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Introduction to the assessment and management of persistent postural-perceptual dizziness

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Main Article

Diego Kaski takes responsibility for the integrity of the content of the paper

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Abstract

Objective. Persistent postural-perceptual dizziness classifies patients with chronic dizziness, often triggered by an acute episode of vestibular dysfunction or threat to balance. Unsteadiness and spatial disorientation vary in intensity but persist for over three months, exacerbated by complex visual environments.

Method. Literature suggests diagnosis relies on a clinical history of persistent subjective dizziness and normal vestibular and neurological examination findings. Behavioural diagnostic biomarkers have been proposed, to facilitate diagnosis.

Results. Research has focused on understanding the neural mechanisms that underpin this perceptual disorder, with imaging data supporting altered connectivity between neural brain networks that process vision, motion and emotion. Behavioural research identified the perceptual and motor responses to a heightened perception of imbalance.

Conclusion. Management utilises head and body motion detection, and downregulation of visual motion excitability, reducing postural hypervigilance and anxiety. Combinations of physical and cognitive therapies, with antidepressant medications, help if the condition is associated with mood disorder.

Introduction

Chronic dizziness is among the most limiting neurological symptoms in individuals of working age.[1] The commonest cause of chronic dizziness is persistent postural-perceptual dizziness – often referred to as 'three-PD'. Although the diagnostic criteria were conceptualised only recently, it is by no means a 'new' disorder, with functional neuro-vestibular symptoms prevalent across the ages, even into antiquity.

Indeed, the nomenclature for chronic dizziness has undergone substantial evolution across the last several decades. This latest term has helped to unify common features of earlier terms such as 'chronic subjective dizziness', 'phobic postural vertigo', 'visual vertigo' and 'space and motion discomfort'. It also facilitated a more modern understanding of persistent physical symptoms, away from purely 'psychogenic' explanations.

Following a consensus document on the diagnostic criteria created by the Behavioural Subcommittee of the Committee for the Classification of Vestibular Disorders of the Bárány Society,[2] the 11th edition of the World Health Organization's International Classification of Diseases ('ICD-11', beta draft), in 2015, defined persistent postural-perceptual dizziness (Table 1) as:

> 'Persistent non-vertiginous dizziness, unsteadiness, or both lasting three months or more. Symptoms are present most days, often increasing throughout the day, but may wax and wane. Momentary flares may occur spontaneously or with sudden movement. Affected individuals feel worst when upright, exposed to moving or complex visual stimuli, and during active or passive head motion. These situations may not be equally provocative'.

Typically, the disorder follows occurrences of acute or episodic vestibular or balance related problems, but may also follow non-vestibular insults that threaten balance or cause real or perceived instability (e.g. syncope or presyncope).

Clinical features

Patients with persistent postural-perceptual dizziness present with two key fluctuating or continuous symptoms: (1) a dizzy, not truly vertiginous sensation, with patients reporting that their head is swimming; and/or (2) unsteadiness, such that patients report swaying, rocking or 'jelly legs'.[3] Symptoms are frequently exacerbated in visually complex environments, and during upright posture and head movements. However, a proportion of individuals will report prominent symptoms when still (e.g. sitting, standing or lying in bed) that reflect both a lack of distractors (note that, in such individuals, symptoms are not present

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Table 1. Bárány Society criteria for the diagnosis of persistent postural-perceptual dizziness

A. At least 1 of: dizziness, unsteadiness or non-spinning vertigo; present on most days for 3+ months
– Symptoms last for prolonged periods, but severity may wax & wane
– Symptoms need not be present continuously throughout entire day
B. Persistent symptoms occur without specific provocation, but are exacerbated by:
– Upright posture
– Active or passive motion, irrespective of direction or position
– Exposure to moving visual stimuli or complex visual patterns
C. The disorder is usually precipitated by conditions that cause acute vestibular symptoms or difficulty with balance, but may develop gradually
– When precipitated by an acute or episodic condition, symptoms settle into the pattern of criterion A with trigger resolution, but may be intermittent before later becoming persistent
– When precipitated by a chronic syndrome, symptoms may develop slowly & worsen gradually
D. Symptoms cause significant distress or functional impairment
E. Symptoms are not better accounted for by another disease or disorder

when driving), and an internal expectation of no movement, whereas in fact our bodies are in constant (low level) motion.

