggerated walking

Table 3: A selection of anxious predictions in PPPD and behavioural experiments to test these

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Avoidance & safety behaviours	Techniques to address these
Limitation of head movement, or keeping head very still or upright	<ul style="list-style-type: none"> • Behavioural experiments to test whether the effects of head motion are as catastrophic as expected • Graded exposure to head motion (of which typical vestibular rehabilitation exercises may form a part)
Avoidance of anything which produces dizziness	<ul style="list-style-type: none"> • Psychoeducation about the balance system, the causes for dizziness, and the vestibular system's need for input for recalibration • Relaxation exercises to reduce anxiety which sometimes contributes to dizziness (such relaxation exercises can also engender a feeling of control) • Interoceptive exposure exercises
Use of safety behaviours to prevent anticipated falls (use of a stick, walking near something grab-able)	<ul style="list-style-type: none"> • Behavioural experiments to test whether catastrophic outcomes occur • Graded exposure to exercises
Avoidance of visual stimulation	<ul style="list-style-type: none"> • Visual desensitization exercises (e.g. computer games with fast motion) • Exposure to visually stimulating environments combined with relaxation exercises (counterconditioning)

Table 4: Common avoidance and safety behaviours in PPPD, and techniques to address these

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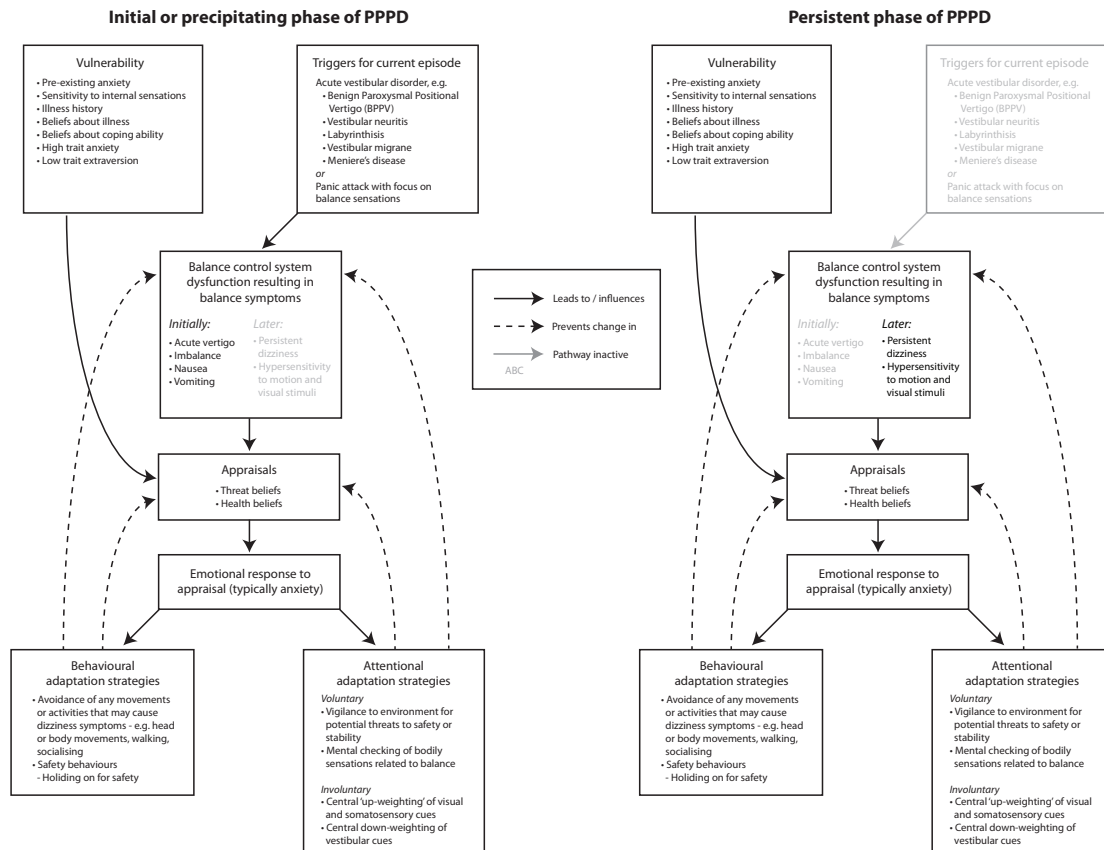


Figure 1: Cognitive behavioral model of persistent postural-perceptual dizziness