

A Functional Neurological Disorder case series utilising a sensory motor integration model including Chiropractic and vestibular stimulation

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Objective: Functional Neurological Disorder (FND) is a complex condition characterised by neurological symptoms that cannot be explained by a structural or organic disease. Traditional interventions often emphasise psychological and physiotherapeutic approaches. This case series explores the efficacy of an integrative treatment combining Chiropractic care, Vestibular Rehabilitation (VR), and Sensory Motor activities in four patients diagnosed with FND.

Methods: Four patients (2 females, 2 males, age range 15-48 years) with confirmed FND were treated. Each presented with distinct neurological complaints including motor weaknesses, sensory disturbances, and movement disorders. An individualised treatment regimen was designed for each patient combining Chiropractic Adjustments, tailored VR exercises, and specific sensory motor activities. The intervention spanned 3 - 36 weeks, and outcomes were evaluated based on symptom reduction and functional improvement.

Results: All four patients exhibited significant improvement post-treatment with three patients achieving complete resolution. The movement disorders, such as seizures, tremors and dystonia, were reported as achieving considerable symptom alleviation.

Conclusion: This case series suggests that an integrative approach, melding Chiropractic care with Vestibular Rehabilitation and Sensory Motor activities, may offer a promising avenue for the management of FND. Larger controlled studies are warranted to validate these findings and to further explore the underlying mechanisms behind this synergistic therapeutic approach.

The clinical presentation and treatment course of each patient is presented, including the specific VR and Chiropractic techniques used, and report on their outcomes. We also discuss the potential mechanisms by which VR and Chiropractic treatment may be effective for individuals with FND.

Indexing Terms: Functional Neurological Disorder; Chiropractic; Sensory Motor Integration; Rotating Chair; Vestibular Rehabilitation; PNES; Homeostasis

... Chiropractic treatment can be considered as a particular type of therapy for the enhancement of sensory motor activity. When combined with vestibular rehabilitation techniques the combination can be very effective for Functional Neurological Disorders.

These undoubtedly complex clinical cases do not respond well to any one method of treatment. It appears the combination of modalities is key to success with this cohort. Spinal Adjustments are powerful re-integrators of the sensory motor system clearly demonstrated daily by chiropractic patients reporting a high level of satisfaction. Vestibular Rehabilitation is also an effective adjunct to improved sensory motor integration. In combination, Chiropractors are well positioned to help this debilitated cohort of FNDs.'



Introduction

The term 'functional' is used to describe a category of disorders or symptoms that do not have an identifiable organic or structural cause but are associated with genuine physical or psychological impairments. These conditions are often referred to as '*Functional disorders*' or '*Functional Somatic syndromes*' and include:

- ▶ Functional Neurological Disorder (FND), as described above
- ▶ Functional Gastrointestinal Disorders (FGID): This group of conditions includes disorders like Irritable Bowel Syndrome (IBS), functional dyspepsia and functional constipation
- ▶ Functional Pain Syndromes: Conditions like fibromyalgia and chronic pelvic pain are considered functional pain syndromes
- ▶ Functional Dysphonia: A voice disorder where individuals experience hoarseness or changes in voice quality that cannot be attributed to physical or structural issues with the vocal cords
- ▶ Functional Movement Disorders: Characterised by abnormal movements, tremors, or dystonia
- ▶ Functional Cognitive Disorder: Is a condition where individuals experience cognitive impairments, such as memory problems or attention issues
- ▶ Functional Heart Disorders include conditions like non-cardiac chest pain and functional arrhythmias
- ▶ Functional Dermatological Disorders include *Psychogenic Purpura* and *Dermatitis Artefacta*
- ▶ Functional Bladder Disorders such as Non-neurogenic bladder dysfunction
- ▶ Somatoform Disorders include somatic symptom disorder and illness anxiety disorder, where individuals experience distressing physical symptoms or have excessive health-related concerns.

While the exact cause of FND is not fully understood, it is believed to involve a complex interplay between physical and psychological factors. Trauma, stress and psychiatric disorders are some of the most commonly recognised predisposing factors for the development of FND, but the research findings are inconsistent. The above predisposing factors are only found in a third of the adult population with functional symptoms. (19) FND is characterised by abnormal patterns of brain functioning. (8, 13, 34) Vestibular symptoms, such as dizziness and balance problems, are a common manifestation of FND and can significantly impact an individual's quality of life.

Vestibular rehabilitation (VR) is a type of physical therapy that is used to treat individuals with vestibular disorders and can help to improve balance and reduce dizziness and other vestibular symptoms. Chiropractic treatment is another complementary therapy that is commonly used to treat a range of musculoskeletal conditions, its role in the treatment of FND is not well-established.

One previous study has suggested that Chiropractic care may be a potential treatment option for individuals with FND, (5) while several studies have shown the effectiveness of VR in reducing vestibular symptoms in individuals with FND. (6, 7). To date, there are not any published studies that have examined the use of a combination of VR and Chiropractic treatment for individuals with FND. VR is a recognised sub-specialty of Chiropractic in Australia.

This case series highlights the potential benefits of a combined approach to treatment for individuals with FND. Further research is needed to better understand the effectiveness of these treatments in this population and to identify the most effective treatment approaches for individuals with FND. (10)

Patient 1

Patient information

Female, 48 y/o, right handed, Caucasian, presenting with Non Epileptic Seizures.

The initial seizure episode occurred in September 2016 following a bout of gastroenteritis. It was violent, fracturing some ribs and lasting 36 hours. Since then P1 has been hospitalised approximately every 10 weeks with severe seizures. Suffering concussion multiple times. Less severe seizure activity occurs daily and is described as '*distressing spasms, freezing or fitting*', they occur approximately 12-20/day and occur nightly approx. 2-3 times, predominantly in the right hand and ankle.

Usually on the weekend a seizure will last 4-6 hours. Consequently P1 confined herself to the family home and found visitors too stressful and causing embarrassing dystonic incidents like '*whole body freezing*', regional or half body spasms and seizures.

Noise, especially '*electronic noise*' was aggravating while a startle would often give rise to a '*stiff body*' episode or a seizure.

P1 could not then attend a supermarket as the noise, crowds, light and movement were too stimulatory.

She usually feels a seizure '*building up*'.

She found it difficult to initiate gait. Sitting made initiating gait much worse than standing. A sliding movement of her feet was more easily initiated while a '*stepping*' gait was also necessary sometimes '*to get going*'.

P1 cannot wear tight clothing as this will initiate a seizure, otherwise she doesn't feel her clothing on her body.

She has to close her eyes when walking through a standard doorway or it will invariably trigger a fall to the left.

Of note P1 stated that:

- Her gait improved with having a weight on her head.
- Scalp massage lasting 5 minutes relieved the dystonia.
- Initially Physiotherapy seemed to help and Acupuncture seemed to improve the condition for the first two treatments but failed subsequently.
- Eating bread made her abdomen 'bloat'.
- She has had Supra-ventricular Tachycardia her whole life.

P1 has had multiple diagnostic workups including EEG, MR scans and MR spectroscopy. Medications including *Rivotril (Clonazepam)*, *Seroquel*, and *Valdoxan* have had little effect.

Clinical findings

Sliding gait with right foot and leg dragging.

Scanning speech.

Markedly tight left TMJ compared to her right

Very poor enunciation of '*British Constitution*'

While performing the '*Reflex Pupillometry App*' testing the left side of P1's face tightened dramatically into a dystonia lasting 5mins.

Maddox Rod Testing showed 3cm convergence error at 300mm.

Cervical Position Error NAD

Saccadometry - Latencies were slow and spread between 220-450 ms.

Horizontal Saccades were all hyper-metric, averaging 11.2° for the 10° target.

Force Plate Analysis - demonstrated Visual and Proprioceptive dysfunction with Vestibular compensation.

