

**Title:**

A Novel Treatment for Persistent Postural Perceptual Dizziness : A Case Report

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**Competing interests**

None.

**Abstract:**

A case of Persistent Postural Perceptual Dizziness (PPPD) or (3PD) presented to a Chiropractor with significant symptoms and few neurological signs. This condition had been present for over three decades. These, largely vestibular signs and symptoms included: 'foggy brain', difficulty concentrating and reading, an almost constant feeling of disorientation, a feeling of "motion sickness", and poor balance. Additional symptoms included peripheral vision being sensitive to bright light, phono-phobia and an intolerance to any constant background noise.

Other symptoms of note included that when travelling in a car, the patient would feel like the vehicle was still moving for about twenty seconds after the car had stopped.

He suffered dizziness when arising from the supine to sitting position, especially upon awakening and had reduced neck mobility, which was the reason for his initial consultation to a Chiropractor. Also, upon lifting binoculars to his eyes, he would fall forward to the degree that he had to brace himself before lifting the binoculars up to his face.

The patient had sought various treatments in the past and was given several diagnoses including: Motion Sickness, Benign Paroxysmal Positional Vertigo (BPPV) and 'vertigo'. Treatment has previously included medication from several general medical practitioners, two neurologists and multiple sessions of physiotherapy. The patient claimed the treatment for BPPV by a physiotherapist reduced symptoms by 50% but the treatment effects did not last beyond 3 days. Medications of various types were reported as ineffective.

Novel vestibular rehabilitation techniques, included using a rotating chair, saccade training, a reaction timer panel and home exercises were utilised to good effect. Four weeks of treatment, alleviated the condition for the first time in over 30 years. The methods used were novel and not considered within the realm of typical general chiropractic management. This case presents a novel 'Functional Neurological' approach to management of a condition by a Chiropractor with a special interest in vestibular rehabilitation.

**Key Words:** PPPD, Chiropractic, Vestibular Rehabilitation, Rotating Chair, Sensory-Motor connectivity.

**Introduction:**

Persistent Postural Perceptual Dizziness (PPPD) is a new condition to describe dizziness symptoms that is a diagnosis of exclusion<sup>(1)</sup>. There is not a consistent set of signs, symptoms or objective findings that clearly define it from the many other vestibular conditions. The ‘History ’ is very important in contributing to the diagnosis. The condition is **Persistent**, usually episodic, **Postural**, in that the signs and symptoms diminish or exacerbate in some postural positions and **Perceptual** in that the **Dizziness** (not spinning) is difficult to objectify and measure. It is a functional vestibular disorder often with conventional diagnostic tests and imaging being negative.<sup>(30)</sup>

It is considered that PPPD is usually a long term maladaptation resulting from an episode of a vertiginous disease or trauma. Anxiety is also thought to initiate the condition <sup>(1,9,10)</sup>. This case utilised vestibular rehabilitation as the context for intervention. In particular the use of spinal manipulation/adjustment, sensorimotor integration exercises, gaze stability exercises and “rotating chair” manoeuvres. A positive, clinically demonstrative result was achieved. However a clear delineation of which interventions were successful and to what degree of success each intervention contributed was not achieved. The purpose of this case report was to contribute to the increasing body of knowledge of the effectiveness of Chiropractic care and expanding the ‘scope of practice’ of Chiropractors.

**Patient information**

The patient was a left handed, Caucasian, 51 year old male with considerable entrepreneurial business ability. He presented as intelligent, articulate and with an affable personality. The patient’s symptoms/concerns are presented in table 1.

Table 1: Patient symptoms/concerns

The main patient concerns were:	Brain Fog
	Disorientation
	Poor concentration
	Difficulty with reading
	Motion sickness
	Poor balance
	Phonophobia
	Light sensitivity of peripheral vision
	Feeling of movement after stopping
	Intolerance to background noise
	Dizziness when arising from lying to sitting
	Decreased neck mobility
	Becoming unbalanced when using binoculars

The patient had seen many practitioners over the years and had multiple examinations including sophisticated investigations like MRI brain scans, Orthostatic testing, Cardiovascular, Ophthalmological, Neurological and Vestibular examinations. These ruled out the other more clearly defined conditions but left the patient with an unsatisfied conclusion and no effective diagnosis or treatment for his 'dizziness'. The author considered that a new and detailed neurological examination might elucidate a mix of subtle neurological signs and symptoms that would shed light upon this man's persistent condition.

### Clinical findings

From the test results below combined with the history, a provisional diagnosis of PPPD was made. The other considerations included other non-acute vertiginous conditions such as Mal De Debarquement Syndrome, Vestibular Migraine, Benign Paroxysmal Positional Vertigo, Visually Induced Dizziness, Meniere's Disease, Motion Sickness, Cervicogenic Dizziness and Anxiety.

Upon presentation, the patient was given an extensive battery of tests with an emphasis on the vestibular system. These tests were chosen to differentiate the vestibular conditions mentioned above with the goal of gleaning information that could guide treatment. Tables 2 & 3 contain the tests that were performed and their outcomes.