Patients often report feelings of disconnectedness from the self and their surroundings (mild depersonalisation and derealisation). Such low-level but mostly persistent dissociative symptoms are very commonly reported by patients with persistent postural-perceptual dizziness, but are also experienced by healthy individuals who are exposed to a strong vestibular stimulus,[4] suggesting such cognitive perceptions are indeed engendered by vestibular symptoms. The character of dissociative symptoms includes a feeling of brain fog or non-specific sensations of disorientation, together with a range of more specific cognitive symptoms that include short-term memory loss, difficulty concentrating and impairments in multitasking (Figure 1).[3]

Persistent postural-perceptual dizziness is triggered by an acute disruption to balance, or a perceived threat to balance, that may be a vestibular or non-vestibular stimulus. Importantly, if there has been a vestibular insult, the development of persistent postural-perceptual dizziness is not attributable to the degree of vestibular dysfunction or failed recovery.[2] Instead, there are premorbid psychological risk and perpetuating factors (see below) that predict the development of persistent postural-perceptual dizziness following the triggering event.[5]

It is worthwhile noting that not all patients who experience prolonged dizziness following a triggering event will go on to develop persistent postural-perceptual dizziness, hence the requirement for symptoms to be present for three months or longer to make a confident diagnosis. Many symptoms of persistent postural-perceptual dizziness are normal adaptations to an acute balance disorder, with a tendency to rely more heavily on vision, a stiffened gait and shorter strides that ensure greater caution.[6] Persistent postural-perceptual dizziness thus represents a failure to re-adapt to a perceived postural threat that no longer exists, hence why this becomes maladaptive or contextually inappropriate.

Risk factors

Regarding the risk factors for development of persistent postural-perceptual dizziness, whilst there are core clinical features, the clinical phenotype, as with many chronic disorders, can vary across individuals. Preceding trait anxiety increases the predisposition to developing persistent postural-perceptual dizziness,[5] together with other personality traits, such as neuroticism and negative illness behaviour. In the context of a destabilising stimulus, be that a fall, near fall or vestibular episode, such psychological factors facilitate the development of a hypervigilant state and introspective self-monitoring.[5] There may be additional physical variables involved in the development and maintenance of persistent postural-perceptual dizziness symptoms, such as lower sensory thresholds with increased sensitivity to stimuli across the range of sensory modalities (vision, vestibular and proprioceptive), which is not fully explained by anxiety.[7–9]

Pathophysiology

Our understanding of the mechanisms that trigger and maintain persistent postural-perceptual dizziness symptoms are beginning to be uncovered, perhaps facilitated by the recognised diagnostic criteria that have helped homogenise this group of individuals. The core pathophysiological mechanism of persistent postural-perceptual dizziness is that of abnormal self-motion processing. Increased self-motion perception, and, thus, heightened instability, may result from a mismatch between expected (efferent) and actual (afferent) motion signals.[10,11] This appears to relate to a reduced tolerance for errors between estimates and sensory inputs.

The generative models used by the central nervous system to actively construct inferences of the causes of sensory inputs, ensures robust internal models that reliably predict self and external motion.[12,13] Any difference between the input observed and that predicted by the generative model and inferred causes gives rise to a prediction error. The central nervous system thus uses predictive coding to reduce surprise and resolve uncertainty about sensory information, reducing the mismatch between unpredicted ('salient') sensations and those predicted under the generative model. This ensures that only unpredicted inputs are further transmitted and analysed at a central level. If we take the example of a patient with persistent postural-perceptual dizziness who is standing 'still', the abnormal processing of seemingly 'unpredicted' postural signals (that unconsciously allow us to maintain stability) may account for abnormal prediction error and the increased perception of sway. Within this framework of predictive coding, the central processing of incoming sensory information is biased by a mismatch resulting from incorrect internal expectations, leading to the perception of impaired posture or balance.