Pupillometry - Using the 'Reflex' App, initially the Left Eye metrics of Average and Maximum Constriction speed with the Average, Minimum and Maximum Diameters were all outside the normal range.

Right eye metrics were initially outside the normal range for the Average, Minimum and Maximum Diameters.

Video-oculography Lateral roll - Left Nystagmus, ageotropic .

Dix Hallpike - square wave jerks without dizziness or vertigo.

Sensory-Motor Integration Reaction Timer (SMIRT) showed a slow (1.2sec N=0.4sec) ability to locate targets 1.2m apart.

One Leg Standing - Poor bilaterally, Right 4sec, Left 3sec.

Dysdiadokokinesia - Hand Slap - poor.

OPK - Slow and dysmetric with multiple Saccadic Inclusions.

Raglans Test - Diastolic maximum variation of 17mm Hg, HR maximum variation 23 BPM

Diagnostic assessment

Functional Neurological Disorder

Functional Dysphonia

Dysautonomia

Visually Induced Dizziness

Anxiety

Therapeutic intervention

Occurred over a six week period and included:

- Peripheral Nerve Stimulation of XII, X, IX, VII ie Tongue (1 Hz Square wave)
- Backwards walking on a treadmill while dual tasking by balancing a ball on a small bat in front of her.
- Entrainment Therapy - described after the patient information.
- Interactive Saccade Training - SMIRT - Gap and Overlap Trials and Anti-saccades.
- Spinal Adjustments - were of the Diversified style (High Velocity and Low Amplitude) applied to whichever subluxations were palpable at the time of presentation.
- Vestibular stimulation was performed by using a Multi-axis Rotating Chair in the Pitch, Roll and Yaw planes.

Home Exercises :

- leg massage 2/day
- Visualisation of walking gracefully
- Vibration to Left Ankle
- Hot and Cold foot baths
- Focus Builder App - Antisaccades 1min tds
- Toe Raiser Exercise X30 3/day

Treatment timeline

Table 1	Days between Tx's	Comment	Seizure Activity	
Tx 1	na			
Tx 2	7	Seizure upon arrival home from 1st Tx lasted 3 hours	Less seizures occurring during the day	Very bad seizure 2 days previously - sore since
Tx 3	3	Lots of spasms post last Tx	No day seizures since last Tx	
Tx 4	4	Spasms occurring multiple times daily	No day seizures since last Tx	
Tx 5	3	Supraventricular Tachycardia attack 1 hour post last Tx	No day seizures since last Tx	Fatigued from exercises
Tx 6	4	Very fatigued post last Tx	No day seizures since last Tx	
Tx 7	3	Less Pain	No day seizures since last Tx	Improved car traveling
Tx 8	4	Pain is worse at night	No day seizures since last Tx	
Tx 9	3	Felt very cold post last Tx	Big seizure 2 days ago	
Tx 10	4	Improved	night seizure last night lasted seconds only	
Tx 11	3	Extreme fatigue	1 seizure since last Tx lasting 15mins	
Tx 12	4	Profound fatigue daily and 'brain fog'	1 mild seizure since last Tx	
Tx 13	3	Final assessment	see below	Night Seizures persist but diminished.

Final assessment

- Right hand and ankle myoclonus 3/day mainly at night, (was 12/day minimum).
- Seizures were daily, usually at night, now 1/week, 80% less intense, lasting 30mins compared to an average of previously >1Hr.
- Gait initiation used to take 1-2 hrs to pass now only a few minutes.

- Speech - much improved, now jaw tightening is a reliable sign of impending seizure activity.
- Walking through doorways was a constant problem occurring every time but now occurs around 1/wk.
- Akinesia (freezing) occurs rarely.
- Pain - fluctuates but overall unchanged.
- Ability to attend a shopping centre is still intolerable but P1 can now tolerate visitors to home mostly without mishap.
- Noise is still a problem, especially electronic noise.
- Saccadometry - Latencies were improved and spread between 180-350ms.
- Horizontal Saccades - were all on target, averaging 9.8° deg for the 10° target.
- Force Plate Analysis - Markedly improved overall balance mainly from improved Proprioceptive function, however the Vestibular compensation persisted.
- Pupillometry - Left eye metrics unchanged. Right eye metrics were all within normal limits.
- Sensory-Motor Integration Reaction Timer (SMIRT) showed improved reaction times of around 0.47sec (N=0.4sec) for the ability to locate targets 1.2m apart.

Follow-up and outcomes.

A telephone conversation in 2 years post treatment revealed P1 still has seizure activity at night, fatigue and dizziness. There has not been a great deal of change in her signs and symptoms since stopping treatment by leaving the area.

Patient 2

Patient information

Female 15 y/o, right handed presented complaining of daily seizures lasting 2-6 hours for 3 years.

P2 has been diagnosed with Asthma, Autism, Attention Deficit Hyperactivity Disorder, Sensory processing Disorder, *Ehlers Danlos Syndrome*, Epilepsy, Functional Neurological Disorder and Complex Regional Pain syndrome between 2012 and 2018.

The seizures began in 2017 and usually present as an Oculogyric crisis being immobility, unconsciousness, whole body fine tremors with the eyes open and looking up and to the left. This was, on one occasion, observed by the author.

Her daily medications include *Catapres*, *Luvox*, *Lyrica*, *Ritalin*, *Nasonex*, *Seratide* and *Ventolin*.

Clinical findings

A comprehensive Neurological examination revealed:

Near point Convergence - 200mm

Repeated Convergence Testing showed insufficiency and dysmetria while the left eye lagged

Corneal Reflex was diminished bilaterally

Glabella reflex was positive

Webbers Bone Conduction was reported as Left sided

The Gag reflex was absent bilaterally

One Leg Standing with eyes closed was poor bilaterally (Left 4s, Right 3s)

Dynamic Visual Acuity Test using the Snellen Chart was 3 Lines deficient

Cervical Joint Position Error testing was poor

Raglans Test was mildly positive with a diastolic variance of 12mm Hg (N<10) and the Heart Rate 21bpm (N<20)

Pupillometry - Left Eye Latency was slow with the minimum diameter also below normal range.

Saccadometry - The majority of P2's saccades were initiated below 150ms indicating poor neocortical control of eye movements.

Force Plate Analysis - strong visual dependence with Proprioceptive and Vestibular deficits were affecting her ability to 'balance'

Diagnostic assessment

Functional Neurological Disorder

Anxiety

Therapeutic intervention

Treatment occurred in 4-5 days per session with multiple treatments of 1-3/day with home exercises between sessions, it included:

- Spinal Adjustments
- Sensory-Motor Integration activities
- Vestibular Stimulation and
- Home exercises(10).

Spinal Adjustments were of the Diversified type (High Velocity and Low Amplitude) applied to whichever subluxations were palpable at the time of presentation.

VOR Adaption training

- Using a Head Laser and standing in front of a 1.2m square panel with alternating lighted targets. The patient is instructed to 'hit' the target with the laser when it illuminates. The timing of such is altered until eventually a velocity of >150°/sec is achievable, thereby readapting the VOR back to the normal range. It was performed in almost total darkness to facilitate target acquisition.
- Entrainment Therapy. See description following patient information.
- Vestibular Stimulation was achieved using a multi-axis rotating chair. See description following patient information.
- Peripheral Nerve stimulation of XII, X, IX, VII i.e. Tongue.
- Backwards walking on a treadmill while dual tasking by Juggling 3 balls.
- Collicular Remapping and Looming Techniques.
- Complex Movements Fig '8's.
- Saccade Training using a SMIRT - Gap and Overlap Trials and Anti-saccades.