Table 2: Tests and results of vestibular testing

	Test	Result	Interpretation - visual, vestibular, proprioceptive
1	Dizziness Handicap Inventory	Pre and Post Treatment testing showed only slight improvement being (12%)	Characteristic of 3PD
2	Standing on one foot with eyes closed	Very poor bilaterally	Proprioceptive +/-or Vestibular system dysfunction
3	Near Point Convergence	14 cms (N<6cms)	Visual system dysfunction
4	Maddox Rod Testing	Minor convergence	Visual system dysfunction
5	Occlusomotor Pursuits - Horizontal	Saccadic intrusions at +/- 30° bilaterally	Vestibular &/or visual system dysfunction
6	Occlusomotor Pursuits - Vertical	Pursuit breakdown at +15° with motor recruitment (ie. head movement).	Vestibular &/or visual system dysfunction
7	Subjective Visual Vertical (SVV)	No Abnormality detected	Peripheral vestibular (otolithic) apparatus

8	Voluntary Saccades	Upper hemifield dysmetria	Visual system dysfunction
9	Reflexive Saccades	Upper hemifield dysmetria	Visual system dysfunction
10	Motion Sensitivity Test	Positive with a severe reaction at only 5 seconds	Vestibular system dysfunction
11	Velocity Storage Testing (10 rotations @ 2 Hz)	The Optokinetic Nystagmus of Left rotation was 82secs, Right 80secs.	Vestibular system dysfunction
12	Dix-Hallpike and Lateral Roll semicircular canal manoeuvre.	Unremarkable	Vestibular system dysfunction (peripheral)
13	Head Shake Nystagmus	Positive with 8 beats	Vestibular system dysfunction <sup>(32)</sup>
14	Dynamic Visual Acuity Testing (DVAT)	Unremarkable	Vestibular &/or visual system dysfunction
15	Optokinetic Tape pursuits	Right sided dysmetria and motor recruitment of the head	Visual system dysfunction
16	Spontaneous Nystagmus	Evident with visual suppression	Visual system dysfunction
17	Gaze Evoked Nystagmus	Occurred on the Right with visual suppression	Visual system dysfunction
18	Cervical Joint Position Testing	Showed a hypometric dysfunctional bias to the right. see fig 1	Cervical proprioception dysfunction <sup>(33)</sup>
19	Neck Torsion Test	Left rotation showed a greater pursuit breakdown	Proprioceptive +/-or visual system dysfunction

20	Finger Tapping	Unremarkable, (left dominant)	Proprioceptive dysfunction
21	Raglans Test	Unremarkable	Dysautonomia, cardio vascular haemodynamic feedback control
22	Tilt Table testing	Unremarkable	Dysautonomia, cardio vascular haemodynamic feedforward control
23	Supine Oculo-cardiac Reflex testing for Heart Rate change.	Unremarkable	Dysautonomia, cardio vascular haemodynamic feedback control
24	Squat to standing Oculo-cardiac Reflex testing for Heart Rate change.	Unremarkable	Dysautonomia, cardio vascular haemodynamic feedback control
25	Supine Carotid Body Reflex testing for Heart rate change.	Unremarkable	Dysautonomia, cardio vascular haemodynamic feedback control
26	Heart Rate Variability Testing	Paradoxical response being an increase in sympathetic activation upon resting and deep breathing.	Dysautonomia
27	Saccadometry	See fig 2.	
	Latency Profile	Bilateral spontaneous saccades	Inappropriate brain stem initiation of voluntary saccades bilaterally
	Velocity Profile	Abrupt velocity changes midway through the saccades	Neural integration of the saccade was defective

	Phase Profile (eye position Vs Velocity)	Saccadic intrusions evident	Visual system dysfunction
28	Force Plate Analysis	Decreased vestibular balance contribution	Vestibular system dysfunction
29	Pupillometry - 'reflex App'	The Average Constriction Speed, Constriction Time, Average Dilation Speed and the Release Amplitude were greater than two standard deviations from normal bilaterally.	Functionally these results suggest both sympathetic and parasympathetic aberrations.
	Quantitative Electroencephalography (QEEG)	High delta activity at temporal sites  Theta and alpha activity was normal,  Slow beta activity at 12-15 Hz had a high amplitude, high beta activity at frontal sites	? Impaired memory, ? hypo-arousal.  Hypo-arousal, impaired vigilance regulation  ? Anxiety. Sensory-motor network connectivity was diminished.

## Diagnostic Assessment

With the extensive list of tests performed, only a few positive tests were returned. Table 3 lists tests which were negative but consequently assisted in the 'diagnosis by exclusion'

Table 3: Patient testing results

Tilt table testing for assessing the otolithic feed forward regulation for the prediction of cardiovascular needs, was unremarkable.

The oculo-cardiac and carotid body reflexes (using digital pressure and a Pulse Oximeter for Heart Rate monitoring) were unremarkable.

Heart rate monitoring of squatting to standing which relies on the negative feedback for cardiovascular regulation. Also negative.

Raglans test was negative and in combination with the above, surprisingly, dysautonomia signs were not outstanding.

The results of these tests help lead to the conclusion that there were symptoms of multiple vestibular conditions including: Motion Sickness, Visually Induced Dizziness<sup>(2)</sup>, Vestibular Migraine<sup>(3)</sup>, Mal De Barquement Syndrome<sup>(4)</sup>, Benign Paroxysmal Positional Vertigo<sup>(5,6)</sup>, PPPD<sup>(7)</sup>.

According to the Bárány Society, the diagnostic criteria for persistent postural-perceptual dizziness<sup>(8)</sup> are:

*“One or more symptoms of dizziness, unsteadiness or non-spinning vertigo on most days for at least 3 months. Symptoms last for prolonged (hours-long) periods of time, but may wax and wane in severity. Symptoms need not be present continuously throughout the entire day. Persistent symptoms occur without specific provocation, but are exacerbated by three factors: upright posture, active or passive motion without regard to direction or position, and exposure to moving visual stimuli or complex visual patterns. The disorder is triggered by events that cause vertigo, unsteadiness, dizziness, or problems with balance, including acute, episodic or chronic vestibular syndromes, other neurological or medical illnesses, and psychological distress. When triggered by an acute or episodic precipitant, symptoms settle into the pattern of criterion A as the precipitant resolves, but may occur intermittently at first, and then consolidate into a persistent course. When triggered by a chronic precipitant, symptoms may develop slowly at first and worsen gradually. Symptoms cause significant distress or functional impairment. Symptoms are not better accounted for by another disease or disorder.”*

From the literature<sup>(1,7, 11)</sup> it is possible PPPD occurs from a previous acute vertiginous condition like: labyrinthitis, vestibular neuritis, and herpes zoster oticus, none of which were evident from the patient history. The literature further claims that PPPD can occur from the many conditions that may involve the vestibular system<sup>(32)</sup>, causing vertigo to dominate as a short episode years before. Episodes may be caused by an acute febrile infection, neck pathology, autoimmune conditions, dysautonomia or ototoxicity<sup>(7,11,36)</sup>. Other possible predisposing factors can be acute anxiety. However, this case did not involve anxiety as an issue.

