Use of Vestibular Rehabilitation as a component of the treatment of Persistent postural-perceptual dizziness or PPPD – a review

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**ABSTRACT:**

The purpose of this article is to review the advancing understanding, differential diagnosis, and the integral role of Vestibular Rehabilitation (VR) in the relatively new, but very common diagnosis, Persistent postural-perceptual dizziness (PPPD). PPPD is a functional vestibular disorder characterized by chronic and persistent non-spinning dizziness, visual/motion hypersensitivities and perceived unsteadiness that is often triggered from a definable peripheral vestibular disorders (PVD). PPPD is being found to be more common than many well-known PVD such as Meniere’s or bilateral vestibular hypofunction. Early and correct identification of PPPD, including appropriately prescribed VR, is being found essential to not only promote optimal recovery but also to prevent more refractory engrained PPPD. PPPD management is steadily advancing and treating clinicians are now able to access internationally-sanctioned diagnostic criteria, WHO ICD-11 classification, and advancing treatment protocols which integrate not only medications and cognitive behavioral therapy but define the crucial role of VR, including education, visual/optokinetic motion desensitization, graded habitation, balance retraining and integrated relaxation/mindfulness.

**Key words:** balance, chronic subjective dizziness, dizziness, exercise, functional dizziness, persistent postural-perceptual dizziness, phobic postural vertigo, rehabilitation, space and motion discomfort, vestibular rehabilitation, visual vertigo

**INTRODUCTION:**

PPPD is one of the most steadily advancing chronic vestibular syndromes in VR with advancing differential diagnosis criteria, and increasing efficacy of treatment techniques.(Dieterich & Staab, 2017; Dieterich, Staab, & Brandt, 2016; Popkirov,
Staab, & Stone, 2018; Seemungal & Passamonti, 2018) PPPD (pronounced "triple P or 3PD"), when properly recognized, is estimated in tertiary practice to be one of the more common causes of chronic or persistent dizziness at incidence of 15-20%. (Popkirov et al., 2018) Research is particularly being facilitated by the recent publication of the Barany Society's International Classification of Vestibular Disorders (ICVD) diagnostic criteria for PPPD (J. P. Staab, Eckhartdt-Henn A., Horii A., Jacob R. Strupp M., Brandt T., Bronstein A., 2017), and the integration of the diagnosis into the WHO, ICD-11 revision. (WHO, 2018) Pilot studies have demonstrated 78% sustained (>1 year) reduction in dizziness utilizing combined antidepressant medications, cognitive behavioral therapy and vestibular rehabilitation. (Best et al., 2015; Schaaf & Hesse, 2015)

**HISTORICAL:**

The historical development of PPPD has been described in detail with particular advancements in the last 40 years. (J. P. Staab, 2012; J. P. Staab et al., 2017). PPPD had its earliest roots with Karl Westphal's agoraphobia in the 1870’s which then really wasn’t further revisited until early 1980’s with Thomas Brandt and Marianne Dieterich’s pioneering work which defined the clinical syndrome, phobic postural vertigo (PPV). This was followed, in the mid-1980’s, with Jacob and colleague’s investigations of the interactive role of anxiety, dizziness and vestibular dysfunction, who coined and define one of the cardinal symptom terms, space and motion discomfort (SMD). Next to contribute was Bronstein in 1995, who’s work defined the next pathognomonic symptom term, visual vertigo (VV) with the final foundation laid by J. P. Staab and colleague’s in 2000 with defining the clinical
syndrome of chronic subjective dizziness (CSD). The birth of the newly defined diagnostic syndrome, PPPD, that literally worked to unify these key features, was released by the international community in the 2017 Barany consensus document for the classifications of vestibular disorders with the diagnosis being integrated into the WHO ICD-11 in 2018. PPPD is not a psychiatric diagnosis, but a functional diagnosis, and it is being proposed that the former more generic term of “psychogenic dizziness”, which blurred the lines between these entities, be questioned. Leading authority, J. P. Staab, has stated that the care and management of chronic vestibular syndromes will be greatly advanced if the 5 distinct behavioral entities of PPPD, generalized anxiety, panic attacks, depression, and the phobia of fear of falling are properly differentiated. (J. P. Staab, 2019) It needs to be remembered that isolated anxiety can account up to 8-10% of patients in tertiary care dizziness clinics (J. P. Staab, 2013). Failure to differentiate has historically offered no proposed pathogenesis and poor guidance to treatment when evidence supports greater differential diagnosis and, most importantly, better specificity of treatment prescription is possible.(Brandt, 1996; Eckhardt-Henn et al., 2008; J. P. Staab, 2006, 2016)

