

TOPICS IN VESTIBULAR PHYSICAL THERAPY

VESTIBULAR REHABILITATION SIG
APTA & Academy of Neurologic Physical Therapy



VESTIBULAR MIGRAINE AND PERSISTENT POSTURAL PERCEPTUAL DIZZINESS

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Message from the Chair

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Chronic dizziness and Persistent Postural Perceptual Dizziness (PPPD) are some of the most challenging vestibular conditions that physical therapists manage. I immensely enjoy working with individuals with PPPD for this challenge and know that when we improve their symptoms, we will change their lives. Management of these conditions is rapidly progressing, and I look forward to this issue of Topics in Vestibular Physical Therapy to give us more insight into this complex and rewarding population.

While managing patients with only PPPD is challenging enough, as you all know, we often see PPPD occurring with other vestibular diagnoses, such as Vestibular Migraine. When I work with individuals with these dual diagnoses, I rely heavily on my differential diagnosis skills. The basics are often not so basic, especially when you must interpret all of the findings together for clinical decision-making. Vestibular physical therapists put together a detailed history, ocular motor exam, and gait/balance examination, along with vestibular

functional exam results and Barany Society Diagnostic Guidelines. We really are practicing at the top of our education and licenses. I hope that in the future, these skills will be widely recognized and appreciated as a formal specialization.

The Vestibular Rehabilitation Special Interest Group (SIG) has a new Chair Elect, Jacob McPherson. Jake is an Assistant Professor at the University of Buffalo Department of Physical Therapy. Jake has served as the Vestibular Rehabilitation SIG Advocacy/Reimbursement representative for the past few years. I am confident he will make an excellent Chair, and I look forward to his ideas about how we can improve this organization.

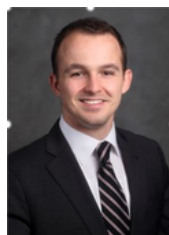
We are an organization only as strong as our leadership team and members! I am thankful for the tremendous Vestibular Rehabilitation Special Interest Group leadership team that volunteers their time to support our initiatives.

Topics in Vestibular Rehabilitation 2024 Best Article of the Year Award

LOW TO MODERATE
 INTENSITY VESTIBULAR
 PHYSICAL THERAPY IMPROVES
 OUTCOMES IN ACTIVE
 CEREBELLAR HEMATOMA
 CAUSED BY RARE VENOUS
 ANOMALY: A CASE REPORT



JANE ALKHAZOV, PT, DPT
 Board-certified specialist in neurologic rehabilitation



BRADLEY MILLER, PT, DPT, CSCS

A Clinical Introduction and Overview of Physical Therapy Phenotyping in the Context of Vestibular Migraine and Persistent Postural Perceptual Dizziness

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Introduction

Although the concept of phenotypes is over 100 years old in the science of genetics,(1) the application to phenotypes for treatment decisions in the larger field of physical therapy has only recently been proposed.(2) In vestibular physical therapy (VPT), individualized and supervised care is recommended over generic care,(3,4) which is consistent with the overall idea that individual variation rather than disease diagnosis drives impairments and should be the focus of rehabilitation interventions. Not surprisingly, individuals with vestibular disorders present with highly variable symptom presentations across a disease model. Canalithiasis and cupulolithiasis (two distinct forms of benign paroxysmal positional vertigo(5)) manifest with different symptom presentations often conceptualized based on symptom duration. Further, the vestibular system nonlinearly represents rotational velocity (6–8) and individuals with vestibular hypofunction may demonstrate frequency specificity to their pathology and associated impairments, i.e., impaired low frequency responses and preserved high frequency responses.(9,10)

In this volume of Topics in Vestibular Physical Therapy, we focus our conversation about phenotypes in vestibular rehabilitation as applied to two conditions: vestibular migraine (11–13) and persistent postural perceptual dizziness.(14–16) Although diagnostically distinct, there is considerable overlap in symptom presentation

between them. Additionally, traditional vestibular function testing is unlikely to identify vestibular pathology specific to vestibular migraine (VM) or persistent postural perceptual dizziness (PPPD), except in the case of unilateral vestibular hypofunction as the precipitating factor for PPPD. As both conditions rely on “not better accounted for by another... diagnosis” as part of the diagnosis,(12,14) these two conditions are ripe for characterizing in a more precise way using phenotypes. Both of these conditions manifest with abnormal integration/perception of sensory (and motor) neural signals due to alterations in sensory-motor and attentional networks within the brain leading to signs and symptoms of vertigo, non-rotational dizziness, and imbalance.(17–22) The challenge before us is how to individualize VPT treatments for individuals with similar but distinct symptom and impairment presentations with identical diagnoses. The following series of case reports provide framework for clinical phenotype contextualization when treating vestibular migraine and persistent postural perceptual dizziness.

Evidence for Phenotypes in VM

As will be presented in greater detail in the case reports, VM has specific diagnostic criteria. (12) Briefly, individuals must have a history of migraine and at least 5 episodes of moderate (or greater) vestibular symptoms lasting 5-72 hours, and at least 50% of episodes must have either headache, visual and/or sound sensitivity, or auras.(12) Individuals with VM commonly may present with sensitization to self-motion perception and roll-tilt

in particular,(23,24), which may reflect alterations in the attentional network or visual vestibular interactions. (21,25) With the proliferation of genetic testing to understand biological determinants of disease, the growing body of evidence supporting genetic susceptibility for vestibular dysfunction is not surprising.(26–31) Interestingly, even within the same family showing the same genetic mutation, there is both commonality and variability in the presenting signs and symptoms of vestibular migraine.(26) Four women representing three generations all reported vertigo, and nausea with headache symptoms that lasted hours, but two of them experienced headaches that lasted days. Three of them experienced interictal gaze evoked nystagmus. Two also experienced hemiplegia associated with their VM episodes and two of them reported light sensitivity. The wide variation in symptom presentation across these women despite a common genetic cause highlights the need for VPT providers to be aware of expressive variation in symptoms (phenotypes) within a condition.(32) A second study used genome wide genetic analyses to determine whether migraine with vertigo had a distinct genetic locus from migraine with motion sensitivity.(33) Thus, those two migraine phenotypes (vertigo vs. motion sensitivity) have different genetic presentations, but also the epigenetic expression of those genotypes are heavily influenced by the environment including movement experiences (exercise).(34)

Support for the existence of phenotypes in VM has recently been demonstrated by several studies,(28,35) driven by the highly variable symptom presentation and treatment response of individuals with VM.(36–39) In fact, in 2018 Teggi and colleagues reported that individuals with VM differed both in headache duration (less than 2 hours to >7 days) and were highly variable with endorsements of symptoms during childhood (motion sensitivity (> 50%) > cyclic vomiting > stomach pain > episodic vertigo > torticollis).(35) Others have examined associated symptoms for individuals with VM (100% report headache and vertigo) and more than 1/3 also report nausea, tinnitus, aural fullness.(38,40,41) Leveraging cluster analyses and large datasets, Teggi

and colleagues revisited the concept of VM phenotypes to explore whether distinct clusters existed across individuals.(28) Based on the symptoms reported in a study where all subjects (n = 244) met the Bárány criteria for VM,(12) five distinct clusters were identified based on four symptoms (age of vertigo onset, duration of vertigo attacks, presence of migraine headache, and auditory symptoms during vertigo). Interestingly, individuals in two clusters no longer endorsed current migraine headaches.(28) Only three clusters included cochlear symptoms, and four of the clusters included symptoms lasting less than 10 hours while the last group averaged 54 hours of vertigo. The vastly different symptom presentation suggests that VPT providers should be tailoring interventions to the individual, thus respecting their phenotypic presentation. For example, habituation dosages for an individual with VM without headaches might have higher frequency and intensity than for an individual who still experiences headaches, since habituation exercises may trigger a migraine headache.(40)

VM events are also often triggered by things like stress, bright lights, weather changes, sleep deprivation, or food.(40,42) Conservative management for VM often includes education related to trigger avoidance (as in the VM case by Rozestraten & Anson), which may be easier in the case of food triggers and harder in the context of stress or weather changes.(43) Individuals with VM may also experience comorbid psychiatric conditions such as depression, anxiety, sleep disturbance, or psychosis.(40,44–46) The VPT approach to an individual with VM plus psychiatric conditions must be different than an individual with VM without psychiatric conditions, and in many cases should include referral to the appropriate psychiatric/mental health provider. Both anxiety and depression are associated with symptom intensity and presence of other vestibular disorders, but only anxiety was also associated with frequency of VM events. (45,47–50)

Sharon and colleagues developed a questionnaire to identify VM,(36,51) but further analyses of individual

questions demonstrated six factors or phenotypes. Specific symptom-driven factors included cognition, anxiety, feeling overwhelmed, disequilibrium, motion sensitivity, and headache equivalents. Which of those factors dominates an individual's presentation should be considered in prescribing interventions. Thus, VPT providers must also be sensitive to the possibility that an individual's phenotypic expression of their disease state is likely to evolve over time both as the individual alters how they behave and interact with the environment and as they recover.(32)

Evidence for Phenotypes in PPPD

Persistent postural perceptual dizziness shares many symptoms with VM,(16,52–54) but distinctions exist that necessitate recognition and a different treatment approach.(55,56) In fact, one of the case studies in this series highlights an individual who developed PPPD after a VM episode.(57) Alterations in neural networks, abnormal sensory perception, and central sensitizations result in persistent maladaptive processes leading to hypervigilance and higher risk postural strategies in individuals with PPPD.(52,53,58–63) In the case of PPPD, it is highly probable that a reduction in movement exposure due to over-emphasizing hypervigilant behaviors promotes the phenotypic expression of central sensitization and heightened self-motion perception.(34) Fortunately exercise regulates gene expression, thus VPT with psychosocial and/or pharmacological interventions may be the impetus necessary to reverse the maladaptive processes in PPPD using epigenetics.(34,64) Key in the diagnosis of PPPD is the presence of symptoms (dizziness, unsteadiness, or non-spinning vertigo) on most days for at least three months.(14,16) Together these symptoms manifest without specific triggers or precipitating events, and are worsened by active or passive motion, being upright, and exposure to complex visual environments. Although the symptoms may wax and wane, they are always present to some degree. Initial symptom presentation also should be tied to a distinct acute event or a persistence of chronic symptoms. Despite commonalities in symptom

presentation leading to the diagnosis of PPPD, there is considerable variability in symptom presentation across individuals leading to recent discussions about sub-types.(65,66)

A major phenotype for PPPD includes comorbid anxiety,(15) and this phenotype is more common in younger adults.(67) Importantly, individuals with PPPD have a distinct neural signature compared to individuals with anxiety in the supramarginal and supra-temporal gyri when viewing images that provoke dizziness or anxiety.(58) Older adults with PPPD tend to have more peripheral vestibular dysfunction and higher sway velocity. These distinct age-related differences in symptoms and balance performance suggest an age-related phenotype. Distinct from both anxiety and age, others have recently demonstrated that based on answers to the Niigata PPPD questionnaire three subtypes or phenotypes can be identified.(65,68) The most common subtype was visual stimuli (45%) followed by passive movement (36%) and least common was active movement (19%); although older adults were more likely to experience more symptoms with active movement.