Regardless of the initiating aetiology, it appears the nervous system may habituate the ‘feeling’ of imbalance and maintains this ‘feeling’. The consequent maladaptive behaviour modification<sup>(23)</sup> further habituates a decreased sensory-motor integration and a plethora of symptoms can develop over time. For example, as previously mentioned, anxiety is associated with PPPD. It is



thought that the feeling of constant imbalance creates anxiety from the stress of constant vigilance to prevent falling.<sup>(19)</sup>

**Therapeutic Intervention**

Considering the prolonged history and the findings of the physical exam, a multimodal management approach was applied. In particular, the vestibular system, required rehabilitation along with improved visual and sensorimotor integration. The Vestibulo-Ocular Reflex (VOR) was likely maladapted. Although the DVAT test (horizontal VOR) was negative it could be that the other, unexamined, VOR Reflexes such as the Vertical, Angular or Translational VOR Reflexes were aberrant. Considering the visually induced dizziness symptoms were marked, this is a likely possibility.

This ‘systems dysfunction’ conclusion was devised by considering the premise of PPPD to be an habituation of poor balance with concomitant reduced movement and consequent adoption of ‘safe’ or protective postures. Consequent behaviour of reduced movement led to an ongoing reduction in proprioceptive and visual inputs, further negatively influencing the perception of balance<sup>(35)</sup>. Treatment was designed to address the positive tests which are listed in Table 3.

Table 3: Vestibular Examination Test results

Positive Vestibular Examination Test results were:	Poor standing balance
	Motion sensitivity testing
	Velocity storage results
	Head shake nystagmus being positive
	Saccadometry analysis
	Force plate analysis
Positive Visual System Examination tests were:	Near point convergence
	Oculo-motor pursuits
	Voluntary and reflexive saccades

	OPK tape pursuits
	Spontaneous and gaze evoked nystagmus
	Pupillometry
	Saccadometry
Positive Sensory-Motor System Tests were:	Cervical range of motion restriction
	Cervical joint position error testing
	Poor standing balance
	Neck torsion test

An extensive multimodal treatment plan was created that focused on musculoskeletal, vestibular, oculomotor and sensorimotor systems. The frequency of care was twice per week for five weeks, see Table 5. The content of this protocol is contained in Table 4.

Table 4: Multimodal treatment plan focusing on musculoskeletal, vestibular, oculomotor and sensorimotor systems.

Multimodal treatment components	
	Spinal manipulation to areas of restricted spinal movement including: upper cervical, mid thoracic and lumbo-sacral junction
	Right lateral rolls using the custom made orbital motion simulator.
	Right yaw using the orbital motion simulator. The 'Yaw' direction is simply turning around in a circle about the vertical midline axis.
	Entrainment therapy using a computer program (see 'Discussion')

	VOR adaption training using a custom made reaction timer
	Sensory motor integration training using a custom made lighted panel (referred to as SMIRT)
Home Exercises Components	'0' gain VOR - The patient focuses on their outstretched thumb and rotates their trunk, arm and head together in an arc of 80 degrees either side of centre taking only 2 seconds to complete, repeatedly for 1 minute.
	X2 VOR exercise for gaze stabilisation involves sitting with the arm outstretched in front and staring at your thumb. Then slowly sweeping your arm left and right for 1 minute 80deg either side of centre while fixating your gaze upon the thumb continuously.
	One leg teeth exercise - The patient is instructed to carefully stand on one foot with their eyes closed while brushing their teeth. Twice daily.
	Near point convergence Exercise - using a 200mm card held on the tip of the nose and pointing forward, the patient is to retrain the horizontal vergence neural integrator by reading letters up and down the card. Like a 'Pencil Pushup'

**Table 5: Treatment Timeline**

Consul tation	Reported Changes in Signs & Symptoms	Home Exercises	Treatment Given	Entrasinment Therapy	Sensory Motor Integration Reaction Timer	VOR Training Horiz & Vertical
1	Assessment	0 'Gain VOR exercise. Perform for 30 secs 4/day.	Spinal adjustments	not performed	not performed	not performed
2	Unchanged	as above and Vergence Card use 30secs 4/day, One Leg Teeth Ex 2/day	Spinal adjustments			
3	Dizziness has changed, "it now comes in waves"  '0 'Gain VOR Ex is less uncomfortable	as above	Spinal Adjustments OMS - Rt Yaw 120deg/sec 1 min. OMS -Right Rolls X4 @ 90deg/sec Optokinetic Nystagmus Type 1 (OKN1) - 30secs	200 Trials on firm surface  34% accuracy	180 trials Gap Condition 0.8/1.2s Ave Reaction time 0.590s Missed 27/180	VOR Adaption Training - Gap condition 0.8/1.2secs while standing on a Vibrating Plate
4	"a lot better - was 'off 'all day post last Tx, less 'dizziness ' generally, supine to sitting 'dizziness 'has gone"	as above	Spinal Adjustments OMS - Rt Yaw 120deg/sec 1 min. OMS -Right Rolls X4 @ 120deg/sec OKN1 - 25secs	200 Trials X 2 while standing on a Vibrating Plate Accuracy 1st attempt- 43%, 2nd attempt-30%	180 trials X 2 Gap Condition 0.7/1.1s Ave Reaction time 1 - 0.551s Missed 43/180 2 - 0.554 missed 30/180	not performed