**PATHOPHYSIOLOGY:**

PPPD is believed to be a maladaptation, or complication to healing, that is most often triggered from an acute vertiginous event and results in a functional shift in the way the central nervous system integrates sensory information in certain individuals. This cortical shift causes increased weighting, or favoring, of visual stimuli over vestibular/proprioceptive cues and as a result creates heightened
attention to motion, heighten autonomic reactions from perceived threat with an end result of visual/body motion intolerance and overall cautious/insecure mobility. There is believed to be a role of behavior conditioning, where a patient 1. experiences an innate threat (vertigo crisis) that in turn leads 2. increased motion awareness with a decreased capacity to properly integrate/understand, and 3. through behavioral conditioning develops PPPD in patients who, due to genetic predisposing or vulnerable temperaments, respond to initial triggering event with high anxiety, catastrophic thinking, and/or body vigilance. PPPD represents a functional shift in neurosynaptic behavior and to date has not been found not due to any structural insult in brain or ear and therefore cannot be identified thru imaging or balance function testing (e.g., MRI or VNG). The bottom line with PPPD is, that whatever initially started or triggered the symptoms, is often no longer responsible for the ongoing, chronic nature of the symptoms and therefore should not be treated or managed the same as the original symptoms. Behavioral models for both the putative mechanisms of PPPD(Popkirov et al., 2018; J. P. Staab, Eckhartdt-Henn A., Horii A., Jacob R. Strupp M., Brandt T., Bronstein A., 2017) and the cognitive behavior component(Whalley, 2017) have been developed and are expanding patient/clinician understanding and allowing more refined care and further research. (see figure 1)
Deeper neuroanatomical and neurophysiological understanding of PPPD is also advancing alongside of the behavioral advancements. Following up on earlier fMRI studies that showed widespread changes in brain networks responsible for spacetmotion (parietoinsular vestibular cortex, visual cortex, and hippocampus)(Indovina et al., 2015), a more recent follow-up fMRI study looking 15 patients with PPPD being stimulated with a “virtual reality rollercoaster” found definably altered responses in insular and occipital regions compared to healthy controls that correlated with levels of perceived disability. (Riccelli et al., 2017) Sohsten et al completed the first investigation of posturography in patients with recently established criteria for PPPD.(Sohsten, Bittar, & Staab, 2016) Patients were found to
have degraded efficiency (more across the board elevated sway) and poorer
performance consistent with theoretic constructs of high risk postural control
strategies and multimodal spatial integration issues compared to controls. This
study calls into question established “aphysiologic test performance
criteria” (Longridge & Mallinson, 2005; Mallinson & Longridge, 2005) to patients
fitting criteria for PPPD.

**DIFFERENTIAL DIAGNOSIS**

Given that vestibular diagnostic testing and conventional imaging are generally
unremarkable, the art of properly identifying PPPD relies on: assessing quality
ongoing subjective complaints, determining what impairments are “driving” the
disability, and differentiating initial presentation versus ongoing presentation.
Patient’s suffering from PPPD will often have difficulty discussing the *quality* of their
dizziness, will describe *prolonged persistence* of that dizziness, describe unique
associations with fear/worry/catastrophizing, and will frequently offer the
pathognomonic descriptions of *environmental or task-provoked* triggers (e.g.
visually-provoked dizziness). There is a recently established ICD-11 code which is
listed under Diseases of the inner ear (10), Chronic Vestibular Syndromes (AB32),
and PPPD (AB32.0) where under the current ICD-10, there is no code other than the
non-specific code R42 for dizziness and giddiness. (WHO, 2018) PPPD diagnostic
criteria have been established and published by the international community with
diagnostic criteria emphasizing a clinical presentation of persistent non-vertiginous
dizziness or unsteadiness that has lasted 3 months or more that is exacerbated
when exposed to sudden moving/complex visual stimuli or during active/passive
head motions particularly when in upright that typically follows a balance-related problem. Box 1 lists these primary criteria with some expanded details to help with clarifying the criteria. (J. P. Staab, Eckhartdt-Henn A., Horii A., Jacob R. Strupp M., Brandt T., Bronstein A., 2017).