Characterizing Phenotypes in VM/PPPD

As VPT providers move to increased emphasis on precision rehabilitation,(34) conceptual ideas about pathology or disease specific phenotypes must give way to practical methods capable of quantifying those phenotypes. Although most studies that demonstrate clusters or distinct factors which are interpreted here as phenotypes rely on relatively large datasets to accommodate the statistical methods, two questionnaires exist that reported which individual items load into each phenotype. Importantly for the diagnoses considered here, there is a validated scale for each one. For VM, the Vestibular Migraine Patient Assessment Tool and Handicap Inventory (VM-PATHI) has excellent psychometrics and discriminates healthy controls (mean score 9.6 +/- 8.5) from individuals with VM (mean score 42.5 +/- 16.1).(36) This 25 item questionnaire can be completed online or manually on paper but either method requires the

provider to manually calculate scores for each factor by summing the scores for each question as indicated in Table 1.

Table 1. Groupings of VM-PATHI questions to create phenotype sub-scores. Adapted from Sharon et al.(36) Summing the item scores for the specified questions will provide the sub-score.

Factor (Phenotype)	Question Numbers
Cognition	25, 20, 17, 14, 13, 4
Anxiety	23, 6, 5
Feeling overwhelmed	24, 18, 16, 15, 9
Disequilibrium	12, 11, 7, 2, 1
Motion sensitivity	21, 10, 3
Headache equivalents	22, 18 8

For PPPD the optimal patient-reported outcome (PRO) to capture patient's phenotype is the Niigata PPPD questionnaire.(66) For this validated 12-item questionnaire, a score of 27 or greater out of 72 discriminates between healthy controls and individuals with PPPD (sensitivity = 0.70, specificity = 0.68). Each of the sub-scales also significantly discriminates between individuals with PPPD and healthy controls using a sub-score of 9. Interestingly, in this original development cohort, there was no group difference in the Hospital Anxiety and Depression Scale or the visual analog scale rating for somatic distress. Each sub-score consists of four questions, grouped around the three required symptoms upright/walking worsens symptoms, movement worsens symptoms, and visual stimulation worsens symptoms. See Table 2 for specific details.

Finally, it is outside the Physical Therapist scope of practice to diagnose psychiatric conditions, but it would be very useful for VPT providers to screen individuals

with VM or PPPD for anxiety so that appropriate referrals can be made or a change in treatment approach such as the psychologically informed VPT similar to the INVEST trial which is described below. (55,56) There are several available screening tools capable of suggesting the patient may have higher than normal anxiety or depression, but the shortest screen for both is the PHQ-4. Summing the first 2 questions provides an anxiety score and summing the last 2 questions provides a depression score. Scores higher than 3 for either category would be considered a positive screen.(69) There are several other PROs that capture anxiety or depression that could also be used including the Positive Affect Negative Affect Scale,(70) The Hospital Anxiety and Depression Scale, (71) or the General Anxiety Disorder-7 questionnaire, (72) which was used in the following PPPD case. This non-exhaustive list of mental health PROs provides options for VPT providers who want to capture mental health status for consideration of psychosocial epigenetic factors impacting VPT goals and potential. (64)

Table 2. Groupings of Niigata PPPD questions to create phenotype sub-scores. Adapted from Yagi et al.(65,66) Summing the item scores for the specified questions will provide the sub-score.

Factor (Phenotype)	Question Numbers
Upright/walking	3, 6, 7, 11
Movement	1, 5, 9, 12
Visual stimulation	2, 4, 8, 10

The following case reports highlight different approaches to patient management for individuals with VM or PPPD, not always mutually exclusive. The authors provide a clinically useful framework for how the patient's phenotype informed the VPT provider's approach to exercise selection, dosage, and progression even when following a more rigid research protocol. Future studies should expand on the concept

of phenotyping, which may have even greater relevance for individuals who experience similar symptoms to VM or PPPD but do not meet the strict diagnostic criteria. (57,73–75) Another future direction for phenotyping in vestibular rehabilitation is to determine whether other PROs, such as the Visual Vertigo Analog Scale(76,77) which distinguishes between healthy controls (lowest scores), individuals with VM, and individuals with PPPD (highest scores)(78), also identify similar phenotypes. And finally, including measures of premorbid and since-onset movement profiles may be highly relevant in both explaining and addressing phenotypic changes related to movement or exercise.(34)

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Vestibular Migraine Hiding a Vestibular Hypofunction: A Case Report

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ABSTRACT

Introduction: Vestibular migraine (VM) is a highly prevalent and disabling condition affecting women at a higher rate than men, impacting >1-3% of the general population, and may be underdiagnosed.

Individuals with VM experience a wide range of symptoms that last from seconds to days. A combined approach of pharmaceutical management, lifestyle modification, and vestibular physical therapy (VPT) is beneficial for individuals with VM. **Case**

Description: A 35-year-old non-binary adult referred to outpatient VPT from the emergency department for management of acute dizziness. They were diagnosed with VM with an across-the-board phenotype presentation including impairments in: gaze instability, cognition, emotion, and sense of being overwhelmed, disequilibrium and central audio vestibular disturbances, anxiety, motion sensitivity, and headache equivalents. They scored below the normal range on standing and walking balance tests, gaze stability testing, and initially reported severe impairment on both the Visual Vertigo Analog Scale and Dizziness Handicap Inventory. **Interventions:** Interventions were selected and modified based on their phenotypic presentation. Early VPT focus was education, autonomic regulation, mild habituation exercises, gaze stabilization, and general movement and wellness exercises. Later VPT focus was on habituation to both visual and self-motion, standing and walking balance activities, while continuing general movement and wellness exercises.

Outcomes: At visit 9, about four months after VPT onset, they demonstrated normal balance and reduced frequency/severity of dizziness, headaches, and other VM symptoms. Overall, they demonstrated

improvements in all physical performance and patient-reported outcomes with most scores normal for age. This case highlights successful application of a multimodal approach to VM management with individualized interventions based on an evolving phenotypic presentation.

INTRODUCTION

Migraine is one of the most prevalent and the second most disabling medical conditions worldwide.(1,2) In the US around 12% of adults have migraines(2) and lifetime prevalence may be as high as 16%.(3) Migraines can occur at any age and are most prevalent in those 25-55 years of age with a rise in prevalence through early adulthood dropping off in middle age.(4) Women more frequently experience migraines compared to men at rates ranging from 1.5:1 to 6:1.(2,5-7) Vertigo, dizziness, or imbalance occur during at least one migraine attack for 30-50% of individuals.(8) Vestibular migraine (VM) is a condition that affects >1-3% of the general population, and is likely underdiagnosed. (3,7,9-12) The Bárány Society and the International Headache Society released an updated consensus document on the diagnostic criteria of definitive and probable VM to clarify diagnostic accuracy.(13)

Rates of VM were higher in specialized clinics - as high as 10% in dizziness clinics and 9% in migraine clinics.(9) Rates of VM diagnosis increased from 1.8% to 20.2% after referral to and evaluation at a specialized neuro-otology clinic.(12) Of the 11.9% of participants in the 2008 NHIS balance and dizziness supplement (n = 21,781) who reported dizziness, 23.4% met criteria for VM, but only 10% of those meeting criteria for VM had been previously diagnosed.(7)

VM may present with symptom timing ranging from five minutes to three days of spontaneous, positional, visual/motion provoked dizziness or vertigo, postural symptoms including unsteadiness and directional pulsion, photophobia and phonophobia, nausea, and aural symptoms that may be with or without headache.(6,9,14) The most common interictal symptoms include persistent dizziness, visual and/or head motion-induced vertigo.(14) A systematic review found the most frequently reported precipitating factors (triggers) to be stress 58%, auditory stimulation 56%, fatigue 43%, fasting 44%, change in hormone levels (women only) 44%, sleep deprivation/irregularities 43%, weather changes 39%, over-stimulating visual environments 38%, olfactory stimulation 38%, and alcohol consumption 27%.(15) Symptoms of VM can evolve over time, with some individuals experiencing vestibular symptoms around 15 years after the onset of migraines, while another study showed 57.3% had migraines and 61.1% had motion sickness prior to vestibular symptoms. (6,14,16) Compared with healthy controls, individuals with migraine have a higher incidence of motion sickness,(17) and a history of motion sickness as a child.(18) The variable and evolving presentation of VM suggests that phenotyping in VM may be useful to guide individualized management.(14,19,20)

CASE STUDY PRESENTATION

History

A 35-year-old non-binary adult (birth-sex: female) presented to the Emergency Department (ED) after five days of dizziness and imbalance with brief spontaneous and motion-provoked vertigo, which was exacerbated by changes in position and head movement. Their symptoms were self-reported as “being on an elevator that suddenly goes sideways” or “sloshing in their head” accompanied by nausea. Additional symptoms at the time of initial presentation included pressure in the head, photophobia, phonophobia, and “difficulty tracking and processing visual information.” At onset they denied

any hearing change, tinnitus, emesis, double or blurry vision. They reported a history of motion sensitivity with driving and flying, and headaches described as stabbing behind the eyes or right side of the head, pressure in the sinuses and across the forehead. The patient denied a prior diagnosis of migraines. Past medical history included anxiety, depression, ADHD, hypermobility syndrome, and Lyme disease. A standard neurological exam was normal including intact limb coordination, narrow based gait, 2+ reflexes throughout upper and lower extremities, normal upper and lower extremity strength, intact light touch, normal cranial nerves II-XII function, full extraocular movements, no nystagmus in room light, and negative Dix-Hallpike testing (nystagmus or vertigo) but subjective complaint of non-rotary dizziness.(21) They had negative imaging for stroke, including computed tomography scan and magnetic resonance imaging. They were treated with scopolamine, ondansetron, Benadryl, and Antivert 25 mg without relief and admitted for observation. Two days later, they were discharged home with a referral to outpatient vestibular physical therapy (VPT).