Moreover, patients with persistent postural-perceptual dizziness also have a heightened awareness of, or increased focus

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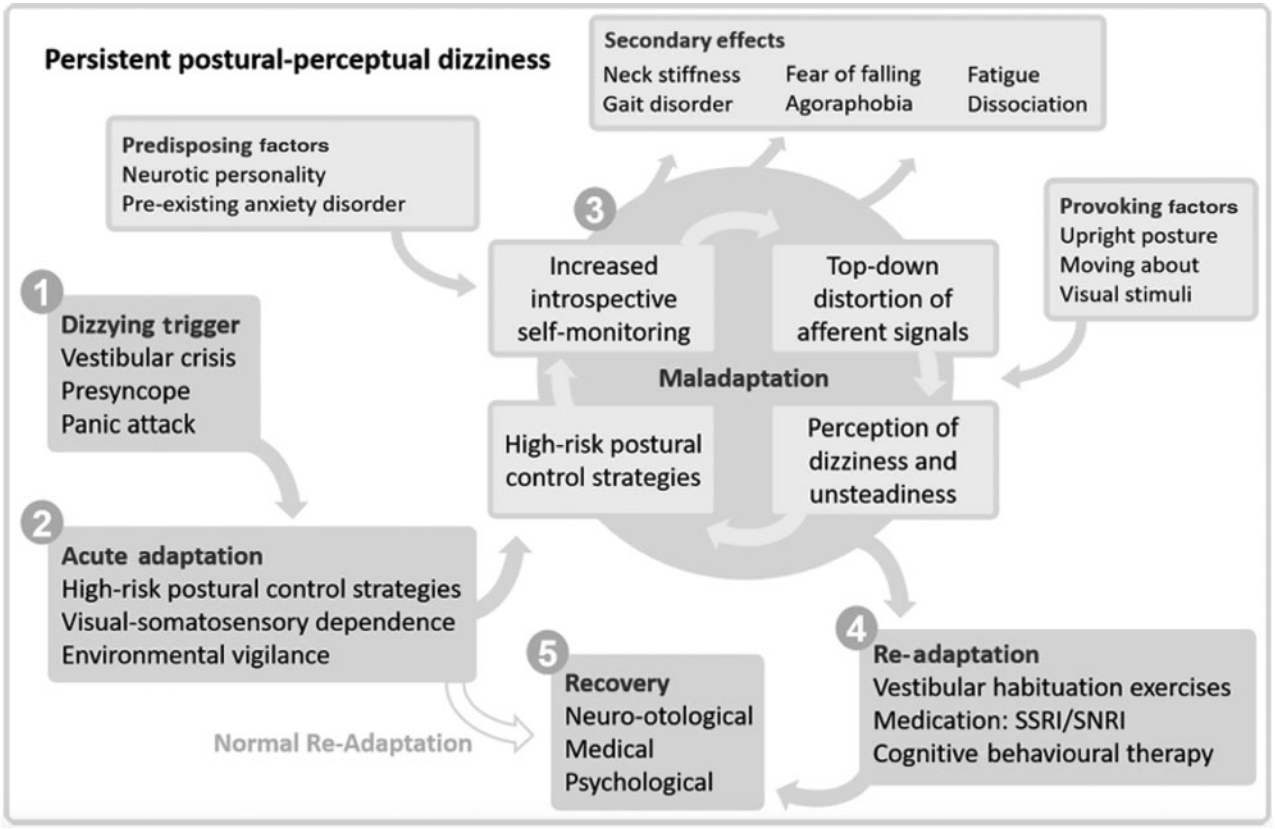