<table>
<thead>
<tr>
<th>Box 1. Adapted from the Barany Society International Classification Diagnostic Criteria for PPPD</th>
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<tbody>
<tr>
<td><strong>Primary Symptoms:</strong></td>
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<tr>
<td>- Dizziness or Non-spinning vertigo (vague, often hard for patient to express):</td>
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<tr>
<td>- rocking, swaying, bobbing, bouncing, cloudiness, fuzziness, fullness, heaviness, lightheadedness, visual focus not clear, orientation not sharp</td>
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<tr>
<td>- Unsteadiness:</td>
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<tr>
<td>- vague instability, wobbling, feelings of veering without directional preponderance (no specific direction)</td>
</tr>
<tr>
<td><strong>Duration:</strong> at least 3 months</td>
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<tr>
<td><strong>Tempo:</strong></td>
</tr>
<tr>
<td>- persistent, prolonged (hours), without specific provocation</td>
</tr>
<tr>
<td>- mild wax/wane qualities can be noted (often accumulation of provoking factors)</td>
</tr>
<tr>
<td>- present on most days (@least &gt;50, but often 24x7)</td>
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<tr>
<td><strong>Provoking Factors:</strong></td>
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<tr>
<td>- Exposures to complex visual motion demands or environments (e.g., moving visual, complex visual patterns, and/or visual precision tasks)</td>
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<tr>
<td>- Active passive head motion without directional preponderance (no specific position/ direction)</td>
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<tr>
<td>- Most severe when walking/standing i.e. upright posture (less, absent or very minor supine)</td>
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<tr>
<td><strong>Onset:</strong></td>
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<tr>
<td>- Sudden/distinct triggering or precipitant event that causes vertigo, unsteadiness, dizziness (can be intermittent initially consolidating to persistent OR may gradually worsen to persistence)</td>
</tr>
<tr>
<td>- Structural (70%)</td>
</tr>
<tr>
<td>- Acute/episodic/chronic vestibular syndromes (25-30%), migraine (15-20%), post concussion syndrome (10-15%),</td>
</tr>
<tr>
<td>- autonomic (7%) other medical illness crisis that disrupts postural control</td>
</tr>
<tr>
<td>- Psychiatric (30%)</td>
</tr>
<tr>
<td>- anxiety/panic (15%)</td>
</tr>
<tr>
<td>- psychological stress</td>
</tr>
<tr>
<td><strong>Disabling:</strong> Significant distress and/or definable functional impairment i.e. changed ADL/iADL capacity</td>
</tr>
<tr>
<td><strong>Symptoms not better accounted or attributed to ongoing neuro-otologic disease/disorder OR disease/disorder cannot fully explain all symptoms and/or level of disability</strong></td>
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It is critical that clinicians realize that the historical/subjective evidence, specifically that a patient meets ALL established criteria, is the key clinical finding in support for PPPD, just as the objective clinical finding of a positionally-provoked, paroxysmal,
torsional-up-beating nystagmus in the Dix-Hallpike is for supporting BPPV. As noted by the first item in the diagnostic criteria, differentiating the nature or quality of a patient’s primary subjective complain (e.g., vertigo, instability, dizziness, motion sickness, space-motion discomfort, or visual vertigo) is key to proper identification of PPPD. According to the International Classification of Vestibular Disorders (Bisdorff, Von Brevern, Lempert, & Newman-Toker, 2009) vertigo should be constrained to a “false or distorted sensation of movement”, unsteadiness a “feeling of rocking or swaying when upright, and dizziness as non-motion “sensation of disordered spatial orientation”. Motion sickness (e.g., car sickness) which is a malaise and nausea that can persist for minutes to hours following a real or illusory sensation or motion, can also often be uniquely identified, and where motion sickness is a common pathognomonic feature with migraine dysfunction, it is rarer as an isolated entity in PPPD. Similarly, differentiating oscillopsia from visual motion sensitivity or visual vertigo can be helpful. Where oscillopsia, or the perception of the visual world blurring/moving specific to when the head is moving, is the cardinal complaint of bilateral vestibular hypofunction, visual vertigo or visual motion sensitivity is the cardinal complaint of PPPD where visual motion sensitivity is NOT distinctively associated with head motion but rather triggered most often by passive watching of computer screens, reading, precision tasks and/or seeing busy patterns floors/walls. One non-published tool, see Box 2, that can be clinically useful not only in documenting disability but also in setting goals and designing treatment is a more formal activity analysis grid that integrates not only the use of either standard or customized Patient Specific Functional Scales (PSFS) which are 1-10
scales where 10 reflects normal or premorbid capacity/tolerance for that activity combined with a rating of the degree of avoidance from none to complete and any commented time limitations, i.e. shopping <5-10 minutes, only certain stores, etc. for common PPPD impacted activities.