Consultation	Reported Changes in Signs & Symptoms	Home Exercises	Treatment Given	Entrasiment Therapy	Sensory Motor Integration Reaction Timer	VOR Training Horiz & Vertical
5	"Improved, 'foggy 'in mornings"	as above	Spinal Adjustments OMS - Rt Yaw 120deg/sec 1 min. OMS -Right Rolls X4 @ 120deg/sec OKN - 30 secs	200 Trials X 2 while standing on a Vibrating Plate Accuracy 45%, 42%	180 trials X 2 Gap Condition 0.7/1.1s Ave Reaction time 1st - 0.551s Missed 43/180 2nd - 0.554 missed 30/180	not performed
6	"bad week, woke 'foggy '4 days ago"	as above	Spinal Adjustments OMS - Rt Yaw 120deg/sec 1 min. OMS -Right Rolls X4 @ 120deg/sec OKN1 - 25secs	200 Trials X 2 while standing on a Vibrating Plate Accuracy 41%, 54%	180 trials X 2 standing on perturbed surface (Foam) Gap Condition 0.7/1.1s Ave Reaction time 1 - 0.548s Missed 22/180 2 - 0.562 missed 41/180	not performed
7	"good, no fogginess today"	as above	Spinal Adjustments OMS - Rt Yaw 120deg/sec 1 min. OMS -Right Rolls X5 @ 120deg/sec OKN1 - 20secs	250 Trials X 2 on Vibration Plate Accuracy 56% 62%	180 trials X 2 standing on perturbed surface (Foam) Gap Condition 0.6/1.0s Ave Reaction time 1 - 0.504s Missed 52/180 2 - 0.493 missed 44/180	not performed

Consultation	Reported Changes in Signs & Symptoms	Home Exercises	Treatment Given	Entrainment Therapy	Sensory Motor Integration Reaction Timer	VOR Training Horiz & Vertical
8	“fogginess 2/10 - good since last visit”	as above X2 VOR Ex ( stop the '0 'gain ex)	Spinal Adjustments OMS - Rt Yaw 120deg/sec 1 min. OMS -Right Rolls X6 @ 120deg/sec OKN1 - 20secs	250 Trials X 2 on Vibration Plate Accuracy 56% 56%	180 trials X 2 standing on perturbed surface (Foam) Gap Condition 0.6/1.0s Ave Reaction time 1 - 0.476s Missed 41/180 2 - 0.484 missed 40/180	Gaze stability Exercises using the Focus Builder OPK projection with central fixation for 3 minutes
9	“fogginess 2/10 only on boat in rough conditions.	as above X2 VOR Ex	Spinal Adjustments OMS - Anterior Pitch X3 120deg/sec.	250 Trials X 2 on Vibration Plate Accuracy 65% 49%	180 trials X 2 standing on perturbed surface (Foam) Overlap Condition 0.7s Ave Reaction time 1 - 0.494s Missed 28/180 2 - 0.476 missed 60/180	not performed
10	“fogginess good since last Tx Can now use Binoculars w'out forward tilt”	as above	Spinal Adjustments OMS - Anterior Pitch X 4 @ 120deg/sec with Visual Fixation. OMS -Right Yaw with Visual Fixation 120deg/sec 2mins.	250 Trials X 2 on Vibration Plate Accuracy 56% 64%	180 trials X 2 standing on perturbed surface (Soft Foam) Overlap Condition 0.6s Ave Reaction time 1 - 0.473s Missed 54/180 2 - 0.486 missed 76/180	not performed

**Follow Up and Outcomes**

Patient reported results were favourable following most consultations and steady progress was achieved. After five weeks and ten consultations the patient reported having whole days without 'fogginess' and no longer felt 'unbalanced'. Any remaining 'fogginess' experienced by the patient post treatment occurred while on a boat in rough conditions and was a mild '2/10'. His neck discomfort and reduced mobility was absent. Both he and his family were very pleased! Whilst symptoms were not completely alleviated, the goals set by the patient prior to treatment were achieved.

The Force Plate Analysis retesting showed only slight improvement. The Saccadometry retest was markedly improved but still showed some significant deviations from normal, briefly described below.

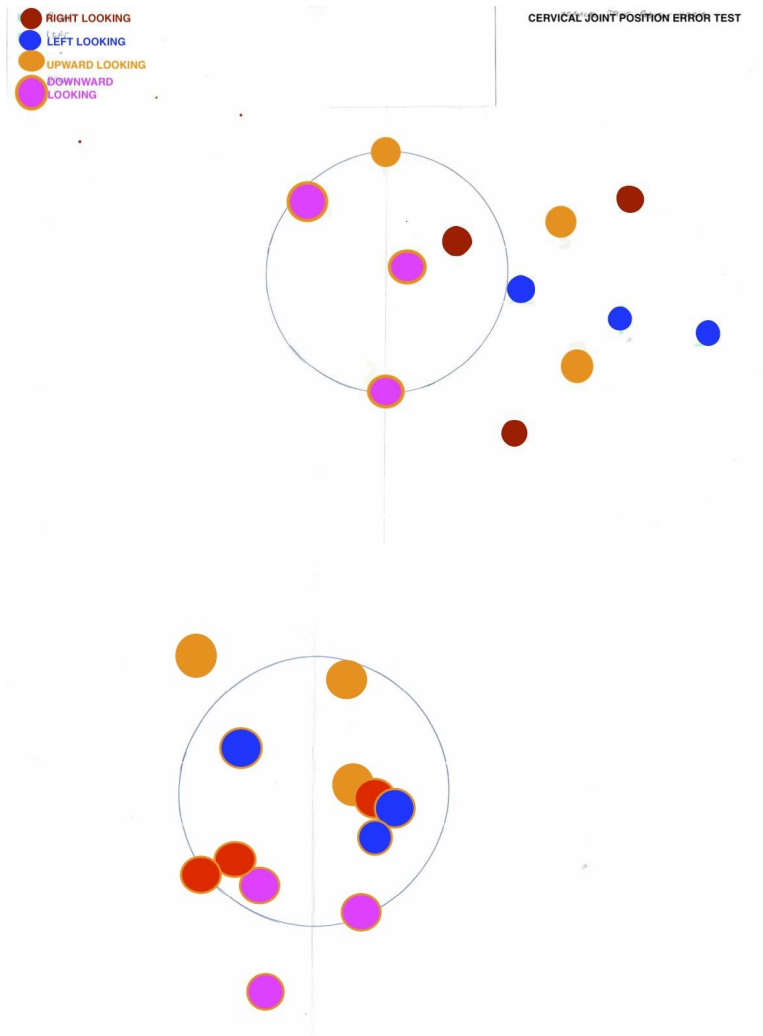
The Dizziness Handicap Inventory is the most widely used scale of dizziness self assessment.<sup>(41)</sup> It had improved from a pretreatment score of 32% to a post treatment score of 20% A score above 10% is considered clinically relevant.