Home Exercises included:

- Oculomotor Exercises - X2 VOR, Eye Rolling
- Breath Pause
- Running 20mins/day

Follow-up and outcomes

45 Treatments delivered in 9 sessions over a period of 9 months completely resolved the seizure activity.

Treatment timeline

Table 2	No. of Tx's	Time between sessions	Seizure Activity
Pre Tx			Daily lasting 2-6 Hours
Session 1	3	38 days	2 days seizure free during the last 38 days
2	6	60 days	3 days seizure free, seizures lasting 5 hours
3	7	17 days	6 seizures (1 every 3d) lasting 1-1.5 Hours
4	11	19 days	frequency same, less intense, lasting <1hr
5	5	45 days	freq same except 6 concurrent days no seizures
6	6	33 days	4-5 days b/w seizures, lasting <45mins
7	7	95 days	Seizures only at night, 1-2 per wk
8	7	25 days	No seizure activity, possibly some at night
9	7	25 days	No seizure activity since last Tx

A recent phone call 2 yrs Post treatment confirmed that no further seizure activity has occurred following the aforementioned treatments and that P2 discontinued all medication over 2 years ago.

Patient 3

Patient information

In July 2021 'P3', a 46 y/o right handed Builder, Caucasian, male presented with a '*vigorous*' coarse bilateral distal upper limb tremor.

Beginning in January 2021 following recovery from a loss of consciousness and hospitalisation, P3's '*seizure like*' arm movements had become a daily occurrence. He could control the movement with gross actions but with rest or lack of guided activity the vigorous movements persisted until falling asleep. He concurrently suffered from business and relationship stress, with headaches, depression and sensory aberrations as '*ants crawling under his skin*' with tingling and numbness of the hands and feet. He was unable to work.

Medication: *Mirtazapine, Duloxetine, Propranolol.*

Investigations prior to attendance included, Brain MRI (x2), MRI Spinal Cord, EEG, CT Chest, Abdomen and Pelvis. All unremarkable.

Clinical findings

Saccadometry - Performed initially and after 4.5 months of treatment.

The initial Latency Plots show a diffuse spread of saccades from 125 - 450ms including many Saccadic Intrusions and Hypometria bilaterally. The average saccade missed the 10° target by 1.1°.

In comparison, the plots taken after treatment show a Scandic spread of only 150 - 350ms with very few Saccadic Inclusions. The average saccade missed the target by 0.4°.

Force Plate Analysis: Revealed a Visual processing dysfunction, in that P3's balance paradoxically improved with his eyes closed. Proprioceptive integration failure was evident in that his balance improved while upon a perturbed surface. Vestibular compensation was occurring as when the vestibular organ was relatively isolated P3's balance was at it's best.

Spinal assessment: Various subluxations at all levels.

HR 135bpm RR 34/min HRV 44 02 98%

Dynamic Visual Acuity Testing: Loss of 4 lines of the Snellen Chart

Force Plate Analysis: Vestibular compensation for both a visual and a proprioceptive functional loss of balance ability. Balance Age was 68.5 Years.

Raglans Test - Systole variation 27mm Hg (N<20)

Diastole = N

HR = N

Diagnosis

Functional Movement Disorder

Anxiety/Depression

Therapeutic intervention

- Treatment included Spinal Adjustments, Sensory-Motor Integration activities, Vestibular Stimulation and Home Exercises and recommendations.
- Interactive Saccade training
- VOR Adaption Training - As previously mentioned.
- Entrainment Therapy - Using a computer program. See Discussion.
- Spinal Adjustments - were of the Diversified type (High Velocity and Low Amplitude) applied to whichever subluxations were palpable at the time of presentation.
- Vestibular Stimulation - was achieved using a Multi-Axis Rotating Chair. See Discussion.
- Home Exercises - (may seem unusual)(10)
- Gaze stability X2 VOR Ex
- Aerobic activity as quickly walking a 15min mile (1.6 Kms)
- Breathing Exercises - Sitting still while watching the second hand of a clock, the patient has to slowly inhale over a 5 second period and exhale over 5 seconds. Repeating for 1 minute, 3/day.

Treatment timeline

Table 3		Other Tx modalities	FMD activity
Tx 1	Spinal adjustments	none	see above
Tx 2 (4 days later)	Spinal adjustments	Home Execises Peripheral Nerve Stimulation	unchanged
Tx 3 (1 day later)	Spinal adjustments Vestibular Stimulation Entrainment Tx	Home Execises Peripheral Nerve Stimulation Saccade Training	slight improvement

Table 3 conc		Other Tx modalities	FMD activity
Tx 4 (1 day later)	Spinal adjustments Vestibular Stimulation Entrainment Tx VOR Adaption Tx	Home Exercises Peripheral Nerve Stimulation Saccade Training	Fine Tremor only
Tx 5 (1 day later)	Spinal adjustments Vestibular Stimulation Entrainment Tx VOR Adaption Tx	Home Exercises Peripheral Nerve Stimulation Saccade Training	No Tremor
Tx 6 (4 days later)	as above	as above	No tremor Occasional Arm twitching at night.
Tx 7 (1 day later)	as above	as above	No tremor Occasional Arm twitching at night.

Follow-up and outcomes

The Functional Movement Disorder of the upper limb subsided to become a 'fine' tremor within 3 treatments over 6 days. This also subsided over the next two treatments leaving only a 'twitching' of his right arm noticeable at night and gradually subsiding.

One month later the 'twitching' would reoccur when encountering a stressful event. The depression and sensory symptoms abated but resurfaced from time to time over the next 18 months. No further progress was achieved. P3 stopped taking the medication shortly after treatment began. Around that time he also sought help from a counsellor but did not persevere with more than a few visits.

P3's 'Balance Age' was initially 68.5 years and 16 months later was 33.1 years. He is working full time.

Patient 4

Patient information

This 17 y/o caucasian right handed unemployed male presented with 'seizures' occurring daily 5 times per week for the previous 18 mo. The episodes lasted minutes to an hour and had prevented him from working.

The seizure begins as a pressure in the stomach followed by lightheadedness progressing to Nausea and 'body shakes' either as a fine tremor or violent shaking and syncope lasting seconds to a few minutes. Upon arousal he may have visual hallucinations leading to a feeling of pressure in his head generally which subsides in an hour. The seizures have been progressively getting worse from the beginning.

P4's father shared a video of an 'episode' with the author and claimed P4 had been extensively investigated at the local hospital and with a Neurologist and the resulting diagnosis was 'Conversion Disorder'. No medication or other treatment was advised while being given the suggestion that it is 'psychological'.

P4 had recently been attending a Naturopath who prescribed Magnesium Powder and B complex vitamins. These appeared to make the seizures less severe but did not alter the frequency.

Clinical findings & diagnostic assessment

Saccadometry - Initially this testing revealed a very slow Latency of saccades averaging 304ms for Left looking and 334ms for Right looking saccades.

Post treatment his Latencies were 189ms for Left looking saccades and 229ms for the right looking saccades. Which are normal values for his age group.

Force Plate Analysis - Comparison of the Vestibular, Proprioceptive and Visual parameters suggested a diminished Proprioceptive awareness.

Raglans - HR variance 30bpm (N<20), Systole within normal limits, Diastole variation 30mmHg (N<10)

Carotid Body reflex - HR slow to respond, O2 initially 90% rapidly rising to 99%.

Oculo-cardiac reflex - HR 63 and unresponsive.

O2 - supine 94%, standing 95%

HR - supine to Standing variance of 37bpm (N=0)

OPK - Right looking saccades were hypermetric.

Vascular Perfusion of the LE 5secs bilaterally.

Dysdiadokokinesia was poor.

Rhomburgs Tandem was poor.