<table>
<thead>
<tr>
<th></th>
<th>Rating:</th>
<th>Avoidance:</th>
<th>Limitation (time)</th>
</tr>
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<tbody>
<tr>
<td>Stores</td>
<td>___/10</td>
<td>0-----1----2-----3-----4</td>
<td></td>
</tr>
<tr>
<td>Computers</td>
<td>___/10</td>
<td>0-----1----2-----3-----4</td>
<td></td>
</tr>
<tr>
<td>Exercise</td>
<td>___/10</td>
<td>0-----1----2-----3-----4</td>
<td></td>
</tr>
<tr>
<td>Overall motion tolerance</td>
<td>___/10</td>
<td>0-----1----2-----3-----4</td>
<td></td>
</tr>
<tr>
<td>Driving</td>
<td>___/10</td>
<td>0-----1----2-----3-----4</td>
<td></td>
</tr>
<tr>
<td>ADL/iADLs</td>
<td>___/10</td>
<td>0-----1----2-----3-----4</td>
<td></td>
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Box 2: Example Activity analysis box looking at documenting a specific activities “normalcy compared to premorbid status where 10 is normal”, avoidance quantified 0-none, 1-little, 2-some, 3-much, 4-complete and any time limitation to that specific activity

Physical exam to r/o structural central or vestibular disease is completed and often is not remarkable with the normal findings being critical in the diagnosis of PPPD. Quantify patients motion sensitivity (visual and head/body) will be imperative given this is one of the key cardinal signs/symptoms supporting PPPD. Motion sensitivity will often be found to be diffuse, without directional preponderance (specific position or motion direction), without any accompanying nystagmus and often severe. Further details additional supportive clinical features are described in Box 3.

**BOX 3 Recommended Clinical Testing and Key Findings :**

- History/subjective
- Predisposing factors/vulnerabilities
- Neurotic, anxious, declined extroversion, obsessive compulsive personality tendencies
- Response to initial triggering event catastrophic
- High sensitivity/somatic focus
- Accompanying low levels anxiety (60%)/depression (25%)
- No associated anxiety/depression in 25%
  - Subjective reports of any fluctuations will be milder in intensity than with structural disease

- **Patient reported outcome measures**
  - High level of disability (e.g. >60/100 on Dizziness Handicap Inventory)
  - Symptoms exhausting and highly distressing
  - High disability on Visual Vertigo Analog Scale (VVAS) (E. Dannebaum, 2011; E. Dannebaum, 2019)
  - Clinically significant Space and Motion Discomfort as ID on Situational Vertigo Questionnaire (Pavlou, Davies, & Bronstein, 2006; Pavlou et al., 2016)

- **Balance function testing (e.g., VNG, Rotary Chair)**
  - Within Normal Limits (WNL)
  - Support no currently active disease i.e. compensation patterns,
  - Can’t fully explain symptoms/level of disability

- **Relatively normal clinical exam findings and balance capacity**
  - Normal but symptomatic Oculomotor testing normal (e.g. smooth, saccade, VOR cancellation testing)
  - Head impulse and positional vestibular testing normal (e.g. no corrective saccades or nystagmus) but symptomatic
  - Abnormal Motion Sensitivity (Motion Sensitivity Quotient or MSQ) (Shepard & Telian, 1995; Shepard, Telian, Smith-Wheelock, & Raj, 1993)
  - Overall integrity of volitional weight shifts, single leg/heel toe capacity but with variability, inefficiencies (increased sway, increased hip vs ankle strategies) and decreased confidence
  - Presence of safety behaviors (e.g., frequent touching walls, working to never be standing/walking unsupported)

- **Computerized Dynamic Posturography (CDP) Sensory Organization Testing (SOT) or Clinical Test for Interaction on Balance (CTSIB)**
  - Elevated postural sway “across the board” patterns
  - Elevated sway with inconsistencies particularly in visual or vestibular dysfunction patterns
  - Decreased tolerance especially in sway-referenced conditions (SOT 3/6)