Figure 1. Pathophysiological processes involved in the development and maintenance of persistent postural-perceptual dizziness. The normal physiological reaction to a feeling of dizziness or a postural threat (1) involves activation of motor control strategies (2) that rely less on vestibular information. Upon removal of the postural threat, patients with persistent postural-perceptual dizziness are unable to revert back to normal function, inducing a vicious cycle of maladaptation (3), driven in part by postural hypervigilance and postural anxiety. Somatosensory information about body position is thus amplified and distorted, which in turn produces subjective dizziness and leaves movement control on 'red alert'. Secondary effects like stiffening of gait, phobic avoidance and mental fatigue can develop. The aim of therapy (4) is to re-adapt the system to normal function by reducing anxiety and self-monitoring, habituating to provoking factors, and promoting automatic movement control until recovery (5) is achieved. Adapted from Popkirov *et al*.[3] SSRI = selective serotonin reuptake inhibitors; SNRI = serotonin noradrenaline reuptake inhibitors

of attention on, moving or complex visual stimuli,[9,14,15] commonly termed visually induced dizziness. It is unclear whether this is a pre-existing trait in patients more susceptible to persistent postural-perceptual dizziness[8] or a consequence of the postural hypervigilance, representing a move towards visual dependency for balance. Either way, such sensitivity to visual motion is compounded by the increased sensitivity to self-motion that together give rise to perceived postural instability.

Making a diagnosis

Persistent postural-perceptual dizziness can be considered a neuropsychological disorder, hence substantial work has explored alterations in brain function and structure in these patients.[16] Currently, the diagnostic approach focuses on a careful history of precipitating events, and symptoms that fit the diagnostic criteria for persistent postural-perceptual dizziness. The clinical examination findings are typically normal, as are associated audiovestibular investigation findings.

Visual dependency can also be measured using the dynamic subjective visual vertical test.[17] In the research setting, patients manifest reduced thresholds for detecting visual stimuli to help guide balance, thus promoting the upweighting of visual cues,[18] in line with visual motion discomfort in these patients (perception), and greater sway when faced with concurrent visual motion stimuli (behaviour).

Studies using posturographic measures in persistent postural-perceptual dizziness have been inconsistent in their findings, but overall reveal greater sway associated with conditions that are more posturally demanding (e.g. standing on foam), compared to controls.[19–21] Automated postural and gait parameters in other studies have shown promise as potential diagnostic biomarkers to differentiate patients with peripheral vestibular, cerebellar and persistent postural-perceptual dizziness,[19,22,23] but these instruments are not routinely available. A stiffened ankle strategy with a high frequency of body sway (3.5–8 Hz) has been reported in patients with phobic postural vertigo (a precursor term to persistent postural-perceptual dizziness), related to a tendency to abnormally recruit afferent feedback loops during a quiet stance. Such patients use postural control strategies appropriate for situations that may threaten postural stability, which become pervasive and contextually inappropriate (Figure 1).

More recently, we have shown that it is possible to provide a semi-quantitative measure of postural instability in patients with persistent postural-perceptual dizziness; thus, patients with persistent postural-perceptual dizziness had a two-fold increase in perceptual instability versus (actual) objective sway when compared to patients with bilateral vestibular failure.[24] Indeed, patients with persistent postural-perceptual dizziness tend to over-estimate head roll tilt substantially more than patients with unilateral vestibular hypofunction,[25] suggesting an error of magnitude estimation.[26]

Brain imaging is not currently regarded an essential component in the diagnostic investigation of patients with persistent postural-perceptual dizziness, unless there are features of

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an alternative neurological disorder that cannot confidently be accounted for within a persistent postural-perceptual dizziness framework (e.g. limb weakness, paraesthesia, headaches). However, several studies have explored structural and functional imaging changes in persistent postural-perceptual dizziness, compared to healthy controls.

Functional imaging study findings include increased activity in the visual cortex, correlating with the Dizziness Handicap Inventory score,[27] reduced or altered connectivity between limbic (attention and arousal centres) and vestibular cortical regions,[27] and reduced connectivity between visual and vestibular cortices, frontal regulatory regions and the hippocampi,[28,29] which may account for the impaired spatial orientation and self-motion perception in persistent postural-perceptual dizziness. Others have found reduced perfusion of vestibular regions (e.g. posterior insular cortex),[28–30] and relative hyperperfusion of the cerebellum bilaterally compared to healthy controls,[30] possibly related to increased computational demands on the cerebellum from increased visual attention and for postural control. Structural brain changes have been reported in frontal, temporal, parietal and hippocampal formation and the cerebellum.[31]