Patient presented to outpatient VPT four weeks after presenting to the ED. At that time, they reported spontaneous episodes of perceived self-motion described as “being on a boat, sloshing in their head, or being on an elevator moving sideways” lasting typically hours – days, like the initial symptom timing. During these episodes, they reported fewer symptoms when being still, lying down, or sitting, and increased symptoms of dizziness and aberrant self-motion perception during movement such as bed mobility or movement of their head. Movement-related symptom increases were more transient, lasting seconds to minutes. They continued to report visual changes including “difficulty tracking and processing visual information,” oscillopsia with head movements, and sensitivity to light. They also reported a temporal synchrony between light and sound sensitivity, sinus pressure, and headache (stabbing behind the eyes, across the forehead, or

over the right side of the head or ear). Outside of these episodes, they experienced headache resolution but continued to experience spontaneous yet episodic dizziness, light and sound sensitivity, and reduced but still present visual and motion-provoked dizziness, imbalance, trouble focusing, and oscillopsia with movement. They also endorsed intermittent ear pain and pressure temporally associated with their migrainous features.

Physical Therapy Examination

No spontaneous nystagmus or gaze-evoked nystagmus in room light, intact convergence with near point convergence at 6 cm, pursuit was smooth with full conjugate gaze, fast and accurate horizontal and vertical saccades, intact vestibulo-ocular reflex cancellation, left and right horizontal head impulses without overt refixation saccades. Positional testing was negative for nystagmus or vertigo in the Roll test and right Dix-Hallpike, but the left Dix-Hallpike was positive for dizziness and persistent low amplitude left torsional nystagmus without a vertical component. While in the left Dix-Hallpike position, left lateral gaze mildly enhanced the torsional component of nystagmus but right lateral gaze did not alter the torsional nystagmus. The patient completed patient-reported and physical performance outcome measures. They scored normally on conditions 1-3, but failed condition 4 (eyes closed on foam) of the modified Clinical Test of Sensory Integration in Balance (mCTSIB) indicating poor sensory integration of vestibular input.(22) The patient scored a 16/30 on the Functional Gait Assessment (FGA), well below age norms,(23) and consistent with increased fall risk in older cohorts.(24) On the clinical Dynamic Visual Acuity Test (DVA) the patient demonstrated a 4-line difference between static and dynamic acuity, indicating impaired gaze stability.(25,26) On the Dizziness Handicap Inventory (DHI) the patient scored a 60/100, indicating severe impairment.(27) On the Visual Vertigo Analog Scale (VVAS) the patient scored 57.7. Higher scores indicate more severe symptoms. (28-30)

Based on history and examination, this patient presented with an across-the-board VM clinical phenotype impacting: cognition, emotion, and sense of being overwhelmed (DHI score), disequilibrium,, and central audio vestibular disturbances (abnormal mCTSIB, DVA, FGA scores), anxiety, motion sensitivity (VVAS score), and headache equivalents which were used to guide individualized treatment. (19)

Management: After a treatment diagnosis of VM was established, the patient's primary care physician (PCP) was consulted regarding the diagnosis of VM and referral to a specialized headache clinic for a trial of anti-migraine medications. This step was specifically to address the headache equivalent aspect of their phenotype, as overall progress was expected to be worse if the headache equivalents were not addressed. Their anti-migraine medication management started with Elavil (amitriptyline) for daily use and Imitrex (Sumatriptan) as a rescue medication. We followed a "Refer and Treat" model in this case, as some patients have improved outcomes with a combination of VPT and anti-migraine medications,(31) and we were confident there was no serious central nervous system abnormality based on prior imaging.

VPT intervention selection was based on impairments and their presenting phenotype identified during the subjective, exam, and testing portions of their evaluation. Initial intervention strategies emphasized education on lifestyle modifications targeting precipitating factors associated with mitigating migraine attacks, including optimal sleep hygiene, avoiding known food and drink triggers, eating regular meals, minimizing highly processed foods, proper hydration, and regular physical activity.(32,33) This emphasis targeted the headache equivalent and anxiety components of their across-the-board phenotype.

Initially, VPT visits were weekly, with a heavy emphasis on education and initiating gaze stability, habituation exercises, and general movement and wellness exercises to address emotion and sense of

being overwhelmed, disequilibrium and central audio vestibular disturbance components of the phenotype. Patients with VM when treated with a combination of strengthening and stretching, gaze stabilization, habituation, vestibular compensation, balance, gait, and sensory re-weighting exercises showed improved headaches, DHI scores, and dizziness frequency.(34) Additionally, whole body strengthening and cardiovascular exercise reduce migraine burden, dizziness severity, vertigo frequency, anxiety and depression in individuals with VM.(33,35–37)

Lifestyle modifications, per patient report, especially sleep and diet changes, combined with medication management, yielded significant improvements in migraine headaches, spontaneous vertigo frequency and intensity within a month. As the headache equivalent became less overt and tolerance to gait, gaze stability, and habituation tasks improved, VPT interventions were progressed in frequency, volume, and postural demand. Additionally, optokinetic exposure and cognitive challenges were progressed, see Table 1. They rapidly improved with static balance and vestibular sensory integration, especially on the foam eyes closed condition of the mCTSIB about 5 weeks after initial evaluation. However, the disequilibrium and central audio vestibular disturbances components of their phenotype continued to highlight deficits in dynamic balance. FGA scores did not reach age norms until 4 months after initial evaluation. This may be akin to faster static central compensations after an acute vestibular insult compared to slower dynamic compensation.

Referral for video nystagmography (VNG) and Sensory Organization Test (SOT) was made at the third visit after discussion on the findings of their positional testing completed at the 1st visit and negative results from canalith repositioning maneuvers at the second visit (incorporated primarily for habituation). This referral decision was also informed by slower-than-expected clinical progression at that point and the need to further evaluate positional nystagmus that appeared inconsistent with benign paroxysmal positional vertigo (BPPV), see Figures 1 and 2.(38) Audiometric testing was normal bilaterally. Significant findings from VNG testing included a 40% caloric weakness of the left ear (Figure 3), >3 beats of left beating post headshake nystagmus, low intensity (3 degrees/second) left beating nystagmus in head left position (Figure 1) and the left Dix-Hallpike position (Figure 2). These findings supported the interpretation of a central positional nystagmus, such as can be seen with VM.(39)

The patient had a five-week absence from VPT, after the 5th visit, due to undergoing a right shoulder capsulorrhaphy for chronic instability and dislocations. The imposed time off work gradually lessened symptom burden in the following phenotype components: cognition, emotion and sense of being overwhelmed, disequilibrium and central audio vestibular disturbances, anxiety, and motion sensitivity. Ultimately, the imposed time off work removed a significant stressor and allowed them to focus on lifestyle modifications, personal trigger identification and management, with renewed

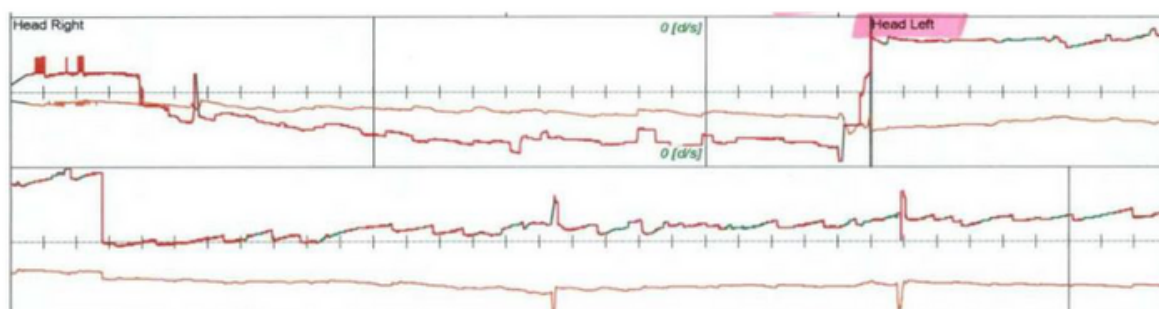


Figure 1: Static positional testing showed persistent left beating (3 d/s) in supine head left position. A mild up- beating nystagmus (4 d/s) was noted in supine with head turned to the right and center (not shown).

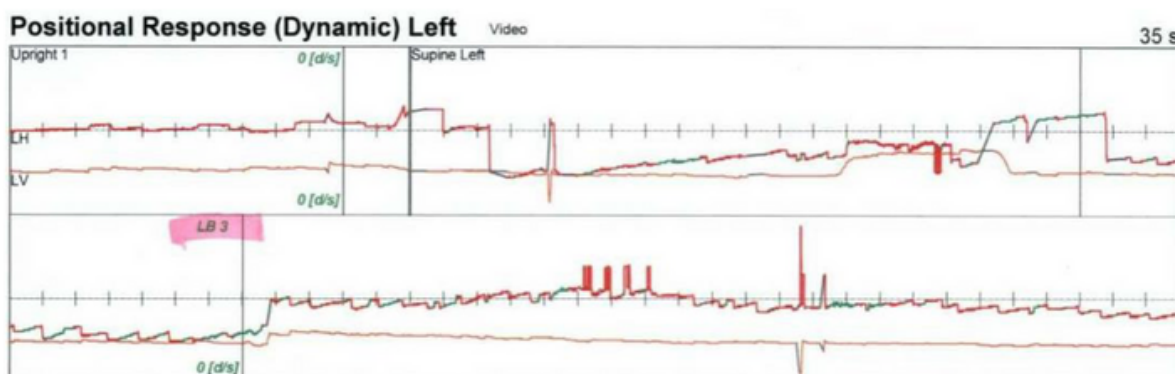


Figure 2: Dynamic positions in left Dix-Hallpike position revealed persistent, low-grade, left-beating nystagmus (3 d/s).