PPPD certainly has strong associations with co-morbid anxiety, with one study showing 60% associated clinically significant anxiety, 45% clinically associated significant depression but with 25% having no psychiatric co-morbidity (J. P. Staab, 2012) Presence and severity of any significant secondary psychiatric or other functional comorbidity will affect treatment approach, treatment complexity and prognosis. Clinicians need to include ruling out for a more complex Functional Movement Disorders (FMD) and more severe psychiatric involvement (e.g., anxiety, depression, phobia) in the clinical workup of for PPPD. Presence of either could support the need for earlier and more comprehensive team interventions and a
possible more guarded prognosis. As mentioned earlier, there is growing evidence that if PPPD can be identified and treated early, these comorbid secondary complications could perhaps be, it not avoided, at least limited. Presence of secondary comorbidities has been found to increase the severity of disability. (Popkirov et al., 2018) Screening hints for identifying additional functional and psychiatric co-morbidities can be found in Box 4.

**Box 4. Screening for additional functional and psychiatric secondary co-morbidities:**

- **Functional Movement Disorder Screening**
  - **Oculomotor**
    - Functional ocular flutter
    - Functional convergence spasm
    - Refusal to perform testing but with evidence of normal smooth, saccade movements spontaneously
  - **Functional Gait Disorder**
    - Moment to moment fluctuations
    - Excessive slowness or hesitation
    - Sudden buckling of knees
    - Uneconomical posturing/mobility
    - Wasted muscular energy
    - Extreme caution and restricted step, walking on ice
    - Leg scissoring without evidence of spasticity
- **Postural**
  - Exaggerated sway on Romberg that improves with distractions
  - Excessive upper body sway
  - High variability performance/capacity
  - Severity of impairments that doesn't match disability
- **Computerized Dynamic Posturography**
  - Gross functional performance, i.e. large rhythmic/circular sway paths, delayed exaggerated responses to linear translations, walking into clinic without hx of falls and unable to perform any condition
- **Neurologic testing**
  - Tremors that disappear transiently or changes in rhythm when copying movements with good arm/leg
  - Difficulty making rhythmic movements with good arm/leg
  - Tremor absent particularly with distraction
  - Variable frequency of tremor
  - Tremor that gets worse when examiner tries to hold the arm or leg still
- **Psychiatric screening**
  - **Anxiety & Depression**
    - Hospital Anxiety Depression Scale (HADs) (Zigmond, 1983)
    - Positive Affect Negative Affects Scale (PANAS) (Watson, 1988)
    - [www.phqscreeners.com](http://www.phqscreeners.com) (no charge or copyright restrictions)
      - Patient Health Questionnaire (PHQ-9, PHQ-4)
      - Generalized Anxiety Disorders Scale (GAD-7)
  - **Phobia (Fear of falling)**
    - Activities Specific Balance Confidence Scale (ABC) (Powell, 1995)
In summary, proper diagnosis of PPPD requires analyzing the quality key subjective complaints, verifying if a patient's clinical presentation meets all established diagnostic criteria, ruling out any ongoing structural central/vestibular disorders, questioning if past disorder/deficit can explain ALL of the patient's current presentation, and modify prognosis and treatment prescription by taking into account degree and severity of secondary functional and psychiatric comorbidities. (J. P. Staab, 2019)

**TREATMENT:**

The role of VR in the treatment for PPPD has been proposed to be most effective when it is integrated with medication and cognitive behavior therapy (CBT) in what has been termed a 3 pronged approach. (J. P. Staab, 2012) Recent smaller studies utilizing combined approach of psychoeducation, CBT, VR, and antidepressant medications have documented 78% reduction in dizziness that is sustainable for 1-3 years. (Nada, Ibraheem, & Hassaan, 2019; Schaaf & Hesse, 2015)

**Medications**

The role and need for medications can certainly be dependent on PPPD severity, engrained chronicity, and just individual tolerance/preference. Treating clinicians should be aware and educate patient’ that the proposed role of medications in PPPD treatment is in helping to open neural pathways for healing and to decouple or calm heightened fight/flight responses while participating in VR. The most commonly utilized medications are anti-depressants which, despite having a common side
effect of dizziness, have been found effective in small titrated doses to approved
middle range. (J. P. Staab, 2012, 2019)