### Interpretation of clinical tests

Figure 1: Cervical joint position error test.

Pre  
Treatment Testing

Post  
Treatment Testing



Pink results are from extending the cervical spine as if looking in the UP direction, but with eyes closed and returning to where the patient thinks is the neutral head position. This is repeated 3 times, Orange Down (Cervical Flexion), Red turning to the Right, Blue turning to the Left. Clearly the patient's pre treatment sense of self positioning was skewed.



These tests are used for the evaluation of the cervical proprioceptive influence contributing to the visual pursuit reflexes (see figure 1). A Normal result would have all 12 trials recorded within the Target Circle.

Each coloured dot is the end result of turning the head 70° and returning to the estimated centre target with eyes closed using a face mounted Laser and repeated three times. Head movement return position recordings inside the circle are considered normal. The subject recorded 4/12 pretreatment, 10/12 post treatment suggesting his cervical proprioceptive input was markedly improved.

Figure 2. Saccadometry before and after treatment.

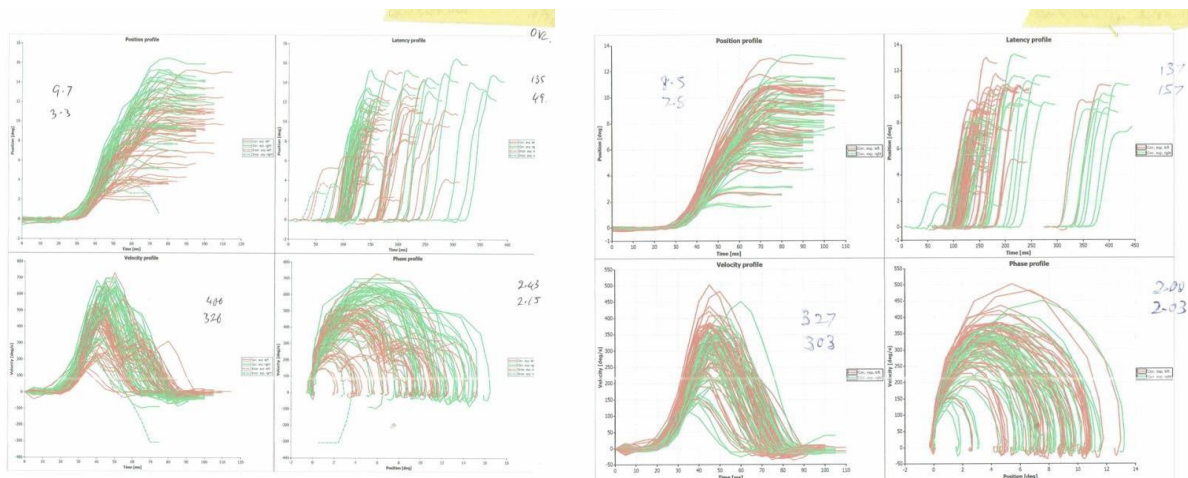
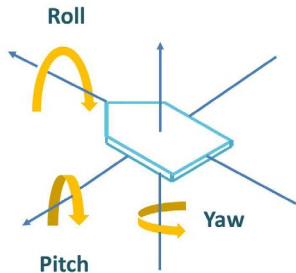


Figure 2 . There is some improvement in the saccadometry. A full explanation of the saccadometry results is beyond the scope of this document.

The green traces are for saccades to the right, red to the left. 100 trials in total. The top left corner of the 'Position Plots', have become more accurate generally, suggesting greater cerebellar integration to the eye movement mechanism. At the bottom right, the 'Phase Profile' has a more normal shape in the 'velocity verses position' plots, that is, the bumps have smoothed out somewhat. This suggests the propensity for 'Saccadic Intrusions' to have diminished, most likely from improved parietal activity. The 'Latency Plot' (top right) shows some improvement but still has a large number of spontaneous saccades at around 100ms. Collectively, these results could be interpreted as the patient's visual system 'reacting' to a visual command rather than efficiently processing that command.

Rotating chair treatment was utilised for vestibular stimulation and improvement of the Vestibulo-Ocular Reflex.



The treatment protocol began in the Axial 'yaw' plane and was followed by the Coronal 'roll' plane. 'Yaw' axial rotation, predominately stimulates the leading lateral canal with inhibition of its contralateral counterpart. Especially so if there is 30° cervical flexion to keep the lateral canals in the horizontal plane. As this vertical axis spinning movement is common to everyday life it is the least stimulatory of the three 'Yaw, Pitch and Roll' movements. Being the least stimulatory it was useful to begin with to allow the patient to get used to the equipment and allay any fears that need addressing. There were none in this case and we proceeded to add 'Rolls' by the third consultation.