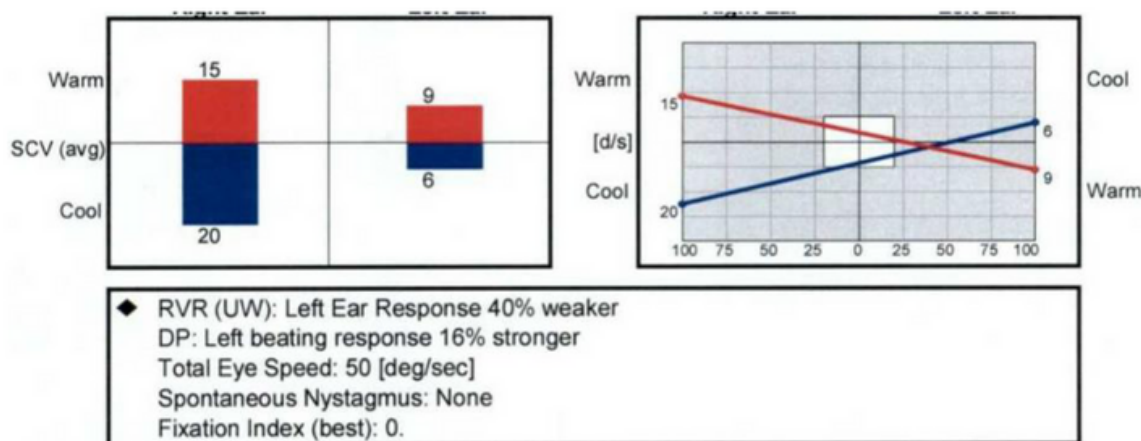


Figure 3: Bithermal caloric air irrigations revealed a moderate left peripheral vestibular loss (40% interaural difference). Fixation suppression was normal.

progression of habituation and gaze stability exercises.

DISCUSSION

Final outcomes were very positive for this patient, with normalization or improvement to the mild impairment range for all patient-reported and performance-based measures. Notably, their DVA improved to a two-line difference by the 9th visit, DHL showed fast improvement after initiation of pharmaceuticals and lifestyle changes by the 5th visit and continued to improve over time. Their VVAS, which was initially higher than expected for an individual with VM, improved significantly, from 57.7 to 10.4; healthy controls score around 4.6 for reference.(30) Ultimately, management of individuals with VM is improved by an interdisciplinary approach while providing individualized management, including pharmaceutical management, lifestyle modifications,

and a VPT approach selecting interventions to target specific phenotypic presentation. In this case, careful progression was important as they initially presented with an across-the-board phenotype. Awareness and use of diagnostic criteria for VM was an essential first step in this patient's management. Establishing a diagnosis of VM, thorough history, examination, and use of patient-reported and performance outcomes establish a clinical phenotype that informed and guided management. While this case highlights an across-the-board phenotype, which required tailored interventions targeting each of the components of the overall presentation, this approach of phenotyping can be used as a framework of best practice for management of individuals with VM.

Diagnosis and testing: The findings from the VNG were consistent with a left sided UVH which was discordant with the negative clinical head impulse test suggesting a well-compensated UVH with covert

Table 1. Interventions based on across-the-board clinical phenotype

Component	Interventions
Migraine and vestibular migraine education	ICHD and Barany Society Diagnostic Criteria¹³ Common symptoms of migraine/VM Management • Pharmaceutical • Physical Therapy/Vestibular Therapy ◦ Vestibular rehab ◦ Cardiovascular exercise ◦ Resistance exercise • Lifestyle/triggers ◦ Diet ◦ Sleep deprivation or irregularities ◦ Stress ◦ Weather changes ◦ Hormones ◦ Stimulation (visual, auditory, and olfactory)
Autonomic regulation	For management of anxiety, depression, and provoked dizziness symptoms (40) • Paced/diaphragmatic breathing • Grounding • Self-deep pressure • Mindfulness/guide meditation • Progressive relaxation
Balance/sensory re-weighting	Biasing vestibular and de-weighting visual system • Static balance ◦ Firm=>foam ◦ Eyes open (EO) => eyes closed (EC) => EC with head movements => adding cognitive dual tasking • Gait ▪ Walking forwards and backwards => + head turns and nods => walking with quick turns (180 degrees => 360 degrees) => progressed visual complexity of environment (quiet hallway => busy PT gym) => adding cognitive and object manipulation dual tasking

Table 1. Interventions based on across-the-board clinical phenotype (continued)

Component	Interventions
Gaze stabilization and vestibular compensation	Progression • Sitting => standing => walking • Gaze shifting => x1 => x2 • Sinusoidal and impulses • Background simple => complex • Volume (5 cycles => increased repetitions => 1 minute) • Frequency (once => multiple sessions per day) • Goal of 20 minutes consistent with unilateral vestibular hypofunction clinical practice guideline (35)
Habituation	Education on what habituation is, benefits, and how it is performed • 2-3 movements, 2-3x/day • Goal of $\leq 3/10$ increase in symptoms, symptoms return to baseline quickly and symptoms not lasting > 15 minutes after each session(41,42) Self-movement • Functional movements selected by patient ◦ Bed mobility, bending over, turning head, sit-to-stand transitions Visual motion • Task and situation specific based on self-report and VVAS • Progression ◦ Volume (duration x frequency) ◦ Size of screen (phone > tablet > computer screen > TV screen > real life environment)
General movement and wellness	Educated on ACSM exercise guidelines (43) Cardiovascular Exercise (33,35) • Educated on benefits, frequency, volume, and intensity • Walking (patient preferred overground) • Stationary bike • Aquatic swimming/walking • Rower • Elliptical Resistance Exercise (36) • Whole body with focus on function movement patterns

Table 2. Patient-reported and Performance-Based Outcome Scores

Outcome Measure	Visit 1	Visit 5	Visit 9	Interpretation
DHI	60/100 Subscores Physical: 14/28 Emotional: 20/36 Functional: 26/36	26/100 Subscores Physical: 14/28 Emotional: 4/36 Functional: 2/36	20/100 Subscores Physical: 8/28 Emotional: 6/36 Functional: 6/36	Range 0-100 Mild: 0-30% Moderate: 31-60% Severe: 61-100% Higher scores indicate greater functional impairment (27)
VVAS	57.7	39/4	10.4	Range 0-100 Higher scores indicate more severe symptoms (28-30,44)
DVA	4-line difference	3-line difference	2-line difference	Normal $\leq$ 2-line difference (25,26)
FGA	16/30	26/30	30/30	Mean score 28.9/30 for ages 40-49 (45) Scores of $\leq$ 22/30 on the FGA predict falls in older adults (46) MCID of 4 for older adults (47)

Table 2. Patient-reported and Performance-Based Outcome Scores (continued)

Outcome Measure	Visit 1	Visit 5	Visit 9	Interpretation
MCTSIB	100 seconds FOEC: 10 seconds	120 seconds	Normal SOT score on all conditions (Figure 4)	Range (0-120) seconds (sum of FLEO, FLEC, FOEO, and F"OEC) Higher values are better (22)

Abbreviations: DHI, Dizziness Handicap Inventory; DVA, Dynamic Visual Acuity; EO, eyes open; EC, eyes closed; FGA, Functional Gait Analysis; FL, floor; FO, foam; MCID, Minimal Clinically Important Difference; mCTSIB, Modified Clinical Test of Sensory Interaction in Balance; VVAS, Vertigo Visual Analogue Scale

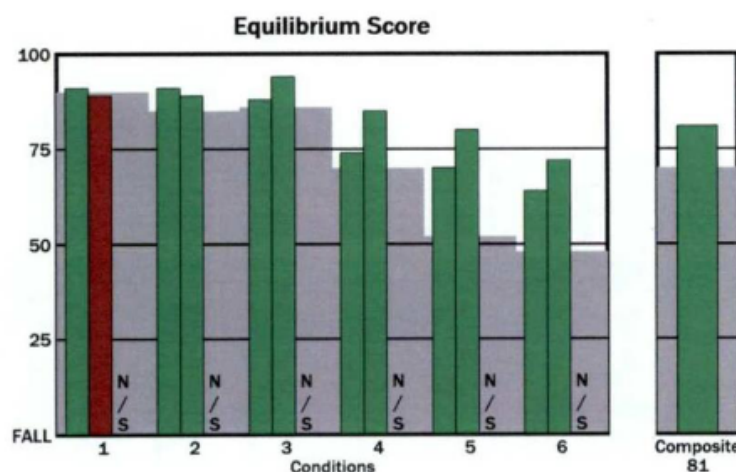


Figure 4. Sensory Organization Test showing normal posturography findings in all conditions suggests intact sensory integration of the visual, somatosensory, and vestibular system by the 9th visit.

saccades, or isolated low frequency UVH. Positive clinical head impulses have been associated with greater than 45% caloric asymmetry,(48) thus this patient presenting with 40% caloric asymmetry may have had false negative clinical head impulses. Although their initial presentation was more consistent with VM than UVH, the sudden onset of VM may have initiated subtle decompensation of a prior left UVH. The timing of their UVH is unclear as they did not report a distinct event of acute vertigo onset with temporal factors consistent with vestibular neuritis.(49) Interestingly, there are a handful of case reports of individuals with abnormal videonystagmography and posturography findings

following Lyme disease infection,(50) a possible a possible explanation for their UVH. Thus, although the left UVH was an unexpected complication to the recovery process the timing and etiology remain a mystery. The presence of a UVH that may have decompensated during VM likely contributed to their degree of gaze instability and disequilibrium and necessitated a higher volume and frequency of head movements during gaze stabilization exercises (34) which were initially poorly tolerated.

Although the positional nystagmus observed in the VNG was inconsistent with BPPV (see Figure 2), there were also incongruent findings across exams. Two-dimensional VNG did not detect the rotational

nystagmus observed in room light at the initial VPT evaluation. Although the nystagmus findings were inconsistent with posterior canal BPPV, the patient was instructed in and performed a home version of canalith repositioning maneuver for the left posterior canal as a habituation exercise.(41,42) Abnormal vestibular function and oculomotor findings have been reported in individuals with VM including 22.9% with abnormal unilateral calorics, 70% with abnormal saccades, 56.7% abnormal smooth pursuits, 26.7% abnormal optokinetics, 33.3% abnormal positional nystagmus (see Figures 1 and 2), and 21.7% spontaneous nystagmus.(5) Oculomotor findings also change over time and phase of migraine.16,51 Thus, it seems reasonable that this patient experienced different positional nystagmus with different directional qualities throughout their course of VPT.

