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CLINICALEVIDENCE
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A novel approach to Neurorehabilitation for persisting Post Concussion Syndrome with occupational reintegration in 2 males aged 34 & 43:

A Case Series of 2

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Overview: Persisting post-concussive symptoms (PPCS), defined as three or more symptoms persisting beyond three months after mild traumatic brain injury, affect 24-43% of concussed individuals and cause substantial occupational disability. This case series of two working-age men with PPCS >12 months documents multimodal neurorehabilitation and objective recovery metrics alongside return-to-work outcomes.

Therapeutic Interventions: Both patients received an individualised multimodal neurorehabilitation program comprising the following modalities applied at patient-specific dose and progression: Vestibular stimulation via the Orbital multi-axis rotational chair, incorporating sagittal roll, yaw, and pitch planes as determined by individual assessment findings; Saccade and pursuit rehabilitation using a weighted head-mounted laser with computer-generated random target panels in LARP and RALP diagonal planes; Sensorimotor integration exercises including backwards treadmill walking with ankle weights and con-current juggling (P1) and reaction timer panel training with gap scenario (P2); Entrainment therapy, audiovisual feedback synchronised with bilateral hand clapping; Spinal adjustments addressing identified segmental dysfunction; Oculomotor home exercises: vergence card, horizon to thumb tracking (P2), anti-saccade training using an iPad App (P1); Repetitive Peripheral Somatosensory Evoked Potential (RPSEP) stimulation of four Trigeminal (CN V) nerve locations using a peripheral nerve stimulator, one minute per location (P2); Dietary and supplemental adjuncts discussed for P2 (1, 2, 3): ketogenic diet as neuro-inflammatory mitigation. P1 completed 45 treatment sessions over 16 months. P2 completed 42 treatment sessions over 10 months.

Outcomes: P1 improved objectively; post-treatment measures rose; returned to full-time electrician with no relapse after four years. P2 substantially advanced professionally.

Conclusion: These cases show chronically symptomatic PPCS patients can achieve neurophysiological gains and employment after individualised multimodal rehab; objective saccadometry and posturography changes align with return to work, including vocational reorientation.

Indexing Terms: Chiropractic; post concussion syndrome; PPCS; case series; return to work; vestibular rehabilitation; sensory motor integration; spinal manipulation; multi axis rotation.

Introduction

> *'... occupational reintegration, in whatever form it takes, as a primary outcome measure rather than a secondary observation, is valuable outcome measure...'*

Mild traumatic brain injury (mTBI) is estimated to affect approximately 69 million people globally each year, accounting for 70–90% of all hospital-treated brain injuries. (4) Persisting post-concussive symptoms (PPCS), defined as three or more symptoms sustained beyond three months from injury, occur in 24–43% of those who sustain a concussion (5) and represent a substantial source of occupational disability in working-age adults. Results of a systematic review suggest that most workers return to employment within three to six months of mTBI; however, 5–20% continue to experience work limitations for one to two years post-injury, (6) and some experience protracted impairment beyond this window.

Despite growing interest in multimodal neurorehabilitation for PPCS, the literature contains few cases in which serial objective neurophysiological assessment is used to document treatment response in parallel with vocational reintegration outcomes. The two cases reported here describe working-age males with PPCS extending beyond twelve months, a cohort often characterised as having a poor rehabilitation prognosis, each of whom underwent an individualised, assessment-driven multimodal program and achieved meaningful occupational reintegration.

The multi-domain deficit profiles of both patients, spanning autonomic pupillary dysfunction, oculomotor impairment, vestibular disruption, postural instability, and cervicogenic pathology, are consistent with the distributed central nervous system disruption that characterises chronic PPCS (7) and with the recognised heterogeneity of post-concussive presentation that limits the effectiveness of unimodal approaches. Saccadometry, particularly the anti-saccade paradigm provides a sensitive and reproducible measure of frontal-cerebellar circuit integrity, with anti-saccade latency correlating with white matter diffusivity, executive function, and post-concussive symptom burden. (8, 9, 10) Instrumented force plate posturography quantifies postural control deficits that persist well beyond symptomatic resolution (11) and are not reliably detected by clinical balance tests alone. (12) Together these tools provide an objective neurophysiological battery capable of documenting both impairment at presentation and treatment response across the rehabilitation course. (7, 13) The occupational outcomes of these two cases, full return to skilled trade employment and novel vocational self-employment, are reported as primary functional endpoints alongside neurophysiological data, consistent with the recommendation that return to work be treated as a primary rather than subsidiary outcome measure in PPCS rehabilitation research. (6, 14)