Rolling, in the coronal plane, is a unique vestibular stimulation unlike any other bodily movement produced in everyday activities. Rotating coronally to the right about the 'L3-umbilicus - centre of gravity' is novel and primarily stimulates the Right Vestibular apparatus. All right hand side canals are excited and all left hand side canals inhibited in this manoeuvre. All the otolithic organs are intermittently stimulated bilaterally by rolling<sup>(21,40)</sup>. The canals only fire when there is an acceleration or deceleration as while constant velocity is maintained the endolymph is stationary but the otolithic organs (utricle and saccule) respond to the changing vertical to horizontal to vertical graviceptive movements.<sup>(21,24,27,39)</sup>

As the patient was instructed to have his eyes open while rotating in the chair there was an additional visual stimulation occurring simultaneously with the vestibular stimulation. This combination allows a recalibration of the vestibulo-ocular reflex.<sup>(29)</sup>

In relation to this case of PPPD the right side was chosen for stimulation based on the OPK Tape pursuits. They showed the biggest deficit to that side, the joint position error findings were deficient to the right and the saccadometry was worse with eye movements to the left indicating poor right sided control, although both sides were dysfunctional. Gaze evoked nystagmus was also present on the right.

Entrainment<sup>(22)</sup> is any procedure that modulates neural activities by synchronising brainwave frequency with that of a stimulus. There are various ways to entrain the nervous system and all involve some sort of rhythmic activity. The computer based program used measures efficiency of repeated movements synchronised by an audio stimuli. The sensory (audio)-motor program is developed in the USA although similar equipment is more widely available called the 'Interactive Metronome'. The neocortex and basal ganglia are heavily involved in the sensory motor integration and modulation of the networks affected by repetition of movement as a response to a stimuli. Optimal interaction with our environment requires processing and relaying sensory information synchronously and precisely. This is particularly important for complex movement, thinking, emotional control, learning and behaviour.<sup>(24,28,31)</sup> Put simply, the above

suggests that repetition will likely improve accuracy. This sentiment is contained in the the statement 'practice makes perfect', and is frequently heard in developing new tasks in sport.

Vestibulo-ocular reflex adaption training, using a head mounted laser, was achieved by multiple repetitions of briefly fixating on an illuminated (0.6s) target while waiting for a new target to appear 200-400ms later and then rapidly re-fixating on that new target. This head and eye movement requires a velocity of  $>150^{\circ}/\text{sec}$ . It was performed in almost total darkness to facilitate target acquisition<sup>(42)</sup>.

The Sensory(Visual)-Motor Integration Reaction Timer (SMIRT) equipment is custom built and can be programmed to predominately stimulate the neocortex with an 'overlap stimulus' or the brain stem using a 'gap stimulus'. This equipment consists of a panel of buttons which can be illuminated in sequence. The patient is instructed to tap an illuminated button as quickly as possible thereby turning it off. This 'reaction time' is recorded and repeated.

The neurology, put very simply, is, the Retina 'see's' the light go on, the primary visual pathways project this to the Frontal Eye Field where the command to move is initiated and sent to the Motor Cortex. This process takes about 100ms to process at the Frontal Lobe and another 100ms to develop the command to act and then a variable time to physically move the hand to the target<sup>(43)</sup>. Typically the whole action takes 300-400ms in a healthy control subject. Our 3PD patient started with a reaction time of 590ms and post treatment was 473ms. The hypothesis was that, with repetition, (360 trials per session) his sensory-motor integration would improve, ie. his visual system would get better at sending commands. There was a modest improvement.

## Discussion

Although the patient was happy with the outcome, which was an improved sense of balance, better concentration, improved reading ability, clearer thinking and diminished phono-phobia, the weakness in this case report is the inability to delineate the therapeutic value of each treatment modality.

It is possible that the purported novel intervention of using the 'orbital motion simulator' (rotating chair) to stimulate the vestibular system contributed to the positive result. This equipment is increasingly being used in research of vestibular function and therapy. Other published results using this equipment in cases of PPPD has recently appeared in the literature to support this claim but as yet the volume of research on this modality is embryonic <sup>(12,13,14,15,16,17,18,19,20,37,38)</sup>.

The value of this report lies in the demonstration, using simple activities, of a relatively rapid, clearly positive outcome to a debilitating chronic disease process which had been ineffectively managed by several types of practitioner over the period of 30 years. The initial detailed examination of key contributing systems was the key to the selection of a multimodal treatment protocol to address component parts of the presenting dysfunction.

It is not possible to determine which treatment methods used here or combination thereof allowed the favourable outcome to occur or if the above treatment applies to other cases of PPPD.

### **Patient Perspective**

“Regarding the treatment, whilst far from enjoyable, I found it to be very beneficial. The various tests showed me what I already knew, that my balance was off the charts. The rotation on the chair was annoying but definitely helped. Overall, the treatment was unusual but gave a better result than anything I have had before.”