It was not until the overall reduction in headache equivalents (symptom burden), ability to focus on lifestyle modifications, personal trigger identification and management, and slow progression of habituation exercises that they were able to better tolerate gaze stabilization exercises prescribed in a manner consistent with the unilateral vestibular hypofunction (UVH) clinical practice guideline.(34) This need for targeted VM management before being able to optimally address the UVH was not clear until later in the clinical course, highlighting the importance of recognizing the across-the-board VM phenotype. Early collaborative management targeting impairments while respecting the temporal time course of overall symptom burden (headache equivalent and anxiety) may have limited central sensitization and dysfunctional changes in brain networks through exercise driven epigenetic processes. This case highlights successful recovery from a complex vestibular migraine phenotypic presentation with hidden vestibular hypofunction and emphasizes the need for an evolving approach to individualized VPT interventions.

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Persistent Postural Perceptual Dizziness, Emotional Phenotype: A Case Report

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ABSTRACT

Introduction: Persistent Postural Perceptual Dizziness (PPPD) is a chronic functional vestibular disorder, with specific diagnostic criteria. There is emerging evidence on the pathophysiology of PPPD as various maladaptations to the initial triggering event. There have been recent discussions on identifying phenotypes of vestibular conditions to tailor interventions. This case explores a patient with PPPD who demonstrates a dominant emotional phenotype. **History:** A 53 year-old female presented with signs and symptoms consistent with PPPD with a history of anxiety, trauma, and recent emotional stress. **Examination:** Her examination revealed decreased balance confidence, severe perception of dizziness handicap, and severe anxiety. Objective testing indicated static and dynamic balance deficits, impaired gaze stability, and hypersensitivity to eye and head movement. **Intervention & Outcomes:** Interventions were tailored to her emotional phenotype by including comprehensive treatment such as education, grounding, and multidisciplinary care with psychiatry and psychology. Her physical therapy intervention was kept salient and task oriented with appropriate pacing for her tolerance. She demonstrated significant objective improvements and achieved her goals of returning to driving, exercise, and caring for her home and pets. Her subjective outcome measures did not significantly improve, but due to her emotional phenotype, emphasis was placed on function rather than a focus on symptoms. **Conclusion:** In order to achieve effective and efficient care, it is important to consider the specific patient

and their individual factors.

INTRODUCTION

Persistent Postural Perceptual Dizziness (PPPD) was defined as a chronic functional vestibular disorder by the Barany Society in 2017.(1) Diagnostic criteria for this condition include: dizziness, unsteadiness, or non-spinning vertigo that is present on most days for 3 months or more, symptoms are exacerbated by upright posture, active or passive motion, and exposure to complex visual environments. It is precipitated by a condition that causes vertigo, unsteadiness or dizziness.(1) While most people who experience an onset of dizziness will move through a process of adaptation with eventual healing and symptom resolution, there is emerging evidence on the pathophysiology of PPPD that suggests central maladaptation to the initial triggering event. This may include over-weighting of the visual system relative to other sensory cues, development of high threat postural control strategies, heightened body vigilance, and misperceptions of perceived motion. (2)

It is important to note that psychological factors may play a role in the development of PPPD, but as they are not present in all patients with this condition, it is not a psychiatric disorder.(3) There has been recent discussion of identifying phenotypes of vestibular conditions in order to truly tailor interventions to each specific patient.(4) For PPPD, this could include patients being categorized based on symptoms: those primarily provoked by visual stimuli, active motion, or standing/passive motion as well as those patients with a dominant

emotional presentation. (4) These patients may present with high anxiety and/or depression with these factors contributing significantly to their symptoms.

This case explores a patient with PPPD who exhibits a dominant emotional phenotype. We will use this case to describe the importance of tailored intervention based on the unique factors of this individual patient.

HISTORY

The patient is a 53-year-old female who reported an acute onset of room-spinning dizziness three years prior to her initial physical therapy evaluation. She was able to recall the exact date of onset and described dizziness as “hitting her like a freight train”. According to the patient, spinning dizziness lasted for a few months at various intensities. At the time of her PT evaluation, she denied room-spinning dizziness but rather reported a constant feeling of “sloshing around on a boat.” Dizziness was reportedly increased in complex visual environments, such as the patterned carpet in the clinic. Household chores that require repetitive forward bending, such as caring for her cat, were difficult for her, and this caused her to refrain from many daily activities. She felt worse with movement, such as walking, and better when sitting still. The patient stopped driving due to reports of her vision feeling “off” when she turned her head to the right. She self-limited her participation in community outings such as grocery shopping or going to restaurants. She was employed as a caretaker but became limited in her ability to complete her job duties due to dizziness. Previously she enjoyed walking for regular exercise, but she stopped due to noticing staggering and stumbling with walking. The patient had been under significant emotional distress recently due to her mother passing away about one week prior to initiating physical therapy. Notably, the patient reported a significant prior history of emotional trauma.

The patient reported a history of atypical sleep patterns, cervical spine and temporomandibular

dysfunction, and stress related headaches. She began taking Clonazepam 0.5 mg nightly one month prior to the physical therapy evaluation, which reportedly helped her normalize her sleep pattern. She was involved in a motor vehicle accident two months prior to her dizziness event, with reported whiplash, in which she received PT for cervical and temporomandibular dysfunction. Since the incident, she has experienced headaches once a week that were triggered by stress. She had one recent fall, but did not require medical intervention. Prior to her current evaluation, she had participated in physical therapy at another clinic multiple times per week for approximately a year to address dizziness without relief. Her physical therapy goals were to return to driving, care for her cat, and walk independently for exercise.

EXAMINATION

The patient completed three subjective questionnaires: Dizziness Handicap Inventory (DHI), Activities Specific Balance Confidence Scale (ABC), and Generalized Anxiety Disorder 7-item Scale (GAD-7). The DHI is a 25-item self-assessment used to evaluate self-perceived emotional, physical, and functional effects caused by dizziness. The questionnaire is scored from 0 (no perceived handicap) to 100 (severe perceived handicap), with categories of mild, moderate, and severe perception of handicap.(5) The patient's score on the DHI was 80/100, consistent with a severe perception of handicap.(6) The ABC is a 16-item self-report measure used to rate balance confidence in various activities. The questionnaire is scored from 0 (no confidence) to 100 (complete confidence). (7) The patient's score on the ABC was 21.9% which is notably below the documented mean score of 63/100 in individuals with vestibular conditions.(8) The GAD-7 is a self-administered tool used to screen for and assess severity of generalized anxiety disorder. The GAD-7 score ranges from 0 to 21 with 5, 10, and 15 serving as respective cut offs for mild, moderate, and severe anxiety. The patient's score on

the GAD-7 was 19/21, suggesting severe anxiety.(9)

An oculomotor examination was completed within the patient's tolerance. Smooth pursuit, as well as spontaneous and gaze-evoked nystagmus, were normal in room light. Vestibulo-ocular reflex (VOR) cancellation and cross-cover tests were within normal limits. The head thrust test indicated an abnormal finding on the right with a notable catch-up saccade, and a normal finding on the left. Dynamic visual acuity (DVA) testing was abnormal with a loss of 6 lines, and the patient reported 7/10 dizziness following the test.

(10) Vestibular/Ocular-Motor Screening (VOMS) showed significant sensitivity to smooth pursuits and saccades, with increased headache, dizziness, nausea, and foggiess reported.(11) She was unable to complete the VOMS due to symptoms remaining severely elevated following smooth pursuit and saccades. Although designed as a screening tool for concussion, the VOMS was used with this patient as a means of quantifying the patient's symptoms with standardized oculomotor tasks. The modified Motion Sensitivity Test (mMST) was not completed due to symptom elevation.(12)

The patient had no functional strength deficits and bilateral lower extremity sensation was normal. Static balance was tested using the Modified Clinical Test of Sensory Interaction on Balance (mCTSIB), single-leg stance, and tandem stance. The patient was able to maintain balance for 30 seconds on conditions 1-4 of the mCTSIB. However, deficits were noted in static balance with single-leg stance for 7.1 seconds (normative data for patient age range = 36 + 12.8 seconds) and tandem stance for 7.6 seconds (normative data for patient age range is 30 seconds).(13,14)

Dynamic walking balance was tested using the Functional Gait Assessment (FGA). The patient scored a 13/30 on the FGA, indicating a dynamic balance deficit lower than fall risk cut-offs identified in older adults and scoring below her age norm.(15, 16)

The patient exhibited gait deficits including significant right lateral lean, intermittent hand-held

assistance from family, and decreased gait speed of 0.7 meters/second (abnormal for age and gender). (17)

Infrared video goggles were used to assess for spontaneous and gaze-evoked nystagmus and during positional testing. Spontaneous and gaze-evoked nystagmus were within normal limits with fixation removed, and positional testing was normal. The patient experienced non-spinning dizziness upon returning to sit after the Dix-Hallpike test.

DIAGNOSIS

The patient completed a full battery of vestibular function testing with audiology in addition to the physical therapy evaluation. Results demonstrated low gain with overt and covert saccades on right anterior and lateral canals on the video head impulse test (vHIT), low gain on right rotational chair at frequencies .01-.64 Hz, 100% reduced vestibular response on the right (with no response to ice water) on caloric testing during videonystagmography with 40% leftward directional preponderance. Her cervical vestibular evoked myogenic potential was absent on the right, and her ocular vestibular evoked myogenic potential showed a 45% right weakness in amplitude. Her electrocochleography and auditory brainstem response (ABR) were normal.

These findings confirmed a right vestibular hypofunction, likely associated with the sudden onset of spinning dizziness three years prior. However, her current dizziness was described as a constant, non-vertiginous dizziness that was triggered by busy visual environments, upright position, and active or passive motion. Due to these factors, it was determined that the primary diagnosis was PPPD.(1) There was strong suspicion of additional psychosocial factors due to the recent passing of her mother, significant anxiety, and past trauma. Therefore, her presentation was consistent with PPPD with an emotional dominant phenotype.

INTERVENTIONS

Intervention took a multifaceted approach tailored

to the patient's symptoms. Before initiating vestibular or balance exercises, treatment focused on education regarding her diagnoses of unilateral vestibular hypofunction and PPPD. Due to the emotional phenotype, emphasis was placed on the importance of multidisciplinary care and mental health support. She was referred to psychiatry and psychology for additional intervention. At her initial evaluation, she was introduced to a seated grounding technique designed to enhance proprioceptive and tactile sensory input, to help reduce her baseline symptoms.