DVAT line 3 lines deficient on a Snellen Chart

Diagnosis

Functional Neurological Disorder

Dysautonomia

Therapeutic intervention

Treatment included:

- Spinal Adjustments
- Sensory-Motor Integration activities
- Vestibular Stimulation and
- Home Exercises.
- Spinal Adjustments - were of the Diversified type (High Velocity and Low Amplitude) applied to whichever subluxations were palpable at the time of presentation.
- Anti-saccades - on the saccade trainer.
- VOR Adaption Training - Using a Head Laser and standing in front of a 1.2m square panel with alternating lighted targets. The patient is instructed to 'hit' the target with the laser when it illuminates. The timing of such is altered until eventually a velocity of >150°/sec is achievable, thereby readapting the VOR back to the normal range. It was performed in almost total darkness to facilitate target acquisition.
- Home Exercises - (may seem unusual). (10)
- Burpee's X30/day - for improving the orthostatic regulation of HR
- Singing Loudly - as a vagal stimulation
- Reading - because he needs to improve his ability to concentrate (Executive-Frontal Lobe development)
- Planning his day in detail - to engage his Executive functionality.

- Running Backwards - novel stimulation of the Frontal lobe and hippocampus
- Breath holding - to train the frontal lobe using executive activity.
- X 2 VOR Ex
- Focus Builder App - Anti-saccades, Go No Go Saccades

Table 4	Saccadometry	Initial Examination	Post Treatment
	Latency - Left Looking Saccades (N<200ms)	304ms	189ms
	Latency - Right Looking Saccades (N<200ms)	334ms	229ms
	Velocity - Left Looking (N>400)	335 °/sec	393°/sec
	Velocity - Right Looking (N>400)	289°/sec	353°/sec

Treatment timeline

Table 5 Timeline	Raglans Test	Spinal Adjust ment	Saccad ometry	Other Treatment	Home Exercises	Seizure frequency
Initial Exam & Tx	Systole change 0 Diastolic variation 30mmHg HR = N	yes	yes		none	5 seizures of every 7 days for 18mo
2nd Tx (4 days later)	Systole change 0 Diastolic variation 12mmHg HR = N	yes	yes		Breath Hold Burpee's X 20/day Sing Loudly Read Plan the day	One episode in last 4 days
3rd Tx (4 days later)	Systole = N Diastolic variation 12mmHg HR = N	yes	yes		Breath Hold Burpee's X 20/day Running backwards	none
4th Tx (7 days later)	Systole = N Diastolic variation 19mmHg HR = N	yes	yes		Breath Hold Burpee's X 30/day Gaze stability Ex's yes yes no no	one seizure in the last 7 days

Table 5 Timeline	Raglans Test	Spinal Adjust ment	Saccad ometry	Other Treatment	Home Exercises	Seizure frequency
5th Tx (7 days later)	Systole = N Diastolic variation 29mmHg HR = 29 bpm	yes	yes		Breath Hold Burpee's X 30/day Gaze stability Ex's yes yes no no	Two seizures in the last 7 days
6th Tx (5 days later)	Systole = 24mm Hg Diastolic variation 12mmHg HR = N	yes	yes		Breath Hold Burpee's X 30/day Gaze stability Ex's yes yes no no	One seizure in the last 5 days
7th Tx (2 weeks later)	Systole = N Diastolic variation 16mmHg HR = 23 bpm	yes	yes		Breath Hold Burpee's X 30/day Gaze stability Ex's yes yes no no X1 VOR '0' Gain VOR	1st wk no seizures 2nd wk 5/7 occurring 20 mins after 'bad' news.
8th Tx (5 days)	n/p	yes	yes	Laser Targets	Breath Hold Burpee's X 30/day Gaze stability Ex's yes yes no no	none
9th Tx (7 days)	Systole = N Diastolic variation 13mmHg HR = N	yes	yes	Anti- saccades	Breath Hold Burpee's X 30/day Gaze stability Ex's yes yes no no X 2 VOR '0' Gain VOR	none
10th Tx (21 Days)	n/p	yes	n/p			none

Follow-up and outcomes

No further Tx was delivered as P4 moved from the area. A telephone conversation with him 4 Years post treatment revealed he had been free of seizure activity since the treatment described but that recently following a stressful event he felt like the activity was *'building up'*.

Saccadometry

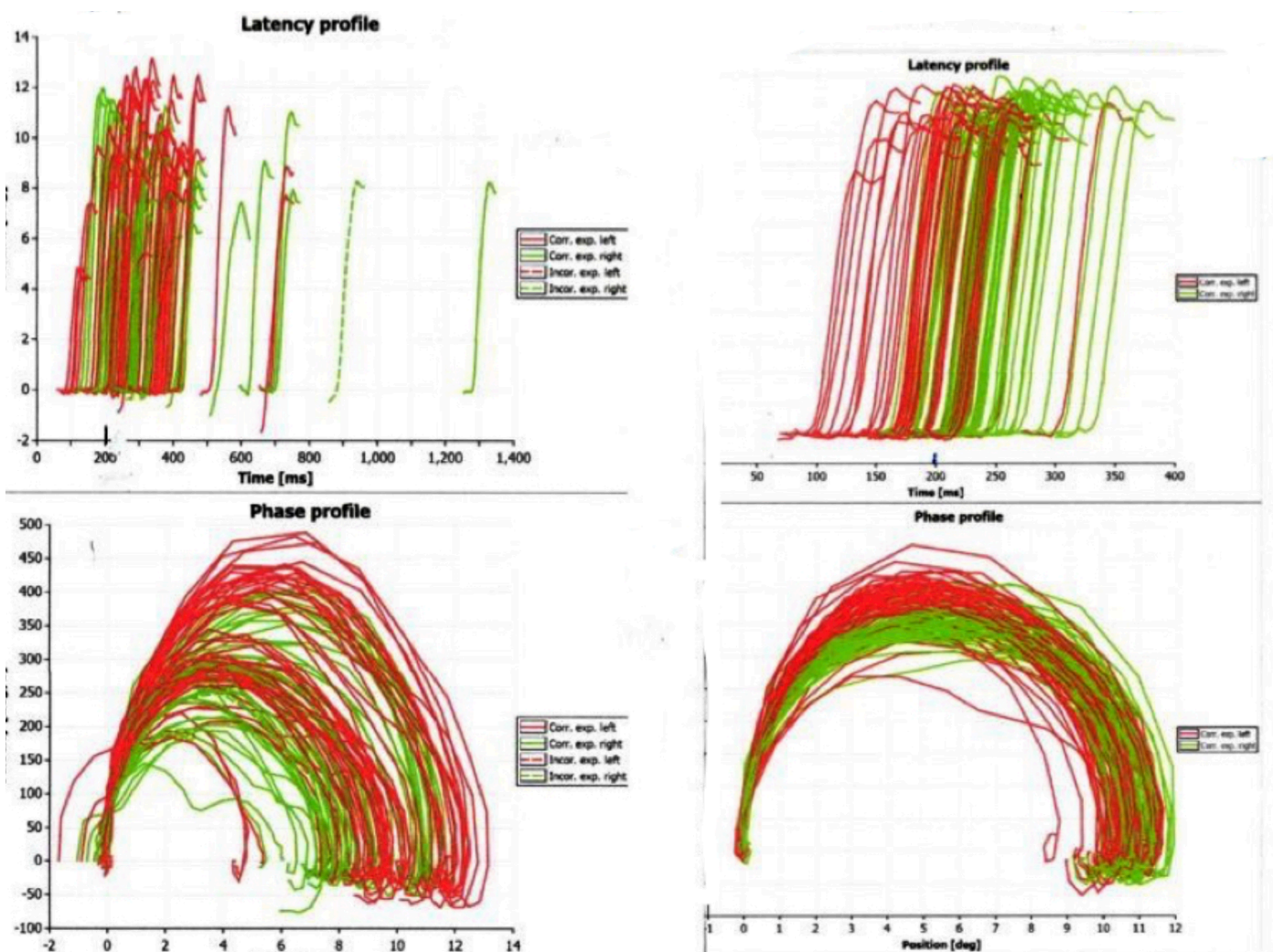
Eye motion when observing the visual environment consists of a sequence of fast eye movements called saccades. These occur around three times per second, followed by fixation periods of relative stability while observing a target object etc. (55)

Saccadometry is the study and measurement of saccades, being rapid, ballistic movements of the eyes that are used to rapidly shift the focus of gaze from one object to another. Saccadometry involves the use of specialised equipment to accurately measure various parameters of saccades, such as their velocity, amplitude, duration, latency, and accuracy. Understanding saccades is crucial in the study of the neurological control of eye movements, brain function, and cognition. Saccades are controlled by a complex neural network. (22, 54, 58)

Saccadometry can be used in the diagnosis and management of various neurological disorders, such as Concussion, Parkinson's disease, Multiple Sclerosis, and Huntington's disease, Progressive supranuclear palsy, and Spinocerebellar ataxia. It is also useful in the diagnosis of certain types of eye movement disorders, such as nystagmus or saccadic dysmetria (23, 24, 55, 57) For this case series Saccadometry was used as a guide to show progress with treatment by comparing baseline measurements to normal values (see table 4).(34 - 52, 61)

Saccadometry interpretation is beyond the scope of this paper but readers will obviously see a difference in the cohesion of the pre and post graphs. Obviously, the more coherent the better.