## References

1. Popkirov S, Staab JP, Stone J. Persistent postural-perceptual dizziness (PPPD): a common, characteristic and treatable cause of chronic dizziness. *Pract Neurol.* 2018;18(1):5–13. <https://doi.org/10.1136/practneurol-2017-001809>.
2. Mucci V., Meier C., Bizzini M., Romano F., Agostino D., Ventura A., et al. Combined Optokinetic Treatment and Vestibular Rehabilitation to Reduce Visually Induced Dizziness in a Professional Ice Hockey Player After Concussion: A Clinical Case. *Front. Neurol.* 2019;10:1200. doi: 10.3389/fneur.2019.01200.
3. Balaban C. D., Black R. D., Silberstein S. D. (2019). Vestibular neuroscience for the headache specialist. *Headache* 59, 1109–1127. 10.1111/head.13550 .
4. Dai M., Cohen B., Smouha E., Cho C. Readaptation of the vestibulo-ocular reflex relieves the mal de débarquement syndrome. *Front. Neurol.* 2014;5:124. doi: 10.3389/fneur.2014.00124.
5. Von Brevern M., Bertholon P., Brandt T., Fife T., Imai T., Nuti D., Newman-Toker D. Benign paroxysmal positional vertigo: Diagnostic criteria Consensus document of the Committee for the Classification of Vestibular Disorders of the Bárány Society. *Acta Otorrinolaringol. Esp.* 2017;68:349–360. doi: 10.1016/j.otorri.2017.02.007.
6. Power L, Murray K, Szmulewicz DJ. Characteristics of assessment and treatment in benign paroxysmal positional vertigo (BPPV) *Journal of Vestibular Research.* 2020;30(1):55–62. doi: 10.3233/VES-190687.
7. Li K., Si L., Cui B., Ling X., Shen B., Yang X. (2020a). Altered intra- and inter-network functional connectivity in patients with persistent postural-perceptual dizziness. *Neuroimage Clin* 26:102216. 10.1016/j.nicl.2020.102216.
8. Staab JP, Eckhardt-Henn A, Horii A, Jacob R, Strupp M, Brandt T, Bronstein A. Diagnostic criteria for persistent perceptual-postural dizziness (PPPD): consensus document of the committee for the classification of vestibular disorders of the Barany Society. *J Vestib Res.* (2017) 27:191–208. 10.3233/VES-170622.
9. Rajagopalan A., Jinu K. V., Sailesh K. S., Mishra S., Reddy U. K., Mukkadan J. K. (2017). Understanding the links between vestibular and limbic systems regulating emotions. *J. Nat. Sci. Biol. Med.* 8 11–15. 10.4103/0976-9668.198350.
10. Balaban C. D., Jacob R. G., Furman J. M. (2011). Neurologic Bases for Comorbidity of Balance Disorders, Anxiety Disorders and Migraine: Neurotherapeutic Implications. *Expert Rev. Neurother.* 11 (3), 379–394. doi: 10.1586/ern.11.19.
11. Edelman, S. Persistent Postural-Perceptual Dizziness (PPPD). *Managing Persistent Postural Perceptual Dizziness.* Date Accessed: 23/11/2021. <https://www.google.com/search?q=managing+persistent+postural+perceptual+dizziness+by+Sarah+Edelman%2C+PhD&oq=managing+persistent+postural+perceptual+dizziness+by+Sarah+Edelman%2C+PhD&aqs=chrome..69i57.26540j0j15&sourceid=chrome&ie=UTF>
12. Carrick FR, Pagnacco G, Oggero E, Sullivan S, Barton D, Esposito S, et al. The Effects of Whole Body Rotations in the Pitch & Yaw Planes on Postural Stability. *Func Neural Rehabil Ergon* 2011;1(2):167-179.
13. Yakushin, S. B., T. Raphan, and B. Cohen. Coding of velocity storage in the vestibular nuclei. *Front. Neurol.* 8:386, 2017.

14. Barresi M, Grasso C, Li Volsi G, Manzoni D. Effects of body to head rotation on the labyrinthine responses of rat vestibular neurons. *Neuroscience* (2013) 244:134–46. doi: 10.1016/j.neuroscience.2013.04.010.
15. Carrick FR, Pagnacco G, Oggero E, Esposito SE, Duffy JL, Barton D, et, al. The Effect of Off Vertical Axis and Multiplayer Vestibular Rotational Stimulation on Balance Stability and Limits of Stability. *Funct Neurol Rehabil Ergon* 2013; 3 (2-3): 341-360.
16. Yang AHX, Khwaounjoo P, Cakmak YO. Directional effects of whole-body spinning and visual flow in virtual reality on vagal neuromodulation. *J Vestib Res.* 2021;31(6):479-494. doi: 10.3233/VES-201574.
17. Bertolini G, Ramat S. Velocity storage in the human vertical rotational vestibulo-ocular reflex. *Exp Brain Res.* 2011 Mar;209(1):51-63. doi: 10.1007/s00221-010-2518-6. Epub 2010 Dec 19.
18. Carrick FR, Clark JF, Pagnacco G, Antonucci MM, Hankir A, Zaman R, Oggero E. Head-Eye Vestibular Motion Therapy Affects the Mental and Physical Health of Severe Chronic Postconcussion Patients. *Front Neurol.* 2017 Aug 22;8:414. doi: 10.3389/fneur.2017.00414.
19. Dai M, Raphan T, Cohen B. Adaptation of the angular vestibulo-ocular reflex to head movements in rotating frames of reference. *Exp Brain Res.* 2009 Jun;195(4):553-67. doi: 10.1007/s00221-009-1825-2. Epub 2009 May 21.
20. Sadeghi NG, Sabetazad B, Rassaian N, Sadeghi SG. Rebalancing the Vestibular System by Unidirectional Rotations in Patients With Chronic Vestibular Dysfunction. *Front Neurol.* 2019 Jan 22;9:1196. doi: 10.3389/fneur.2018.01196.
21. Ramos de Miguel A, Zarowski A, Sluydts M, Ramos Macias A, Wuyts FL. The Superiority of the Otolith System. *Audiol Neurootol.* 2020;25(1-2):35-41. doi: 10.1159/000504595. Epub 2020 Jan 10.
22. Thaut MH, McIntosh GC, Hoemberg V. Neurobiological foundations of neurologic music therapy: rhythmic entrainment and the motor system. *Front Psychol.* 2015 Feb 18;5:1185. doi: 10.3389/fpsyg.2014.01185.
23. Mucci V, Indovina I, Browne CJ, Blanchini F, Giordano G, Marinelli L, Burlando B. Mal de Debarquement Syndrome: A Matter of Loops? *Front Neurol.* 2020 Nov 10;11:576860. doi: 10.3389/fneur.2020.576860.
24. Isoda M, Hikosaka O. Cortico-basal ganglia mechanisms for overcoming innate, habitual and motivational behaviors. *Eur J Neurosci.* 2011 Jun;33(11):2058-69. doi: 10.1111/j.1460-9568.2011.07698.x.
25. Todd CJ, Schubert MC, Figtree WVC, Migliaccio AA. Incremental Vestibulo-ocular Reflex Adaptation Training Dynamically Tailored for Each Individual. *J Neurol Phys Ther.* 2019 Apr;43 Suppl 2:S2-S7. doi: 10.1097/NPT.0000000000000269
26. Raphan T. Vestibular, locomotor, and vestibulo-autonomic research: 50 years of collaboration with Bernard Cohen. *J Neurophysiol.* 2020 Jan 1;123(1):329-345. doi: 10.1152/jn.00485.2019. Epub 2019 Nov 20.
27. Wada Y, Kodaka Y, Kawano K. Vertical eye position responses to steady-state sinusoidal fore-aft head translation in monkeys. *Exp Brain Res.* 2008 Feb;185(1):75-86. doi: 10.1007/s00221-007-1137-3. Epub 2007 Oct 2.