The patient participated in eight vestibular physical therapy sessions over 10.5 weeks. To reduce the overall burden of therapy and accommodate the patient's limitations, she participated in a hybrid model with an in-person initial evaluation followed by telehealth visits from home. Due to symptom provocation with motion, she was unable to drive and lived a considerable distance from the clinic. Telehealth enabled her to participate in care without the added stress of travel or the risk of symptom exacerbation from community outings. This format also allowed for observation of functional activities in her home environment, facilitating tailored strategies for daily mobility and symptom management. Additionally, the convenience of telehealth promoted consistent participation, which was critical given her complex presentation and comorbid emotional stressors.

As described in the examination, the patient was quite symptomatic with head motion (DVA, FGA) and oculomotor tasks (VOMS). The concept of habituation was introduced during this session. The patient received a written explanation of the habituation process, along with instructions on symptom monitoring, including a 0–10 visual symptom rating scale to emphasize the importance of an appropriate level of symptom provocation. Initial habituation exercises included smooth pursuit and saccades, each performed for 15 seconds per set, two sets, three times daily. Parameters were based on the patient's symptom response during the

supervised session. She was also reintroduced to grounding techniques to aid symptom regulation throughout the day.

At the subsequent telehealth visit the patient reported reduced dizziness during saccades and smooth pursuit and increased tolerance for leaving the house. Her exercises were progressed to include ocular movements performed against busy visual backgrounds (e.g., looking at the staircase or at windows within the home). Head movement habituation was initiated, starting in a seated position and advancing to standing and walking with various visual surrounds. Symptom increases remained within a mild to moderate range and consistently returned to baseline within minutes of rest. The patient was encouraged to look at meaningful items around the house when performing head movements, such as her cat or pictures of family, to improve saliency. She was encouraged to incorporate grounding techniques during rest periods, and continued education was provided regarding the importance of nervous system regulation and symptom control.

Around this time, the patient became established with both psychiatry and psychology. Pharmacologic management was initiated to address anxiety and trauma-related symptoms, and she began attending weekly cognitive behavioral therapy sessions with a licensed clinical social worker who has experience with the diagnosis of PPPD. The importance of psychological care and pharmaceutical intervention in combination with vestibular physical therapy for PPPD is well-established in the literature.^(1,3)

At the following visit, the VOR x1 exercise was introduced. Given her ongoing sensitivity to motion, exercises were limited to 15-second intervals initially to prevent excessive provocation. The patient received detailed education about the role of the vestibular system in gaze stability and its functional significance. She was educated on the clinical practice guideline targets for VOR exercise duration of 20 minutes per day in cases of chronic unilateral vestibular hypofunction. However, her

dprogram was intentionally modified to reflect an individualized, tolerance-based progression.

The patient reported a high level of adherence throughout her sessions and continued to show motivation to achieve goals of therapy, including return to driving, exercise, household tasks, and vacation with family. She reported the benefits of counseling, including techniques for nervous system regulation and implementation of positive reinforcement and positive associations related to her symptoms.

Symptoms with smooth pursuit and saccades improved within the first few weeks of therapy. At this time, formal participation in saccades and smooth pursuit exercises was eliminated in order to focus on VOR x 1 and movement habituation. VOR x 1 was gradually advanced in time, speed, and situation (i.e., various balance positions, marching in place, various visual backgrounds). Multi-sensory balance training was initiated, including various tasks with eyes closed, such as head turns or forward bends. An important goal for the patient was to be able to bend down to pet or feed her cat without dizziness. Therefore, habituation exercises were advanced to include forward bending and head turns and were centered around this activity. Graded exposure to forward bending was performed by reaching down to pet her cat on an elevated surface, such as a chair or a couch. When this improved, she was able to reach down to pet her cat at ground level.

Once she was able to tolerate VORx1 for two minutes in each direction and she reported improvement in target stability, she was educated on VORx2 training. This advancement allowed for increased functional use of gaze stability during daily activity. Balance and habituation exercises continued to be advanced to incorporate elements of dual task effort and visually complex environments. At this time, the patient was becoming more active, with a return to walking and water aerobics for exercise, driving short distances, and vacationing with family at the beach. Intervention was gradually shifted away from formal

oculomotor, habituation, or balance exercise and toward functional participation in activities such as driving, going to restaurants, and participating in workout classes.

OUTCOME

Over a two-month period of vestibular physical therapy, the patient was able to significantly increase her activity and participation levels. Despite overall functional improvement, her subjective questionnaires showed only slight, but not significant, improvement in the DHI (78/100) and ABC (28%). DVA testing improved to a 3-line loss, with reduced dizziness symptoms. She was able to complete VOMS with no increase in symptoms of headache, nausea, or dizziness with smooth pursuit and saccades and minimal increase in symptoms of foggy (2/10) with all test conditions. She was also able to complete the mMST and demonstrated a moderate level of motion sensitivity. Despite not making large gains related to symptoms, she was able to tolerate provocative testing that she could not tolerate at the initial evaluation, indicating improved symptom tolerance. Her static balance improved in single-leg stance to 16 seconds and tandem stance to 30 seconds. Dynamic balance improved to 28/30 on the FGA. Her gait speed increased to 0.94 meters/second, and her gait pattern normalized. See Table.

She reported improvement in overall symptoms with reduced feeling of “sloshing on a boat.” Some dizziness with overstimulation was still present, but the patient felt confident in managing her symptoms with grounding, breathwork, and techniques learned in counseling. She was attending water aerobics and walking for exercise. She was able to return to riding in a car, driving, and caring for her cat with minimal symptoms.

DISCUSSION

The importance of targeted intervention is highlighted in this case through use of multi-disciplinary intervention, individualized exercise to

Table. Outcome measure scores from initial evaluation to discharge visit.

Outcome Measure	Initial Evaluation	Visit 4	Discharge (Visit 8)
Dizziness Handicap Inventory	8//100	Not tested	78/100
Activities-specific Balance Confidence Scale	21.9%	Not tested	28.1%
Generalized Anxiety Disorder-7	19/21	Not tested	Not tested
Baseline dizziness	3/10	0/10	0/10
Dynamic visual acuity	6 line loss	Not tested	3 line loss
Single-leg stance Eyes open Eyes closed	7.6 seconds Not tested	Not tested	16 seconds 1 second
Tandem stance Eyes open Eyes closed	7.6 seconds Not tested	Not tested	30 seconds 16 seconds
Functional Gait Assessment	13/30	25/30	28/30
Gait speed	2.3 feet/second	Not tested	3.1 feet/second
Vestibulo-Oculomotor Screen	Unable to complete	Not tested	Tota: 20 Highest individual score: 2
Modified Motion Sensitivity Test	Unable to complete	Not tested	15.4

through use of multi-disciplinary intervention, individualized exercise to emphasize function and salience, and patient-specific education. through use of multi-disciplinary intervention, individualized exercise to emphasize function and salience, and patient-specific education. This is especially

important in patients experiencing chronic symptoms that are confounded by psychosocial and emotional factors. This patient was responsive to the extensive education regarding her diagnosis, original precipitating condition, and psychosocial factors. Education was exceedingly important in this

case, as she felt that she was not being heard or understood by other medical providers. The emphasis placed on function, rather than symptoms, was essential in symptom reduction and improving quality of life. As described in the OPTIMAL theory for motor learning, it is important to emphasize the task/goal and de-emphasize self-focus. (18) Motor learning is also improved by autonomy and salience of the task. In this case, the patient was motivated to perform forward bending to be able to care for her cat and to perform gaze stability and head movement activities to return to driving. Her care was centered around the meaningful achievements she was making towards her specific goals. Her subjective outcome measures did not improve significantly, but this has been shown in other patients with comorbid anxiety and depression. (19) She also continued to demonstrate a moderate level of motion sensitivity per mMST, which was an improvement from the initial evaluation when she could not tolerate the testing. Even though she still had moderate symptoms, she had mostly achieved her functional goals. Rather than focus on these symptom reports, her progress was based on the very salient and meaningful functional improvements she demonstrated. Overall, this case demonstrates a disconnect between functional ability and symptom reports for some individuals with PPPD.

Her case shows the value of multi-disciplinary and individualized intervention. The additional support of psychology and psychiatry facilitated her ultimate progress by providing diagnosis-specific medical intervention, emotional support, and coping strategies. As she made limited progress with previous vestibular rehabilitation, initiating a referral for psychology and psychiatry while focusing more on function may have been the combination that allowed her to improve. While PPPD is a distinct diagnosis, it is clear that not all patients with PPPD present with the same factors such as life experiences, background, genetics, and stressors. Although this patient clearly presented with an emotional phenotype, not all patients with PPPD will

need a referral to psychology and psychiatry. This case highlights the importance for vestibular therapists to identify these distinguishing features and phenotypes to best address patient needs and tailor interventions for optimal outcomes.

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5th Annual ANPT Annual Conference 2025 Preview

The 5th Annual ANPT Conference will take place at the Hilton Omaha Downtown in Omaha, Nebraska, from October 16-18. The event includes education sessions, poster presentations, networking sessions, and time to interact with vendors. For registration, updates, and more event details, visit the ANPT website (www.anpt.org). Below is the current list of confirmed presentations pertaining to vestibular physical therapy.

Friday 3:45 PM-5:45PM

From Observation to Action: Using the Universal Framework for Movement Analysis & Balance Diagnoses
Presented By: Ana M. Sanchez Junkin, PT, Christine Tyrell, PT, DPT, PhD*, and Christine Zimmerman, PT, DPT*, CBIS

Saturday 7:00AM-8:00AM

Hot Topic: Atypical Vestibular Physical Therapy Settings
Hosted by: Rachel Wellons, PT, DPT*

Saturday 2:00 PM-4:00 PM

Challenges in Benign Paroxysmal Positional Vertigo: A Video Case Approach to Atypical Presentations
Presented By: Mary Crumley, PT, DPT* and Beth Hughes, PT, DPT*

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Psychologically-Informed Vestibular Physiotherapy Using the INVEST Approach for Persistent Postural Perceptual Dizziness

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ABSTRACT

Introduction: Management of persistent postural perceptual dizziness (PPPD) has been challenging historically, with limited high-quality studies that can identify best practice. More recent research has shown positive outcomes with a multi-disciplinary approach to treatment combining cognitive behavioural therapy, vestibular rehabilitation, and pharmacological interventions. However, access to this type of approach is difficult. This article details the clinical journey of a patient treated with a psychologically informed approach by a physiotherapist, known as integrated vestibular therapy (INVEST). **Case description:** 47-year-old female with a diagnosis of PPPD, vestibular migraine and panic disorder. **Intervention:** A physiotherapist delivered the INVEST protocol over six sessions. The protocol includes education, formulation to make sense of their dizziness from a psychophysiological perspective, and balance therapy, focusing on reducing hypervigilance, goal setting, in-vivo exposure with response prevention, and relapse management. **Outcomes:** The patient demonstrated improvements in dizziness handicap, dynamic gait, PPPD symptoms, and anxiety-depression scores. Functional goals were also achieved, including the patient returning to work. **Conclusion:** The INVEST method as a treatment approach for PPPD indicates positive outcomes as a physiotherapy-led combined cognitive-behavioural approach to vestibular rehabilitation. Further multicentred randomised controlled trials investigating combined approaches are in preparation.