Pre (Left) and Post (Right) Treatment Saccadometry traces



Pupillometry

Pupillometry is the measurement of the pupil diameter and reactivity (changes in pupil size). It is conducted using a *Pupillometer*, a device that measures the size of the pupil and its reactions to various stimuli. Pupillometry provides insights into a variety of physiological, psychological, and neurological states and is considered an accurate indicator of Autonomic Nervous System function. (24 - 32)

Two types of Pupillometry Phone Applications were used to collect data with this case series:

- ▶ 'Reflex', which measures each eye singularly and
- ▶ 'Ocula' which measures both eyes simultaneously.

Both Applications produce response graphs and metrics.

This type of testing is quick, easy and accurate. Which makes it lend itself to repeatedly monitoring progress, especially of the symptoms pertaining to the Autonomic Nervous System.



Force Plate Analysis

Force plate analysis is used to assess balance and postural control by measuring the shifting centre of pressure when standing still on a balance plate. This is performed with the patient's eyes open and closed and repeated on a soft surface again with eyes open and closed. This allows for a dissection of the specific balance components and their interactions

The Vestibular, Visual and Proprioceptive sensory input is normally integrated and adjusts the outgoing motor response to maintain balance, posture and gaze stabilisation. Each sensory system contributes disproportionately depending on the stability of the surface. On a firm surface with eyes open, the Proprioceptive system contributes 70% to the overall sensory input needed for balance. The visual system 10% and the Vestibular System 20%. However, standing on a soft surface, when the feet are not in contact with the solid surface ensuring the body weight is suspended, the Proprioceptive System contributes around 10% of input and the Vestibular system then contributes 70% and the Visual system 20%.

Vestibular dysfunction arising from peripheral or central components of the vestibular system may manifest as illusory self motion (dizziness/vertigo) and spatial disorientation, which in turn impairs balance.(53) Clearly, the Eyes Closed, Soft Surface testing almost isolates the Vestibular system except for a small amount of Proprioceptive input.

The Proprioceptive and Visual concomitants can be deduced using the same logic. This produces a mixture of results from poor balance due to a functional loss in one, two or three systems. Or 'normal' balance but due to one or two systems compensating for a third system loss. Each unique set of system loss' or compensations suggests unique rehabilitation strategies.

Interactive Saccade Training

Using the custom built SMIRT (Sensory Motor Integration Reaction Timer) the patient stands in front of a panel of temporarily illuminated buttons/targets. The patient repeatedly and randomly sees a lighted target in their peripheral vision and reacts to this by quickly reaching to the target and pushing the target button. This combination of activities make the CNS perform at a high level requiring Peripheral and Central Vision, Gross and Fine Motor movements and demands exquisite coordination. (59, 60, 61)

The selective timing of the lighted targets enable different combinations to predominantly enhance performance of the cortex or brain stem eye movement command generators.

The accuracy and average speed are recorded to gauge improvement from treatment. The equipment is custom built and is programmed to match and challenge the users ability. Theoretically, it can be adjusted to predominantly stimulate the Neocortex with an '*Overlap Stimulus*' or the Brain Stem using a '*Gap Stimulus*' which was utilised with this case series.

This equipment is also used in it's '*Antisaccade*' mode. Where the user has to respond to an illuminated target by activating the button diametrically opposite the illuminated target. This protocol requires 'a double take' of the frontal cortex to achieve it correctly and quickly. That being, the reflexive saccade to the lighted target must be inhibited, the corresponding position calculated and subsequently acted upon.

It integrates the asymmetric tonic neck reflex, helps correct a maladapted vestibulo-ocular reflex and or a deficient cervical-ocular reflex, consequently improves hand-eye coordination, basal ganglionic pathways and frontal lobe efficiency. Potentially, leading to improved cognition and self control over unwanted habituation, i.e. behaviours.

Saccade training - VOR Adaption training

Vestibulo-ocular reflex (VOR) adaptation training is a type of rehabilitation exercise designed to improve the function of the reflex. The VOR is a critical neurological mechanism that stabilises

vision during head movements by producing eye movements in the opposite direction of head movement, thus maintaining the image on the centre of the visual field. When the VOR is impaired due to injury, disease, or aging, it can lead to symptoms like dizziness, vertigo, and balance problems.

The VOR is very important for keeping the brain supplied with visual information to build a clear picture of the environment. (56) When the information is hazy, the brain makes less accurate judgements for movement through the environment. Which could mean banging an elbow when going through a familiar doorway, or spilling a drink, tripping or being clumsy.

The VOR uses information from the inner ear (vestibular labyrinth) to generate eye movements that allow the eyes to fixate upon an object during head movements.

With severe dysfunction, it is impossible to read signs or even recognise faces while just walking. A dysfunctional VOR can also cause blurred vision. There are Vertical, Horizontal, Angular and Translational VOR Reflexes. During rotation of the head, the VOR can stabilise the eyes accurately even with rapid movements, mainly because the VOR pathway is relatively short and very fast. Precisely because it is so quick, (6ms) the VOR must be intrinsically accurate; on this timescale, vision is too slow to be useful to use for feedback. The brain has to be fast and automatic. Consequently, many diseases, injuries and traumas will adversely affect the functioning of the VOR. It is likely to be a contributing problem with many chronic conditions.

When the VOR becomes dysfunctional the brain neuroplastically adapts to the dysfunction and it becomes a '*maladapted VOR*'. This can persist for our entire lives being a detrimental influence to attaining rich and vibrant maps of our external world.

VOR Adaption Training is achieved by relearning how to accurately and quickly find visual targets. For this case series we used a lighted panel with computer generated targets. The patient wore a Laser headpiece to rapidly follow the randomly generated ever changing targets. Initially the times are set to whatever is achievable and this is increased as the ability to perform the activity improves.

This training aims to recalibrate or enhance the effectiveness of the VOR. The goal is to reduce symptoms of vestibular dysfunction by training away the mismatch between head movement and the corresponding eye movement.

The training also included:

- ▶ Gaze Stabilisation Exercises: These involve maintaining focus on a target while moving the head. This practice can be varied in speed and complexity depending on the patient's progress.
- ▶ Dynamic Visual Acuity Training: This involves reading or recognising objects while the head is in rapid motion, which can help improve the ability to see clearly during movement.
- ▶ Advanced stages of VOR adaptation training might integrate head movements with walking or other body movements to enhance overall vestibular function eg. head shaking while walking.

Entrainment therapy

Entrainment therapy refers to a therapeutic approach that involves synchronising biological rhythms with external stimuli. This synchronisation can be achieved through various forms, such as movement with light or sound cueing. The goal is to entrain poorly regulated neurological processes. It is thought to achieve this by improving the synchronisation of various brain networks.