Collectively, these studies consistently indicate a reduction in brain structure, function and connectivity within the regions associated with multisensory vestibular processing and spatial cognition in individuals diagnosed with persistent postural-perceptual dizziness. Conversely, an elevation in function and connectivity is observed in the areas responsible for visual processing. Confounding factors, including personality traits (i.e. neuroticism), psychiatric co-morbidities (i.e. anxiety and depression) and triggering factors (i.e. peripheral vestibular lesions), have not however been systematically accounted for in most imaging studies, meaning that any brain changes cannot be exclusively attributed to persistent postural-perceptual dizziness. Importantly, imaging studies only provide association data, not causality.

Towards a targeted treatment

Given its clinical features, it is perhaps best to consider persistent postural-perceptual dizziness as a truly perceptual disorder, with behavioural consequences that may subsequently alter cortical function and structure, the latter being a consequence, not a cause, of the perceptual disorder.[32] Behavioural data are therefore important, not just to understand the basis for persistent postural-perceptual dizziness, or maladaptive strategies employed by patients, but also to design and implement targeted treatments, most likely still involving physical therapy as a core domain, but one that is psychologically informed;[33] such a framework has been termed cognitive physical therapy.[34]

- Persistent postural-perceptual dizziness is a common cause of chronic dizziness, often triggered by an acute event that threatens balance
- Aim to prevent persistent postural-perceptual dizziness with intervention at the earliest possible stage, to avoid development of chronic dizziness symptoms
- In patients with established persistent postural-perceptual dizziness, appropriate outcome measures can help identify and track progress in abnormal perception, postural deficits, function and quality of life
- There is limited but growing evidence for psychologically informed therapies, including vestibular rehabilitation and holistic management strategies

Treatment of persistent postural-perceptual dizziness starts with a clear explanation of the basis for the symptoms, and a careful exploration of the underlying beliefs a patient holds about the cause of the symptoms. A targeted explanation that addresses these beliefs is more likely to result in positive health outcomes. Current treatment approaches draw from the understanding that persistent postural-perceptual dizziness encompasses vestibular, postural, cognitive, emotional and motor control factors, which influence the development and maintenance of symptoms. As such, patients tend to benefit from vestibular rehabilitation, as well as cognitive behavioural therapy, and these are best employed concurrently. Thus, cognitive behavioural therapy techniques should inform and augment physical therapy approaches, and vestibular exercises[33] or relaxation techniques should be integrated into cognitive behavioural therapy programmes.

Antidepressant medications, most notably serotonin noradrenaline reuptake inhibitors and selective serotonin reuptake inhibitors, may play a role as adjuncts to cognitive behavioural therapy,[35] particularly in individuals with strong psychological symptoms (anxiety or depression) and sleep disturbance,[36] but there is insufficient evidence for these treatments in persistent postural-perceptual dizziness when in isolation. Other approaches include non-invasive vagal nerve stimulation,[37] again with limited evidence, and treatment strategies that use virtual reality or mixed reality to help induce a state of embodiment with the aim of trying to alter fixed illness beliefs and traits (Sereda *et al.*, submitted for publication).

Conclusion

Research into the predictors of persistent postural-perceptual dizziness, to enable intervention at the earliest possible stage and perhaps avoid the development of chronic dizziness symptoms, seems a fundamental approach, with an understanding that prevention is better than cure. In patients with established persistent postural-perceptual dizziness, however, the development of biomarkers to track progress and measure the response to therapies needs to incorporate abnormal perception, not just posturography deficits or imaging abnormalities, given the degree of heterogeneity in behavioural adaptations to a perceived postural threat.