INTRODUCTION

Persistent Postural Perceptual Dizziness (PPPD) is a

common functional vestibular disorder seen in many specialised outpatient clinics.(1,2) The disorder is characterised by a persistent sensation of dizziness, unsteadiness or non-spinning vertigo that is exacerbated by upright posture, active or passive movement, and exposure to complex visual environments.(1)

Management of PPPD has historically been challenging, and different branches of management have been suggested, including pharmacotherapy, psychological therapies, and vestibular physiotherapy.(3) However, the most recent Cochrane review of treatment approaches for PPPD did not identify any high-quality studies that could support recommendations for best practice.(4) There are promising outcomes shown for multidisciplinary inpatient programmes that combine these approaches (5), although this is not available to most. Despite evidence supporting the link between psychological factors and PPPD, access to high-quality cognitive behavioural therapy (CBT) remains limited (6), and many individuals are reluctant to engage with psychological therapy.

In response to those challenges, Herdman, et al (8) developed a 'cognitive physiotherapy' (a.k.a., psychologically informed) approach, enabling physiotherapists to integrate cognitive-behavioural strategies into vestibular rehabilitation (CBT-VR). The intervention was based on existing research data and theoretical modelling of the psychological factors that contribute to dizziness handicap.(9,10) The integrated vestibular therapy (INVEST) protocol is delivered with a patient manual and physiotherapist support over 6 sessions.(8) The protocol includes education, a joint formulation to make sense of their dizziness from a

psychophysiological perspective, balance therapy focusing on reducing hypervigilance, goal setting, in vivo response prevention, exposure and relapse management. It was initially evaluated in a feasibility study (11) and it has since been implemented in our department as part of routine care for patients with PPPD.

This case study will present the clinical journey of a recent patient diagnosed with PPPD, in addition to vestibular migraine and panic disorder. The patient was treated using the INVEST method, and the study will highlight how this approach was applied in practice.

CASE STUDY

History

A 47-year-old female was referred for vestibular rehabilitation by a specialist balance clinic following a diagnosis of PPPD. She had a history of episodic vertigo, nausea, and headaches since February 2022. She had a past medical history of migraine, presenting since her mid-20s. The initial episodes lasted 24-48 hours, but over time, she started to develop more persistent inter-ictal non-spinning dizziness and disequilibrium. She also experienced recurrent panic attacks and worsening anxiety alongside the ongoing episodes of headache and vertigo.

She was subsequently referred to a specialist neuro-otology balance clinic and diagnosed with vestibular migraine and PPPD. Brain imaging was normal, and she had normal vestibular function testing (video-nystagmography, vibration-induced nystagmus, head-shaking nystagmus, rotational chair tests, video head impulse test and cervical-vestibular evoked myogenic potentials). She made lifestyle modifications to manage her migraines and started taking riboflavin and magnesium. Before starting physical therapy, her migraine frequency had reduced to 3 headaches a month. She was taking sertraline for her anxiety, and she completed a course of CBT, which helped her anxiety symptoms, but did not change her symptoms of dizziness.

When she attended physiotherapy, she described a constant sensation of feeling like she was drunk, with associated nausea and imbalance. These symptoms were aggravated by standing, walking, being in supermarkets, and moving her head. Her symptoms were causing her significant distress and having a considerable impact on her quality of life. She had been working as a psychologist full-time, but having tried to return to work several times, she had been unable to manage. In summary, her symptoms were in keeping with a diagnosis of PPPD (1) with an initial trigger of vestibular migraine (12) and associated panic disorder.

Examination

- Oculomotor: normal
- Horizontal canal head impulse testing: normal.
- Dix-Hallpike and supine roll tests: normal
- Romberg: normal balance. However, she reported feeling unstable, with an increased perception of sway.
- Gait: cautious, with an increased threat response, narrowed gaze, shortened stride length, unwillingness to move her head, and there was clear conscious processing of her gait.
- Dynamic Gait Index – modified 4-item version (13): 8/12 (gait = 2, change in gait speed = 2, horizontal head turns = 2, vertical head turns = 2).

Self-Report Measures:

The Dizziness Handicap Inventory (DHI) is a 25-item self-reported outcome measure designed to measure the degree of perceived disability caused by dizziness or unsteadiness.(14) The patient scored 70/100, which is consistent with Graham, et al (15), who showed that a score greater than 60 is highly specific for the presence of functional or psychiatric disorder.

The Patient Health Questionnaire – Anxiety and Depression Scale (PHQ-ADS) (16) combines the Patient Health Questionnaire – 9-item scale of depression (17) and the Generalised Anxiety

Disorder – 7-item scale of anxiety (18) resulting in an overall level of distress. The benefit of the combined scale is that anxiety and depression often occur simultaneously, so it can detect ‘subclinical’ levels of distress, and this has been validated in patients with vertigo and dizziness.(16) Her initial score suggested a moderate level of distress.

The Niigata PPPD Questionnaire (NPQ) is a 12-item questionnaire that has been shown to have high reliability and validity in measuring severity of PPPD. (19) The patient initially scored 35/72 on the overall score and 14 on the visual stimulation sub-score. An overall score of over 27 and a visual stimulation score of greater than 9/24 is sensitive and specific in distinguishing between individuals with and without PPPD.(19)

Intervention

The physiotherapist was trained to deliver the INVEST protocol, which was delivered over six sessions of physiotherapy. The first session was one hour long and follow up sessions were 30 minutes each.

Session 1: Assessment and Formulation

At the end of the initial assessment, a formulation was developed with the patient, integrating their own words to demonstrate a cohesive story that explained how her symptoms had developed over time (Figure). Importantly, this highlighted the link between her symptoms, thoughts, feelings, and behaviours, and that what may have at one time been an adaptive strategy (e.g., not moving) was no longer helping, opening up the possibility for behavioural change. She was provided with a workbook and encouraged to read the educational worksheets about persistent dizziness before the next session. She experienced fewer symptoms during Romberg with cognitive distraction, highlighting the maladaptive influence of symptom focusing and attention on balance.

Home Tasks: Reading the manual, performing balance tasks with cognitive distraction, develop a

consistent daily routine, stabilising activity/rest.

Session 2: Goal Setting

The focus of goal setting was to move away from symptom-focusing and towards specific, measurable, functional tasks. The patient identified long-term goals for walking, shopping, and socialising, which were then broken down into manageable steps.

Her gait was reviewed, and exercises were provided to normalise her walking, tapping into more automatic movement, without engaging in maladaptive vigilance or tension. She was advised to put this into practice during a daily walking programme.

Home Tasks: Habituation exercises based on provocative activities — vertical and horizontal head turns in standing, walking while throwing and catching a ball, a daily independent outdoor walking program for 10 minutes.

Session 3 and 4: In-vivo exposure

In-vivo behavioural experiments were conducted to challenge fear avoidance and catastrophic thoughts about her symptoms. Experiments are set up to test an ‘If-Then’ prediction. For example, she rated that walking quickly downstairs without a banister would result in her falling over. An experiment was set up to test this expectation, working up to running up and down the stairs. By challenging these thoughts through experiments, we were able to reduce her expectation that these would be the intended outcomes.

Home tasks: Progressed habituation exercises to vertical and horizontal head turns while walking, bending down from sitting, daily independent walking program increased to 20 minutes.

Session 4 and 5: Cognition

In addition to the in-person intervention delivered she was encouraged to complete worksheets at home on cognitive training, which challenged her dizziness-related beliefs. This was designed to be

self-managed, with the therapist able to review homework tasks and answer questions. In session 5, any ongoing blocks to recovery were reviewed.

Home tasks: Daily independent walking program increased to 30-45 minutes daily.

Session 6: Transition to self-management

During the final session, we reflected on what she had learned about her condition, what strategies were helpful, and set goals for the next 3 months. We also discussed relapse management, how she could identify warning signs that her dizziness would worsen, and the steps that she could undertake if she was struggling. She was discharged but could contact the department to book further follow-up if required within a 3-month period, which was not required.