There are many ways to entrain the nervous system but all involve some sort of rhythmic activity. For this case series we used a computer based program developed in the USA, which

measures efficiency and effectiveness of repeated movements synchronised by an audio stimuli while clapping the hands together.

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. (58)

Optimal interaction with our environment requires processing and relaying sensory information synchronously and precisely. This is particularly important for complex movement, cognition, emotional control, learning and behaviour.

The brain has a strong tendency to make movements automatic, i.e. riding a bike, learning to drive. Learning a new movement begins with difficulty and quickly builds '*muscle memory*'. The brain does this to free up processing power for new possibilities. It is efficient for the brain to make repetitive movements automatic i.e. '*habituated*', which is why habits are hard to break.

The better the brain is at doing this habituation, the more efficient it can be with learning. Entrainment therapy builds to create a brain that is a fast learner.

Dancing is a very good entrainment therapy!

Vestibular stimulation

Vestibular stimulation refers to activities or therapies that activate the vestibular system, the part of the inner ear and brain that helps control balance and eye movements. It can help improve balance, coordination, spacial orientation and motor skills, particularly in individuals who have brain injuries, motor development delays or balance impairments.

Vestibular stimulation is used to treat conditions like vertigo, dizziness, and balance disorders related to vestibular dysfunction and is a key component in sensory integration therapy. It helps in regulating the sensory system. Some studies suggest that vestibular stimulation can have a positive impact on cognitive functions and psychological well-being, reducing symptoms of anxiety and depression. It is used in various neurodevelopmental therapies for children with developmental delays, helping in the development of gross motor skills and spatial orientation.

Every head movement stimulates the vestibular system, both the peripheral organ and the central pathways. Typically, Gymnastics and dancing will stimulate the whole vestibular system.

The central vestibular components are reciprocally connected to many other areas of the central nervous system. Especially the Cerebellum but also the Hypothalamus, Thalamus, Extra-Ocular Muscles, Reticular Activating System, Temporal and Parietal Lobes and Spinal Cord. Directly or indirectly stimulating these structures will also stimulate the vestibular system. (56)

The Multi-Axis Rotating Chair can be rotated in Left or Right Yaw, Anterior or Posterior Pitch with Left or Right sagittal Roll. It has a novel feature in that it will roll in the sagittal plane which is unlike any activity of daily living making it a unique vestibular stimulation. This equipment was used for three of the four cases presented here.

Strengths and limitations in my approach to these cases

The value of my approach is that it has added to the growing elucidation of a seemingly very effective treatment for FND. Its limitation is the small number of cases to statistically verify this statement. Also the combination of treatment approaches presented raises currently unanswerable questions regarding how much effectiveness each element of the treatment contributes.

Discussion of the relevant literature

FND is a common diagnosis in Neurology clinics, 10-30% of initial presentations, (10, 13) 4-10/100,000 adults, (18) with a lot of varying presentations.

In modern times it has been the Neurologist who has treated this cohort. Medication is not recommended for this condition. Despite this, 75% of FND seizure patients have had anti seizure medication. (18) Specialised FND clinics are now emerging (12, 13, 19, 21) This multidisciplinary approach to FND is discussed in the literature where several types of healthcare professionals may be involved in the care of these FND afflicted individuals. (10, 13, 16, 18)

Including Neurologists, Psychologists or Psychotherapists; Psychotherapy, especially cognitive-behavioural therapy (CBT), (21) and other forms of talking therapy are utilised. Physical Therapists may help individuals with FND manage and improve their physical symptoms through physical therapy techniques and exercises. (10, 18) Occupational Therapists, Speech and Language Therapists and Rehabilitation Specialists offer individuals with FND programs that focus on physical and functional recovery. (19) Social Workers can provide support and counselling to both individuals with FND and their families to help cope with the emotional and social challenges associated with the condition. Support Groups, either in-person or online, can be valuable for individuals with FND to connect with others facing similar challenges and to share experiences and coping strategies.

There is no 'one size fit's all' therapy available, (21, 10) meaning individualised therapy is required.

Functional Neurological Disorder typically has a poor prognosis. (18)

Of note is the lack of references to the Chiropractic profession.

From the literature there is a broad consensus that FND is generally a '*brain network dysfunction*' (8, 13, 33) with limbic dysregulation (8, 6, 3, 13) involving sensorimotor, prefrontal areas, the Supplementary Motor Area and many other brain areas. (2, 13)

Neuroimaging has not precisely but generally delineates the structures, tracts and networks that contribute to functional dystonia and functions in motor planning. (18, 21, 33)

Sensory afferents are being poorly modulated which interferes with subsequent motor planning. (13, 21)

Stress, which may be a comorbidity, (13) plays a consistent role with FMD.

An initiating physical event, in 74% of cases, is often involved as are triggers that exacerbate paroxysmal events in 88% of sufferers. (17) Movement is the most common 'Trigger' for paroxysmal episodes of FMD followed by emotional, visual, touch and auditory stimuli. (17) This emphasises the afferent sensory motor involvement.

Other common co-morbid conditions are depression, anxiety, bladder and bowel dysfunction and fibromyalgia.

FND may develop as a functional comorbidity along with other neurological diseases. (15)

Studies have estimated that 12% of neurological patients had functional symptoms coexisting with their recognised neurological conditions. (11, 1)

Building trust and discussing that the patient's symptoms are due to network dysfunction is considered important, rather than allowing the belief that '*it's all in their head*' to dominate. Even using a metaphoric quote from J. Stone '*it's a software problem not a hardware problem*' might be helpful. (16, 20, 21). That '*software*' problem may also be described as a '*disconnection between will and motivation*'.

Either analogy fits with a problem with '*predictive processing*'. (17) In that, with normal movement there is the Bereitschafts potential for estimating gravitational equilibrium before initiating a movement, the generation of the actual command, the creation of an efferent copy of the that command, followed by the monitoring of the movement with comparison to the efferent copy and subsequent correction of the movement. It can be thought that within this complex and

rapid scenario the sensory motor data is corrupted at some point leading to an aberrant movement, thought or emotion. Further that '*corruption*' might be a '*bias*' to a brain network causing it to become dysregulated.

In particular, a decrease in Self-Agency has been considered in the research as possibly contributing to the seizure type FND. That being, the feeling of being able to control external events affecting an individual is called '*self agency*'. When voluntary movement is not matched by it's sensory feedback the mismatch has to be equilibrated and possibly affects associated brain networks and structures.

The vestibular system is phylogenetically, one of the oldest of all the sensory systems while also the earliest to mature during development. (53) The peripheral vestibular organ has changed very little since prehistoric times.

It is a large afferent contributor, therefore it also plays a role in modulating the afferent input to the central nervous system.

The rationale for my conclusion

It is likely Sensory disequilibrium leads to motor disequilibrium. Vestibular Rehabilitation is recognised as an effective method for recovery from brain injury involving TBI ie concussion. (14)

Concussion is a global injury paradigm affecting brain Networks, FND is a network dysfunction paradigm (8, 13, 33) Vestibular Rehabilitation is not specific, ipso facto Vestibular Rehabilitation will also affect the brain networks involved in FND.

There is a significant overlap in the symptoms of both conditions either directly associated or as co-morbidities. Such as '*fogginess*', poor concentration, dizziness, poor balance, poor coordination, headache, loss of consciousness, tinnitus, light headedness, confusion, vision problems, memory problems and cognitive issues, vertigo, etc.

Networks are built from genetic expression and environmental interaction. FND is a network dysfunction. Increasing the environmental/sensory activation either re-establishes the network or resynchronises it. Vestibular Rehabilitation Therapy encompasses exercises of adaption, habituation and substitution, such as Gaze stability, VOR training, COR training, Sensory motor training, balance training, gait training, Cervical proprioceptive retraining. Spinal Adjustments also contribute to the reintegration of the sensory motor system.