**Cognitive Behavioral Therapy**

The second prong, CBT, can often require treating VR clinicians to not only
understand principles for treatment and advocate for treatment when necessary but
also to identify, educate and actively network with allied behavioral clinicians in
their community/facilities. CBT approaches utilized in this population can include
treatment techniques to help patients be less somatically hypervigilant, develop
strategies for lessening anxiety (e.g., relaxation mindfulness training, distraction),
lessening cognitive distortions i.e. reframing catastrophic thinking, threat reduction
education, and lessening obsessive-compulsive controlling as well as perfectionist
tendencies. Techniques utilized by clinicians are similar for techniques use for non-
cardiac chest pain and irritable bowel syndrome. Trials of CBT in past decade
looking at PPPD predecessors “CSD and PPV”, support that CBT may be more
effective when PPPD is not fully engrained i.e. within few months of a triggering
event rather than when it is fully established which further supports the need for
2012; Holmberg, Karlberg, Harlacher, & Magnusson, 2007; Holmberg, Karlberg,
Harlacher, Rivano-Fischer, & Magnusson, 2006)

**Vestibular Rehabilitation**
Last, but certainly not least, is the role of VR rehabilitation in PPPD treatment. VR treatment techniques are being found vital in lessening impairments of visual and head/body motion intolerance, normalizing sensory balance processing, reducing high threat balance responses, normalizing balance confidence, lessening fatigue, and lessening autonomic dysregulation. Effectiveness of PPPD has been documented not only with growing clinical anecdotal success of leading authorities but also with several uncontrolled medication trials and moderately sized controlled investigations of VR. Treatments often include providing empowering education, visual motion desensitization, progressive habituation, sensory balance retraining, fitness progressions, core stabilization, dissociative movement retraining, motoric balance retraining for efficiency and confidence, and integrative applied relaxation. (Popkirov et al., 2018; Whalley, 2017).

Education

Education principals include teaching an understanding of the pathophysiology of PPPD and providing evidence whenever possible that the triggering event is healed. Providing evidence concerning the brain’s capacity to heal, particularly given the normal findings that represent “assets to healing” where non-localizing/normal findings historically have led only to stress and disappointment. Empowering patients with the concept that literally what triggers their dizziness, if carefully dosed over long enough period of time, can heal them especially when coupled with integrated relaxation responses. Educating them on the capacity to lessening their visual motion intolerance by helping their brain learn to choose the correct sense
and better utilize the powerful resources of inner ear and ground/proprioception. Education concerning that yes, they will need to carefully pace provocative activities and exposures to certain environments, but that this can steadily improve when not being completely avoided and power of balancing graded exposures with sufficient rest/sleep. And finally, informing them of the powerful website that has developed to better standardize education and offer patient support for patient’s suffering from functional disorders with a printed fact sheet about PPPD available called neurosymptoms.org that can be found at:


Relaxation training and integration

Current research, and most commonly discussed advancements, in the treatment of PPPD revolves almost solely around visual motion desensitization techniques, which is understandable given the primary pathognomonic PPPD impairment is visual vertigo or visual motion intolerance. However, the art of PPPD from anecdotal experience supports that diving to quickly and aggressively with visual motion progressions without first giving patients some foundational skills related to decoupling fear-based responses, i.e. relaxation training, and more “grounding” sensory processing, i.e. normalizing sensory weighting, can set patient’s up to fail. Military Studies completed in the 80’s demonstrated the effectiveness of integrating habituation with relaxation in lessening motion sickness (air sickness) in pilots.(Jones, Levy, Gardner, Marsh, & Patterson, 1985) Pilots, who were grounded secondary to irretractable airsickness, were trained in autogenic biofeedback
relaxation techniques (muscle tension/relaxation, warming hands, diaphragmatic breathing and mental imagery) and then exposed them to graded rotary chair motion exposures (20 sessions, 30-45 min twice a day for 2 weeks) where they would integrate the techniques they had learned in increasing complexity of motion exposures with 79% of pilots being able to return to full duty which was sustained. It was theorized the treatment helped by both inhibiting the involuntary autonomic response to the motion stimuli i.e. habituating the response and by alleviating the anxiety concerning becoming sick. There is now increasing evidence on the role of mindfulness and structured relaxation practice on fostering healing across many diagnoses and intuitively is particularly needed in a diagnosis where faulty neuroprocessing understandably heightens flight/fight defensiveness and causes disconnects from patient’s internal assets which are not lost or broken but waiting to be awoken and trusted-in once again as a foundational principal of PPPD healing. Anecdotal experience has found that teaching some level of relaxation prior to any more aggressive habituation and visual motion desensitization as well as applying relaxation during treatment progressions fosters intrinsic strategies for success as patient’s progress through the necessary graded provocative exposures. Again, the overarching mantra in all of PPPD-based VR is that “what makes you dizzy can heal you” if you can just grade it carefully enough but most importantly link that exposure to sense of trust and control. Depending on clinician’s skill as well as patient’s presentation and personality, this training can consist of referral to specialized practitioner, educating on multitude of available podcast/cell phone app programs, or teaching fundamental skills of breath, muscle tension awareness and
mindfulness in the clinic. What is important, no matter what the approach, is that all future habituation/balance VR training needs to be done with more finesse and with greater attention not only to the exercise progression you are giving but to how the patient is responding to that exercise much more that with structural peripheral vestibular VR. Evidence is lacking as to the exact role of integrated relaxation in PPPD treatment i.e. relaxation applied in conjunction with balance/habituation efforts but is often implied in descriptions of how various exercise paradigms are prescribed in successful treatment programs, often being a key aspect of any counseling/CBT interventions.

**Balance Retraining**

Balance retraining is a fundamental aspect of VR(Best et al., 2015; Shepard & Telian, 1995; Shepard et al., 1993) and particularly crucial in addressing the underlying PPPD pathophysiology of sensory misweighting. Initial techniques generally involve some type of proprioceptive uptraining or what has been termed “surface orienting or grounding” where patients are taught to selectively attend to weight bearing, muscle stabilization, and ground stability perceptions to lessen perceptions of dizziness. Activities often look similar to core stabilization programs and can start in supine/sitting and progress to more challenging postures and walking. Applied Pilates, Feldenkrais and/or yoga techniques can often be facilitory. Eyes closed balance progressions both with narrowing base of support (e.g. static center of gravity holding) and active weight shifts (e.g. limits of stability work) are common activities with end goal to evolve to full vestibular uptraining where both ground
and vision are altered with progressions from static to more dynamic demands. Drills where patient’s “bathe in inner ear” processing and learn to trust this deepest vestibular procession, which is best taught by cuing to the orientation to vertical and that evidence of honing responses that pulls us back to midline, is critical for success as patients with PPPD can just as easily become “ground dependent” as they are visually dependent. There is often a loss of dissociative motions in patients with PPPD, in which case, integration of Tai Chi type movements or dance applications are indicated. A particular area of balance retraining unique to PPPD is what could perhaps be termed “weight shift desensitization”. Patients with PPPD can display a hypervigilance to what are normal subtle postural responses, particularly if they come unexpected (e.g., a slight shift in a chair, being nudged, or normal subtle static sway). These motions can become perceived as highly threatening and in some cases highly provocative and often lead to development of what has been described as “safety behaviors” or patients feeling the need to always have a 3rd point of contact at all times which is usually a upper extremity reach/contact. Treatment involves literally working to increase the range of postural disturb patients are willing to feel and work to develop confidence in intrinsic postural responses without the need for additional contacts. Strategies include providing patients with successful movement experiences and literally challenging patients to get there head up, look ahead and stop touching walls. It is also imperative to, whenever possible, stop the use of inappropriate assistive devices.

Habituation
Habituation is perhaps the most cardinal and mainstay of any VR treatment and is main therapy technique to resolve motion intolerance issues associate with PPPD. (J. P. Staab, 2011) However in PPPD VR as noted earlier, prescriptions need to be carefully doses with emphasis on grounding/integrated relaxation and often require long rest breaks. Control of symptom de-escalation is imperative and treating clinicians need to identify exact triggers to make the treatment prescription the most effective. Generally, doses are prescribed in 3-5 repetitions, 3 different motions 2-3 x/day with the guideline that all symptoms should return to baseline within 20 minutes. Habituation is generally integrated with balance retraining in final progressions.