Outcomes

The patient engaged well throughout the intervention, achieving her goals and identifying future goals to return to work and to start running. By the end of the intervention, she had started a graded return to work. All her outcome measures improved (Table), the DHI improved by 36 points, which is more than the 18 points that indicate reliable improvement (14), and her symptoms had reduced from severe to moderate dizziness.(20) Her PHQ-ADS scores reduced from moderate to no levels of distress.(16) The 4-item DGI also returned to within normal range.(13) The NPQ score reduced from 35 to 9, indicating her symptoms had reduced below the threshold for meeting a diagnosis for PPPD.(19) Most importantly, the patient herself could see the improvement, feeling like she had regained control of

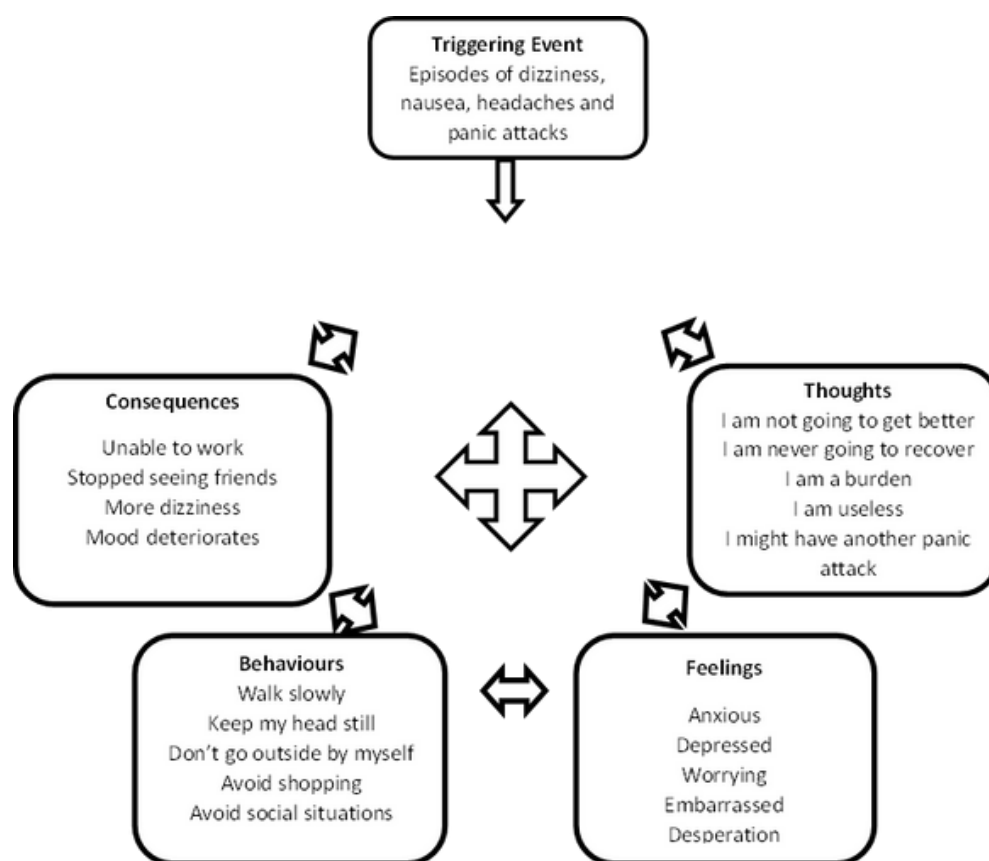


Figure. Flow diagram of the formulation developed during the initial assessment to improve patient understanding of the problem. The bidirectional arrow not only show directionality to the relationships within the formulation, but also offer opportunities to modify those relationships.

her life and could look forward to the future:
“I didn’t have any hopes or expectations of how this treatment might help me, I thought physiotherapy might help a little bit, but I thought this was something I was going to have to live with but this treatment has helped me to get my life back and I can now look forward to things again.”
(Patient feedback)

DISCUSSION
The case study highlights the exciting potential for the use of the INVEST approach, which is a combined CBT-VR approach for improving outcomes for those with PPPD.¹¹ Improvements in this case were demonstrated in dizziness, anxiety, depression, distress and gait outcome measures. This evidence further adds to a recent feasibility study, which

Table. Outcome measure scores at initial assessment and discharge.

Outcome Measure	Initial Assessment	Discharge Visit
Dizziness Handicap Inventory	70	34
Patient Health Questionnaire - Anxiety and Depression	28	9
Nigata PPPD Questionnaire	35	9
Upright posture	10	2
Movement	11	2
Visual stimulation	14	5
4-Item Dynamic Gait Index	8	12

her life and could look forward to the future:
“I didn’t have any hopes or expectations of how this treatment might help me, I thought physiotherapy might help a little bit, but I thought this was something I was going to have to live with but this treatment has helped me to get my life back and I can now look forward to things again.”
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DISCUSSION
The case study highlights the potential for the use of the INVEST approach, which is a combined CBT-VR approach for improving outcomes for those with PPPD.⁽¹¹⁾ Improvements in this case were demonstrated in dizziness, anxiety, depression, distress, and gait outcome measures. This evidence further adds to a recent feasibility study, which

showed promising results.⁽¹¹⁾ However, as this is based on a single case study, further high-quality multicentred randomised controlled trials are planned.
Anxiety has been shown to be a predictor for patients developing PPPD following a vestibular insult.^(21,22) High rates of anxiety and panic attacks have also been shown to be present in patients with vestibular migraine ⁽²³⁾, and can increase the risk of developing chronic symptoms.⁽²⁴⁾ In this case study, the triggering event was the development of vestibular migraine, while subsequent anxiety and panic attacks were likely an important contributing factor towards the development of PPPD. This could be categorised as a mixed neurologic etiology, whereby vestibular migraine triggered psychopathology and subsequent development of

PPPD.(25)

Selective serotonin reuptake inhibitors (SSRIs) and serotonin-norepinephrine reuptake inhibitors (SNRIs) have both been suggested as treatments for PPPD. However, there is currently limited high-quality evidence to support their efficacy.(26) Proposed mechanisms for pharmacological efficacy may include altering hyperexcitability, reducing anxiety, or direct effects on the balance system.(26) In this case, the patient started taking sertraline before commencing treatment. While she did report improvements from an anxiety perspective, there was no reported reduction in her dizziness symptoms with medication alone. This highlights the need for further trials to determine the efficacy of pharmacological interventions in treating PPPD, to explore the role of medications in combination with other treatments, and to identify if certain subgroups of patients with PPPD would benefit from pharmacological intervention.

CBT has also been suggested as a treatment approach for patients with PPPD. A recent retrospective study (27) looked at specialised CBT as a single intervention for PPPD and found a significant reduction in DHI scores; however, getting access to specialised CBT is challenging.(26) It has been suggested that a combined approach may be more beneficial for patients.(23) In this case, the patient had received prior treatment for CBT. However, it was not clear whether this therapy was specialised and directed towards addressing the dizziness. CBT includes a range of different techniques, some of which may have greater effects on dizziness outcomes than others, so it is important that interventions are empirically derived and precisely targeted.

Vestibular migraine is one of the most common triggers for PPPD but can also typically co-exist alongside PPPD.(28) Fifty-three percent of patients with PPPD meet the diagnostic criteria for headache, while 17% meet the diagnostic criteria for definite vestibular migraine.(28) There is considerable overlap between vestibular migraines and PPPD that

can make diagnosis challenging. Due to the lack of biomarkers, the diagnosis for both conditions relies on diagnostic criteria based on subjective information, further adding to the challenge. In this case, the patient's vertigo and headache attacks clearly met the diagnostic criteria for vestibular migraine (12) with a subsequent improvement with lifestyle changes and supplements, which are both commonly used to treat vestibular migraine.(29) However, despite the improvements in her headaches and vertigo attacks, the patient's persistent symptoms of dizziness and unsteadiness remained, thus highlighting the importance of the additional diagnosis of PPPD. It was these more persistent symptoms that responded to the biopsychosocial approach offered by the INVEST approach. This highlights the importance of adequately identifying all possible vestibular diagnoses and treating these appropriately in line with evidence-based recommendations to achieve overall optimal improvement for the patient.

The diagnostic criteria for PPPD illustrate the potential for heterogeneity within functional disorders and PPPD.(1) In the future, the ability to identify dominant behavioural and sensory phenotypes may help clinicians predict and target interventions in a more specific way.(30) Visual intolerance, active motion, passive motion, and standing intolerance have been identified as potential phenotypes for PPPD.(30) However, despite some of these factors being present in this case, they were not particularly significant. Instead, fear avoidance behaviours in the context of fear and embarrassment were a significant driving factor towards the development of PPPD. This case raises the potential for a separate fear avoidant phenotype, however further studies would need to explore this within a larger data set.

CONCLUSION

This case study demonstrates the potential for the INVEST method as a treatment approach for PPPD. Due to the paucity of evidence-based approaches to

treat PPPD, further multicentred randomised controlled trials investigating combined approaches should be considered.

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ARE YOU INTERESTED IN WRITING A LITERATURE REVIEW? DO YOU HAVE AN INTERESTING CLINICAL CASE?

The TVPT Editors are interested in sharing your clinical experience and can provide mentorship to help you contribute.

Contact: Holly Roberts at hjroberts@pugetsound.edu

Going to CSM 2026? Be Sure To Stop By the ANPT Myelin Melter and Business Meeting!

VRSIG representatives will have a table there to discuss SIG engagements and pick up a badge buddy!

When: Friday, February 13th at 5:30 pm
Check CSM website for location

Vestibular SIG Service Award

Maureen Clancy, PT, DPT, Cert. MDT

- Vestibular SIG Social Podcast Coordinator for the past 8 years
- Responsible for the podcast committee members
- Produced 38 podcasts
- Consistently high download numbers and listeners from across the globe!!



Vestibular SIG Research Award

Eric Anson, PhD

- Leader of the Vestibular Research Lab at the University of Rochester
- 31 (and counting) publications in peer reviewed journals such as Journal of Vestibular Research, Archives of Physical Medicine and Rehabilitation
- Over \$800,000 in external grant funding for Vestibular Research
- Active mentor of junior researchers, predoctoral, and postdoctoral researchers
- Research into eye movements and vestibular perceptual mechanisms have significantly contributed to the field



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Thank you to the Vestibular Rehabilitation SIG Outgoing Officers



Nominating Committee

Dawn Fitzgerald, PT, DPT

Board-certified specialist in neurologic physical therapy

Vice Chair

Lisa Heusel-Gillig, PT, DPT

Board-certified specialist in neurologic physical therapy



JOIN US FOR **VERTIGO-GO**



**We would love for you to join us at
the ANPT Conference for a social event to
mingle with other vestibular therapists!**



Friday, October 17
7:30 PM
Blatt Beer and Table
610 N. 12th St., Omaha, NE 68102

*Individuals are responsible for their own food/drink bill.

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Topics in Vestibular Physical Therapy (TVPT) is the official publication/newsletter of the Vestibular Rehabilitation Special Interest Group (VRSIG) of the Academy of Neurologic Physical Therapy (ANPT). The purpose of the publication is to disseminate clinically relevant information to our members who treat individuals who have vestibular related symptoms.

The editors of the TVPT will accept literature reviews, brief research reports, clinical perspectives, conference presentation summaries, and clinical case studies. Editors will support and mentor clinicians who wish to contribute clinical experience and knowledge in this forum. The editors invite members to suggest topics and guest editors with expertise in a targeted topic.

TVPT is published biannually and is available online through the ANPT/VRSIG web page.

[https://www.neuropt.org/special-interest-groups/vestibular-rehabilitation/topics-in-vestibular-physical-therapy-\(newsletter\)](https://www.neuropt.org/special-interest-groups/vestibular-rehabilitation/topics-in-vestibular-physical-therapy-(newsletter))

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