Competing interests. None declared

References

1 Murdin L, Schilder AG. Epidemiology of balance symptoms and disorders in the community: a systematic review. *Otol Neurotol* 2015;**36**:387–92
2 Staab JP, Eckhardt-Henn A, Horii A, Jacob R, Strupp M, Brandt T *et al.* Diagnostic criteria for persistent postural-perceptual dizziness (PPPD): consensus document of the committee for the Classification of Vestibular Disorders of the Bárány Society. *J Vestib Res* 2017;**27**:191–208
3 Popkirov S, Staab JP, Stone J. Persistent postural-perceptual dizziness (PPPD): a common, characteristic and treatable cause of chronic dizziness. *Pract Neurol* 2018;**18**:5–13
4 Aranda-Moreno C, Jáuregui-Renaud K. Derealization during utricular stimulation. *J Vestib Res* 2016;**26**:425–31
5 Trinidade A, Harman P, Stone J, Staab JP, Goebel JA. Assessment of potential risk factors for the development of persistent postural-perceptual dizziness: a case-control pilot study. *Front Neurol* 2020;**11**:601883
6 Staab JP. Persistent postural-perceptual dizziness. *Semin Neurol* 2020;**40**:130–7
7 Powell G, Derry-Sumner H, Shelton K, Rushton S, Hedge C, Rajenderkumar D *et al.* Visually-induced dizziness is associated with sensitivity and avoidance across all senses. *J Neurol* 2020;**267**:2260–71
8 Holle D, Schulte-Steinberg B, Wurthmann S, Naegel S, Ayzenberg I, Diener HC *et al.* Persistent postural-perceptual dizziness: a matter of higher, central dysfunction? *PLoS One* 2015;**10**:e0142468

https://doi.org/10.1017/S0022215123002153 Published online by Cambridge University Press