28. Hitier M, Besnard S, Smith PF. Vestibular pathways involved in cognition. *Front Integr Neurosci.* 2014 Jul 23;8:59. doi: 10.3389/fnint.2014.00059.
29. Garrick-Bethell I, Jarchow T, Hecht H, Young LR. Vestibular adaptation to centrifugation does not transfer across planes of head rotation. *J Vestib Res.* 2008;18(1):25-37.
30. Schröder L, von Werder D, Ramaioli C, Wachtler T, Henningsen P, Glasauer S, Lehnen N. Unstable Gaze in Functional Dizziness: A Contribution to Understanding the Pathophysiology of Functional Disorders. *Front Neurosci.* 2021 Jul 20;15:685590. doi: 10.3389/fnins.2021.685590.
31. Smith PF, Zheng Y, Horii A, Darlington CL. Does vestibular damage cause cognitive dysfunction in humans? *J Vestib Res.* 2005;15(1):1-9.
32. Zuma E, Maia FC, Cal R, D'Albora R, Carmona S, Schubert MC. Head-shaking tilt suppression: a clinical test to discern central from peripheral causes of vertigo. *J Neurol.* 2017 Jun;264(6):1264-1270. doi: 10.1007/s00415-017-8524-x. Epub 2017 May 23.
33. Treleaven J. Dizziness, Unsteadiness, Visual Disturbances, and Sensorimotor Control in Traumatic Neck Pain. *J Orthop Sports Phys Ther.* 2017 Jul;47(7):492-502. doi: 10.2519/jospt.2017.7052. Epub 2017 Jun 16.
34. Gurvich C, Maller JJ, Lithgow B, Haghgooie S, Kulkarni J. Vestibular insights into cognition and psychiatry. *Brain Res.* 2013 Nov 6;1537:244-59. doi: 10.1016/j.brainres.2013.08.058. Epub 2013 Sep 6.
35. Raghu V, Salvi R, Sadeghi SG. Efferent Inputs Are Required for Normal Function of Vestibular Nerve Afferents. *J Neurosci.* 2019 Aug 28;39(35):6922-6935. doi: 10.1523/JNEUROSCI.0237-19.2019. Epub 2019 Jul 8.
36. Zamysłowska-Szmytke E, Adamczewski T, Ziąber J, Majak J, Kujawa J, Śliwińska-Kowalska M. Cervico-ocular reflex upregulation in dizzy patients with asymmetric neck pathology. *Int J Occup Med Environ Health.* 2019 Oct 16;32(5):723-733. doi: 10.13075/ijom.1896.01428. Epub 2019 Oct 7.
37. Bertolini G, Ramat S, Bockisch CJ, Marti S, Straumann D, Palla A. Is vestibular self-motion perception controlled by the velocity storage? Insights from patients with chronic degeneration of the vestibulo-cerebellum. *PLoS One.* 2012;7(6):e36763. doi: 10.1371/journal.pone.0036763. Epub 2012 Jun 15.
38. Ray CA. Interaction between vestibulosympathetic and skeletal muscle reflexes on sympathetic activity in humans. *J Appl Physiol (1985).* 2001 Jan;90(1):242-7. doi: 10.1152/jappl.2001.90.1.242.
39. Laurens J, Angelaki DE. The functional significance of velocity storage and its dependence on gravity. *Exp Brain Res.* 2011 May;210(3-4):407-22. doi: 10.1007/s00221-011-2568-4. Epub 2011 Feb 4.
40. Chetana N, Jayesh R. Subjective Visual Vertical in Various Vestibular Disorders by Using a Simple Bucket Test. *Indian J Otolaryngol Head Neck Surg.* 2015 Jun;67(2):180-4. doi: 10.1007/s12070-014-0760-0. Epub 2014 Aug 8.
41. Mutlu B, Serbetcioglu B. Discussion of the dizziness handicap inventory. *J Vestib Res.* 2013;23(6):271-7. doi: 10.3233/VES-130488.
42. Rinaudo CN, Schubert MC, Cremer PD, Figtree WVC, Todd CJ, Migliaccio AA. Once-Daily Incremental Vestibular-Ocular Reflex Adaptation Training in Patients With Chronic Peripheral

Vestibular Hypofunction: A 1-Week Randomized Controlled Study. J Neurol Phys Ther. 2021 Apr 1;45(2):87-100. doi: 10.1097/NPT.0000000000000348.

43. Leigh R, Zee DS. The neurology of eye movements. 5th Edition. Published in the United States. Oxford University Press 2015, Chapter 11, pp657-658.