Patient overview

Patient 1, male aged 34

Patient 1 is a 34 year old male electrician. He presented to the clinic having suffered a motor vehicle accident 2 years previously. He was driving a motor scooter on the island of Cyprus and hit a tree, sustaining multiple skull fractures, necessitating an airlift to Athens hospital with subsequent treatment (October 2018).

His presenting concerns were foggy brain, poor memory, depression, social anxiety, excessive flatulence, morning headaches upon arising that lasted for two hours, low back pain with left leg radiation to the knee and an inability to work as an electrician since the accident.

Medication at that time included Valoxin, Endep, Graceda being a statin for hypercholesterolemia and Prenapril for hypertension.

He had no significant pre-injury neurological or vestibular history. Psychosocial history was notable for the occupational and financial consequences of prolonged inability to work in his trade, with associated motivational and mood impact that was present throughout the treatment period. No family history of neurological or vestibular disorder was identified. No pharmacological treatment with known influence on vestibulo-oculomotor function was recorded at the time of initial assessment.

The patient reported receiving standard medical management following mTBI prior to presentation. The specific nature and duration of prior interventions in Europe were unknown to him.

Patient 2, male aged 43

Patient 2 was an Automotive Spray Painter, and presented with constant headache, dizziness, memory loss, fatigue, and back pain.

On his initial visit, he was unable to converse coherently without leaning against a wall. He complained of blurred vision, phonophobia, confusion, insomnia, depression, irritability, poor memory, and felt overwhelmed. The symptoms had appeared following an accident, 4 years previously, when he fell from a child's scooter hitting his head on concrete to fracture his skull, describing it as a fracture from his left ear to his left eye. P2 had been on a disability pension since the accident and has suffered some seizures at the time of the incident and occasionally since.

The seizures were described as 'tonic clonic' for which he has the medication sodium valproate, other medications include Diazepam, Temazepam, Citolopram, Atisan, Periactin a Norspan patch.

He had been previously diagnosed with chronic PPCS.

The patient had no significant pre-injury neurological or vestibular history. Psychosocial history was notable for significant occupational and financial disruption consequent to prolonged work absence and inability to resume his trade.

Patient clinical findings

Patient 1

Spinal and Neurological Examination

Physical examination identified cervical segmental dysfunction with restricted range of motion consistent with co-existing cervicogenic pathology. Upper cervical joint dysfunction has been documented as a feature of post-concussive headache and dizziness presentations, arising from the soft tissue and facet joint loading generated by the same biomechanical event producing the cerebral injury. (15) A systematic review and meta-analysis of spinal manipulative therapy (SMT) for cervicogenic headache demonstrated significant moderate-to-large effects in favour of SMT for headache frequency and intensity at short-term follow-up, (16) supporting inclusion of spinal adjustments in the management of this cohort.

Neurological examination was otherwise consistent with multi-domain PPCS without focal lateralising deficit. UPDRS Finger Tapping scored Right - slow with 3-5 interruptions, Left - >5

interruptions, Dysdiadokokinesia scored Right - <10s and dysmetric, Left - >5 interruptions. Rhomberg's Tandem failed at 12s, One Leg Standing failed on the Left at 9s and Right 10s.

Dynamic Visual Acuity Testing using the Snellen Chart showed a static ability to read Line 8 whereas with head movement P1 could only achieve Line 4, suggesting a dysfunctional Vestibulo-occula Reflex. Near Point Convergence testing was 12cms (N=6). Horizontal Smooth Pursuit showed Left eye movement dysmetria.

Fakuda testing indicated a left vestibular deficit.