From the aforementioned cases it can be seen that a multimodal approach to the correction of FND is likely to be a successful model.

It can be considered that the Vestibular system, with it's vast interconnectedness to the central nervous system can be relied upon to modulate the neuraxis perhaps even being a driver of '*homeostasis*' allowing the sensory system to more efficiently integrate into the central nervous system.

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References

1. Stone, J. et al. (2009) 'Symptoms "unexplained by organic disease" in 1144 new neurology out-patients: How often does the diagnosis change at follow-up?'. *Brain*, 132(10), pp. 2878–2888. DOI 10.1093/brain/awp220.
2. Perez, D.L. et al. (2014) 'An integrative Neurocircuit perspective on psychogenic nonepileptic seizures and functional movement disorders', *Clinical EEG and Neuroscience*, 46(1), pp. 4–15. DOI 10.1177/1550059414555905.
3. Tinazzi, M. et al. (2021) 'Functional motor phenotypes: To Lump or to split?'. *Journal of Neurology*, 268(12), pp. 4737–4743. DOI 10.1007/s00415-021-10583-w.
4. Kanaan, R.A. (2022) 'Functional neurological disorder and other unexplained syndromes', *The Lancet Neurology*, 21(6), pp. 499–500. DOI 10.1016/s1474-4422(22)00095-3.
5. Ndetan, H. et al. (2015) 'The role of chiropractic care in the treatment of dizziness or balance disorders', *Journal of Evidence-Based Complementary & Alternative Medicine*, 21(2), pp. 138–142. DOI 10.1177/2156587215604974.
6. Roelofs, J.J., Teodoro, T. and Edwards, M.J. (2019) 'Neuroimaging in functional movement disorders', *Current Neurology and Neuroscience Reports*, 19(3). DOI 10.1007/s11910-019-0926-y.
7. Bach-y-Rita, P. (1990) 'Brain plasticity as a basis for recovery of function in humans', *Neuropsychologia*, 28(6), pp. 547–554. DOI 10.1016/0028-3932(90)90033-k.
8. Hallett, M. et al. (2022) 'Functional neurological disorder: New subtypes and shared mechanisms', *The Lancet Neurology*, 21(6), pp. 537–550. DOI 10.1016/s1474-4422(21)00422-1.
9. Potgieser, A.R. et al. (2014) 'Insights from the supplementary motor area syndrome in balancing movement initiation and inhibition', *Frontiers in Human Neuroscience*, 8. DOI 10.3389/fnhum.2014.00960.
10. Polich, G. et al. (2022) 'Development of an inpatient rehabilitation pathway for Motor Functional Neurological Disorders: Initial reflections', *NeuroRehabilitation*, 50(2), pp. 231–243. DOI 10.3233/nre-228006.
11. Stone, J. et al. (2011) 'Which neurological diseases are most likely to be associated with "symptoms unexplained by organic disease"', *Journal of Neurology*, 259(1), pp. 33–38. DOI 10.1007/s00415-011-6111-0.
12. Delgado, C. et al. (2021) 'Clinical and demographic characteristics of patients with functional movement disorders: A consecutive cohort study from a specialized clinic', *Acta Neurologica Belgica*, 122(1), pp. 97–103. DOI 10.1007/s13760-021-01648-8.
13. Kelmanson, A.N., Kalichman, L. and Treger, I. (2023) 'Physical rehabilitation of Motor Functional Neurological Disorders: A narrative review', *International Journal of Environmental Research and Public Health*, 20(10), p. 5793. DOI 10.3390/ijerph20105793.
14. Schlemmer, E. and Nicholson, N. (2022) 'Vestibular rehabilitation effectiveness for adults with mild traumatic brain injury/concussion: A mini-systematic review', *American Journal of Audiology*, 31(1), pp. 228–242. DOI 10.1044/2021_aja-21-00165.
15. Gómez-Mayordomo, V. et al. (2022) 'Functional neurological symptoms as initial presentation of Creutzfeldt-Jakob Disease: Case Series', *Journal of Neurology*, 270(2), pp. 1141–1146. DOI 10.1007/s00415-022-11376-5.
16. Phansalkar, R. et al. (2022) 'Management of Functional Vision Disorders', *Current Neurology and Neuroscience Reports*, 22(4), pp. 265–273. DOI 10.1007/s11910-022-01191-w.
17. Geroin, C. et al. (2022) 'Triggers in functional motor disorder: A clinical feature distinct from precipitating factors', *Journal of Neurology*, 269(7), pp. 3892–3898. DOI 10.1007/s00415-022-11102-1
18. Perjoc, R.-S. et al. (2023) 'Functional neurological disorder—old problem new perspective', *International Journal of Environmental Research and Public Health*, 20(2), p. 1099. DOI 10.3390/ijerph20021099.
19. Polich, G. et al. (2022) 'Intensive rehabilitation for Functional Motor Disorders (FMD) in the United States: A Review', *NeuroRehabilitation*, 50(2), pp. 245–254. DOI 10.3233/nre-228007.
20. Kola, S. and LaFaver, K. (2022) 'Updates in functional movement disorders: From pathophysiology to treatment advances', *Current Neurology and Neuroscience Reports*, 22(5), pp. 305–311. DOI 10.1007/s11910-022-01192-9.
21. Gilmour, G.S. et al. (2020) 'Management of functional neurological disorder', *Journal of Neurology*, 267(7), pp. 2164–2172. DOI 10.1007/s00415-020-09772-w.
22. Leigh, R.J. and Zee, D.S. (2015) 'Chapter 4 The Saccadic System', in *The neurology of Eye Movements*. Oxford, New York: Oxford University Press, pp. 169–251.