Visual Motion retraining and progressive systematic desensitization

As mentioned earlier, one of the most steadily advancing fields in PPPD treatment is the role of optokinetic or visual motion desensitization programs. There is mounting evidence that habituation exercises in the form of visually provocative scenes can lessen PPPD disability. Vitte and Semont in 1994 exposed patients with vestibular deficits to 8-15 minute sessions of optokinetic flow while on the treadmill and documented decreased symptoms, improved sensory organization, improved symmetry of optokinetic nystagmus, and most importantly improved tolerance to shopping and being in busy environments. (Vitte, Semont, & Berthoz, 1994) Some of the most pioneering work has been done by leading authority, M. Pavlou, with her seminal work in collaboration with Dr Bronstein in 2004 with the birth of “simulator-based rehabilitation" for refractory dizziness and with her development
of validated symptom questionnaire specific for quantifying the visual/situational
tolerance so classic in PPPD presentation called the Situational Vertigo
Questionnaire (SVQ) that was just recently also validated in children. (Pavlou et al.,
2006; Pavlou, Lingeswaran, Davies, Gresty, & Bronstein, 2004; Pavlou et al., 2016;
Pavlou et al., 2017) Desensitization of visual pathways has been found in some
studies to be more difficult to treat than sensitivity to body motions. (Thompson,
Goetting, Staab, & Shepard, 2015) Treatment strategies generally consist of starting
when necessary with exposures (1-2 minutes, 2-3x/day as symptoms allow) of
isolate eye motion exercises (smooth pursuit, saccades, ball catching/throwing,
marble watching), computer/phone app eye games, progressing to fuller field
(waving fans, spinning large umbrellas), to more situational-based exposures most
commonly utilized on TV screens with “youTUBE” video motion exposures or disco
ball rooms. Increasing access to virtual reality is becoming more utilized both with
head mounted devices and full field devices such as Bertec posturography
technology. Whenever possible, systematic desensitization in community
environments is also progressed i.e. grocery store/library progressions. What has
been well defined in the literature is that treatment of PPPD is not completed until
phobic avoidance is no longer being reported. Exact dosing and best prescription is
unknown with much more research needed but what is very evident is every
prescription needs to be highly individualize for optimal success.

Responsiveness
It is felt that if PPPD is ID and addressed early (within weeks to months of onset that 3-6 treatment sessions often with primary education, simple habituation/balance progressions highly effective. If PPPD is, what has been termed “engrained” meaning it is long standing and/or complex (e.g., increased co-morbid issues), then it can often be a process of reclaiming “one’s life” gradually over months to years. In cases of more engrained PPPD, 8-12 sessions over 6 months is not uncommon, with occasional need to adopt a “dental model” of therapy where a patients are established on an optimal home exercise program and then brought back 6-12 months later to reassess and advance once again. Whenever possible, finding community resources for promoting ongoing wellness can be very augmentative i.e. tai chi, pilates, dance, senior fitness. Dr Pavlov’s work has supported that PPPD needs to be “in remission” before treatment is stopped, i.e. no phobic avoidance, no ongoing safety behaviors/crutches and optimal activity level with the quickest way to no treatment, full treatment. PPPD once in remission can be vulnerable to exacerbation often associated with stress, stopping medications, illness or change in activity level. Although not documented in the literature, anecdotal observation has shown exacerbations to be different than the initial presentations with higher levels of anxiety and less visual motion disability. VR is being found to be a key modality for successful treatment of PPPD. (Dieterich & Staab, 2017; Dieterich et al., 2016; Popkirov et al., 2018; Seemungal & Passamonti, 2018)

**SUMMARY:**
PPPD is relatively new but common diagnosis when properly identified for which VR is being found integral for optimal management both in VR’s capacity to provide clinical diagnostic evidence and its capacity to offer unique customizable treatment approaches. With the recent establishment of diagnostic criteria, PPPD is being found not just to be a diagnosis of exclusion or garbage term for anything not common or psychogenic but is distinguishable not only from structural disorders but also from psychiatric morbidities which, if embraced, could allow for more timely and best patient care. VR treatment for PPPD will frequently need to tap into a multidisciplinary team approaches tapping not only into exercise-based approaches but also CBT and/or medication with anticipated healing occurring over months to years for optimal results in many cases. Multiple VR strategies including education, habituation, balance retraining, relaxation training/integration, and visual motion desensitization are being found promote optimal recovery that is sustainable. Future studies are needed to better define interdependence of various treatment modalities and most effective specific treatment strategies.