9 Cousins S, Cutfield NJ, Kaski D, Palla A, Seemungal BM, Golding JF *et al.* Visual dependency and dizziness after vestibular neuritis. *PLoS One* 2014;**9**:e105426
10 Dieterich M, Staab JP, Brandt T. Functional (psychogenic) dizziness. *Handb Clin Neurol* 2016;**139**:447–68
11 Wuehr M, Pradhan C, Novozhilov S, Krafczyk S, Brandt T, Jahn K *et al.* Inadequate interaction between open- and closed-loop postural control in phobic postural vertigo. *J Neurol* 2013;**260**:1314–23
12 Klingner CM, Axer H, Brodoehl S, Witte OW. Vertigo and the processing of vestibular information: a review in the context of predictive coding. *Neurosci Biobehav Rev* 2016;**71**:379–87
13 Friston K. A theory of cortical responses. *Philos Trans R Soc Lond B Biol Sci* 2005;**360**:815–36
14 Jacob RG, Redfern MS, Furman JM. Space and motion discomfort and abnormal balance control in patients with anxiety disorders. *J Neurol Neurosurg Psychiatry* 2009;**80**:74–8
15 Bronstein AM. Visual vertigo syndrome: clinical and posturography findings. *J Neurol Neurosurg Psychiatry* 1995;**59**:472–6
16 Indovina I, Passamonti L, Mucci V, Chiarella G, Lacquaniti F, Staab JP. Brain correlates of persistent postural-perceptual dizziness: a review of neuroimaging studies. *J Clin Med* 2021;**10**:4274
17 Ulozienė I, Totilienė M, Paulauskas A, Blažauskas T, Marozas V, Kaski D *et al.* Subjective visual vertical assessment with mobile virtual reality system. *Medicina* (*Kaunas*) 2017;**53**:394–402
18 Vermehren M, Carpenter MG. Virtual postural threat facilitates the detection of visual stimuli. *Neurosci Lett* 2020;**736**:135298
19 Brandt T, Strupp M, Novozhilov S, Krafczyk S. Artificial neural network posturography detects the transition of vestibular neuritis to phobic postural vertigo. *J Neurol* 2012;**259**:182–4
20 McCaslin DL, Shepard NT, Hollman JH, Staab JP. Characterization of postural sway in patients with persistent postural-perceptual dizziness (PPPD) using wearable motion sensors. *Otol Neurotol* 2022;**43**:e243–51
21 Söhsten E, Bittar RS, Staab JP. Posturographic profile of patients with persistent postural-perceptual dizziness on the sensory organization test. *J Vestib Res* 2016;**26**:319–26
22 Krafczyk S, Schlamp V, Dieterich M, Haberhauer P, Brandt T. Increased body sway at 3.5-8 Hz in patients with phobic postural vertigo. *Neurosci Lett* 1999;**259**:149–52
23 Schniepp R, Möhwald K, Wuehr M. Clinical and automated gait analysis in patients with vestibular, cerebellar, and functional gait disorders: perspectives and limitations. *J Neurol* 2019;**266**:118–22
24 San Pedro Murillo E, Bancroft MJ, Koohi N, Castro P, Kaski D. Postural misperception: a biomarker for persistent postural perceptual dizziness. *J Neurol Neurosurg Psychiatry* 2022;**94**:165–6
25 Yagi C, Morita Y, Kitazawa M, Nonomura Y, Yamagishi T, Ohshima S *et al.* Head roll-tilt subjective visual vertical test in the diagnosis of persistent postural-perceptual dizziness. *Otol Neurotol* 2021;**42**:1618–24
26 Arshad Q, Saman Y, Sharif M, Kaski D, Staab JP. Magnitude estimates orchestrate hierarchal construction of context-dependent representational maps for vestibular space and time: theoretical implications for functional dizziness. *Front Integr Neurosci* 2022;**15**:806940
27 Riccelli R, Passamonti L, Toschi N, Nigro S, Chiarella G, Petrolo C *et al.* Altered insular and occipital responses to simulated vertical self-motion in patients with persistent postural-perceptual dizziness. *Front Neurol* 2017;**8**:529
28 Li K, Si L, Cui B, Ling X, Shen B, Yang X. Altered intra- and inter-network functional connectivity in patients with persistent postural-perceptual dizziness. *Neuroimage Clin* 2020;**26**:102216
29 Li K, Si L, Cui B, Ling X, Shen B, Yang X. Altered spontaneous functional activity of the right precuneus and cuneus in patients with persistent postural-perceptual dizziness. *Brain Imaging Behav* 2020;**14**:2176–86
30 Na S, Im JJ, Jeong H, Lee ES, Lee TK, Chung YA *et al.* Cerebral perfusion abnormalities in patients with persistent postural-perceptual dizziness (PPPD): a SPECT study. *J Neural Transm* (*Vienna*) 2019;**126**:123–9
31 Wurthmann S, Naegel S, Schulte Steinberg B, Theysohn N, Diener HC, Kleinschnitz C *et al.* Cerebral gray matter changes in persistent postural perceptual dizziness. *J Psychosom Res* 2017;**103**:95–101
32 Castro P, Bancroft MJ, Arshad Q, Kaski D. Persistent postural-perceptual dizziness (PPPD) from brain imaging to behaviour and perception. *Brain Sci* 2022;**12**:753–64
33 Herdman D, Norton S, Murdin L, Frost K, Pavlou M, Moss-Morris R. The INVEST trial: a randomised feasibility trial of psychologically informed vestibular rehabilitation versus current gold standard physiotherapy for people with persistent postural perceptual dizziness. *J Neurol* 2022:**269**:4753–63
34 Castro P, Vadera S, Bancroft MJ, Buttell J, Kaski D. Case report: acute onset fear of falling and treatment with "cognitive physical therapy". *Front Neurol* 2021;**12**:707840
35 Yu YC, Xue H, Zhang YX, Zhou J. Cognitive behavior therapy as augmentation for sertraline in treating patients with persistent postural-perceptual dizziness. *Biomed Res Int* 2018;**2018**:8518631
36 Staab JP, Ruckenstein MJ, Solomon D, Shepard NT. Serotonin reuptake inhibitors for dizziness with psychiatric symptoms. *Arch Otolaryngol Head Neck Surg* 2002;**128**:554–60
37 Eren OE, Filippopulos F, Sönmez K, Möhwald K, Straube A, Schöberl F. Non-invasive vagus nerve stimulation significantly improves quality of life in patients with persistent postural-perceptual dizziness. *J Neurol* 2018;**265**:63–9

https://doi.org/10.1017/S0022215123002153 Published online by Cambridge University Press