23. Preteggiani, E. and Optican, L.M. (2017) 'Eye movements in parkinson's disease and inherited parkinsonian syndromes', *Frontiers in Neurology*, 8. DOI 10.3389/fneur.2017.00592.
24. Kassavetis, P. et al. (2022) 'Eye movement disorders in movement disorders', *Movement Disorders Clinical Practice*, 9(3), pp. 284–295. DOI 10.1002/mdc3.13413.
25. Cortez, M.M. et al. (2017) 'Altered pupillary light response scales with disease severity in migrainous photophobia', *Cephalalgia*, 37(8), pp. 801–811. DOI 10.1177/0333102416673205.
26. Pinheiro, H.M. and da Costa, R.M. (2021) 'Pupillary Light Reflex as a diagnostic aid from Computational Viewpoint: A systematic literature review', *Journal of Biomedical Informatics*, 117, p. 103757. DOI 10.1016/j.jbi.2021.103757.
27. Shah, S.S. et al. (2020) 'Establishing a normative database for quantitative pupillometry in the pediatric population', *BMC Ophthalmology*, 20(1). DOI 10.1186/s12886-020-01389-x.
28. Joshi, S. and Gold, J.I. (2020) 'Pupil size as a window on neural substrates of cognition', *Trends in Cognitive Sciences*, 24(6), pp. 466–480. DOI 10.1016/j.tics.2020.03.005.
29. Truong, J.Q. and Ciuffreda, K.J. (2016) 'Objective pupillary correlates of photosensitivity in the normal and mild traumatic brain injury populations', *Military Medicine*, 181(10), pp. 1382–1390. DOI 10.7205/milmed-d-15-00587.
30. Carrick, F.R. et al. (2021) 'The pupillary light reflex as a biomarker of concussion', *Life*, 11(10), p. 1104. DOI 10.3390/life11101104.
31. McDonald, M.A., Danesh-Meyer, H.V. and Holdsworth, S. (2022) 'Eye movements in mild traumatic brain injury: Ocular biomarkers', *Journal of Eye Movement Research*, 15(2). DOI 10.16910/jemr.15.2.4.
32. Ciuffreda, K.J., Joshi, N.R. and Truong, J.Q. (2017) 'Understanding the effects of mild traumatic brain injury on the Pupillary Light Reflex', *Concussion*, 2(3). DOI 10.2217/cnc-2016-0029.
33. Perez, D.L. et al. (2014) 'An integrative Neurocircuit perspective on psychogenic nonepileptic seizures and functional movement disorders', *Clinical EEG and Neuroscience*, 46(1), pp. 4– 15. DOI 10.1177/1550059414555905.
34. Knox, P.C. and Pasunuru, N. (2020) 'Age-related alterations in inhibitory control investigated using the minimally delayed oculomotor response task', *PeerJ*, DOI 10.7717/peerj.8401.
35. Jacobson, G.P. et al. (1993) 'Interpretation and Usefulness of Ocular Motility Testing', in *Handbook of Balance Function Testing*. St. Louis, Missouri: Mosby Year Book, pp. 101– 122.
36. Heitger, M.H. et al. (2009) 'Impaired eye movements in post-concussion syndrome indicate suboptimal brain function beyond the influence of depression, malingering or intellectual ability', *Brain*, 132(10), pp. 2850–2870. DOI 10.1093/brain/awp181.
37. Pinheiro, H.M. and da Costa, R.M. (2021) 'Pupillary Light Reflex as a diagnostic aid from Computational Viewpoint: A systematic literature review', *Journal of Biomedical Informatics*, 117, p. 103757. DOI 10.1016/j.jbi.2021.103757.
38. Quaia, C., Lefevre, P. and Optican, L.M. (1999) Model of the control of saccades by superior colliculus and cerebellum ..., *Journal of Neurophysiology*. Available at: <https://journals.physiology.org/DOI/abs/10.1152/jn.1999.82.2.999> (Accessed: 21 November 2023).
39. Termsarasab, P. et al. (2015) 'The diagnostic value of saccades in movement disorder patients: A practical guide and review', *Journal of Clinical Movement Disorders*, 2(1). DOI 10.1186/s40734-015-0025-4.
40. Johnston, J.L., Daye, P.M. and Thomson, G.T. (2017) 'Inaccurate saccades and enhanced vestibulo-ocular reflex suppression during combined eye-head movements in patients with chronic neck pain: Possible implications for cervical vertigo', *Frontiers in Neurology*, 8. DOI 10.3389/fneur.2017.00023.
41. TERA0, Y., FUKUDA, H. and HIKOSAKA, O. (2017) 'What do eye movements tell us about patients with neurological disorders? An introduction to saccade recording in the clinical setting', *Proceedings of the Japan Academy, Series B*, 93(10), pp. 772–801. DOI 10.2183/pjab.93.049.
42. Strupp, M. et al. (2011) 'Central oculomotor disturbances and nystagmus', *Deutsches Ärzteblatt international* [Preprint]. DOI 10.3238/arztebl.2011.0197.
43. Shaikh, A.G. and Zee, D.S. (2017) 'Eye Movement Research in the twenty-first century—a window to the brain, mind, and more', *The Cerebellum*, 17(3), pp. 252–258. DOI 10.1007/s12311-017-0910-5.
44. Strupp, M. and Brandt, T. (2009) 'Review: Current treatment of vestibular, ocular motor disorders and Nystagmus', *Therapeutic Advances in Neurological Disorders*, 2(4), pp. 223– 239. DOI 10.1177/1756285609103120.
45. Terao, Y. et al. (2016) 'Distinguishing spinocerebellar ataxia with pure cerebellar manifestation from multiple system atrophy (MSA-C) through Saccade Profiles', *Clinical Neurophysiology*, 128(1), pp. 31–43. DOI 10.1016/j.clinph.2016.10.012.
46. Krauzlis, R.J., Goffart, L. and Hafed, Z.M. (2017) 'Neuronal control of fixation and fixational eye movements', *Philosophical Transactions of the Royal Society B: Biological Sciences*, 372(1718), p. 20160205. DOI 10.1098/rstb.2016.0205.
47. Geiger, A. et al. (2017) 'Eyes versus hands: How perceived stimuli influence motor actions', *PLOS ONE*, 12(7). DOI 10.1371/journal.pone.0180780.

48. Otto, M.A. (2017) Eye movement, not CT or MRI, rules out posterior stroke, MDedge Cardiology. Available at: <https://www.mdedge.com/cardiology/article/149510/stroke/eye-movement-not-ct-or-mri-rules-out-posterior-stroke> (Accessed: 21 November 2023).
49. Zee, D.S. (2012) 'What the future holds for the study of saccades', *Biocybernetics and Biomedical Engineering*, 32(2), pp. 65–76. DOI 10.1016/s0208- 5216(12)70037-2.
50. Srivastava, A. et al. (2018) 'The relationship between saccades and Locomotion', *Journal of Movement Disorders*, 11(3), pp. 93–106. DOI 10.14802/jmd.18018.
51. Coe, B.C., Trappenberg, T. and Munoz, D.P. (2019) 'Modeling saccadic action selection: Cortical and basal ganglia signals coalesce in the superior colliculus', *Frontiers in Systems Neuroscience*, 13. DOI 10.3389/fnsys.2019.00003.
52. Lloyd-Smith Sequeira, A., Rizzo, J.-R. and Rucker, J.C. (2017) 'Clinical approach to supranuclear brainstem saccadic gaze palsies', *Frontiers in Neurology*, DOI 10.3389/fneur.2017.00429.
53. Cronin, T., Arshad, Q. and Seemungal, B.M. (2017) 'Vestibular deficits in neurodegenerative disorders: Balance, dizziness, and spatial disorientation', *Frontiers in Neurology*, 8. DOI 10.3389/fneur.2017.00538.
54. Demian, D. et al. (2023) 'Clinical saccadometry: Establishing evaluative standards using a simplified video oculography protocol in the adult population', *Journal of the American Academy of Audiology* [Preprint]. DOI 10.1055/s-0043-1772582.
55. Otero-Millan, J. et al. (2018) 'Modeling the triggering of saccades, microsaccades, and saccadic intrusions', *Frontiers in Neurology*, 9. DOI 10.3389/fneur.2018.00346.
56. Kheradmand, A. and Winnick, A. (2017) 'Perception of upright: Multisensory convergence and the role of temporo-parietal cortex', *Frontiers in Neurology*, 8. DOI 10.3389/fneur.2017.00552.
57. Blume, J. et al. (2017) 'Saccadic impairments in patients with the norrbottnian form of Gaucher's Disease Type 3', *Frontiers in Neurology*, 8. DOI 10.3389/fneur.2017.00295.
58. Preteggiani, E. and Optican, L.M. (2017) 'Eye movements in parkinson's disease and inherited parkinsonian syndromes', *Frontiers in Neurology*, 8. DOI 10.3389/fneur.2017.00592.
59. Leech, R. and Sharp, D.J. (2013) 'The role of the posterior cingulate cortex in cognition and disease', *Brain*, 137(1), pp. 12–32. DOI 10.1093/brain/awt162.
60. Rizzo, J.-R. et al. (2017) 'Eye control deficits coupled to hand control deficits: Eye–hand incoordination in chronic cerebral injury', *Frontiers in Neurology*, 8. DOI 10.3389/fneur.2017.00330.
61. Rizzo, J.-R., Hosseini, M., et al. (2017) 'The intersection between ocular and Manual Motor Control: Eye–Hand Coordination in acquired Brain Injury', *Frontiers in Neurology*, 8. DOI 10.3389/fneur.2017.00227.

About the Chiropractor

David Richardson has an interest in chronic and difficult cases that fail to respond to the usual treatments. Since graduating from RMIT University Melbourne in 1982, David has run practices in Europe and Australia. He emphasises the 'team work' approach to care; in that the doctor-patient relationship must be mutually respectful, completely honest and with a high expectation of co-operation.

David has a particular interest in clinical applications of Neuroscience involving Vestibular stimulation and sensory-motor integration.