Saccadometry

Pre-treatment saccadometry demonstrated elevated latency with bilateral asymmetry and high inter-trial trajectory variability, consistent with frontal-cerebellar circuit disruption following chronic mTBI. (8, 17)

Oculomotor findings extended to abnormalities in saccadic pursuit and overlap paradigm performance. These findings are consistent with the published saccadometric characterisation of mTBI populations, in which latency is prolonged relative to age-matched controls (8) and correlates with corpus callosum white matter diffusivity abnormality, executive function impairment, and PPCS symptom burden. (9) Saccade and oculomotor performance in the week following mTBI has been shown to be significantly predictive of greater symptom burden at three and six months post-injury.10 Pre- and post-treatment saccadometric latency plots are presented in P1 Saccadometry Latency Plots (Figure 1).

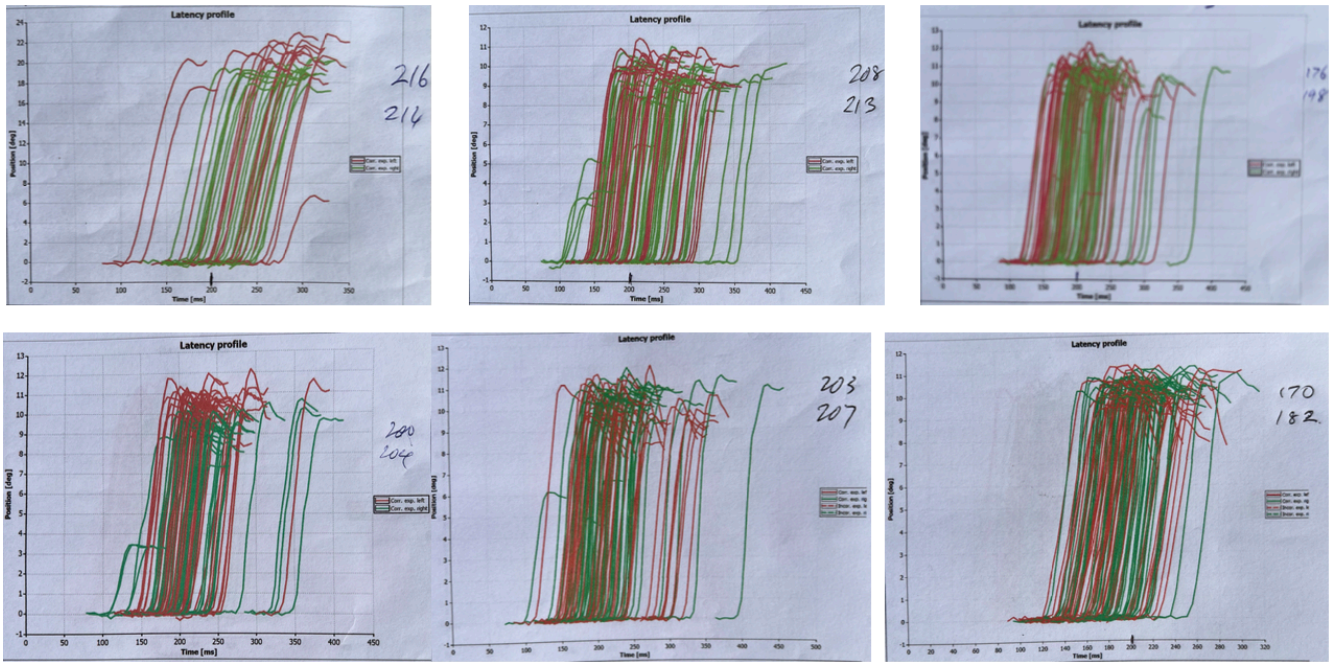

Figure 1: Patient 1 Saccadometry Latency Plots

Of note in this series is that from the top left, the initial plot shows a sparsity of readings, indicating that the brain initiated eye movements that were too slow to be recorded while the spread of the entire group represents the brain's inability to reproduce an identical saccade to the same target with the same timing consistently. Clearly a sign of a dysfunctional nervous system.

The top middle plot shows some green rightward looking preemptive saccades to the left of the main group, which are erroneously initiated by the superior colliculus of the brain stem. Again a dysfunctional reaction to a voluntary saccade. Throughout the treatment period there is a positive progression, however the disparity between sides represents a neurological conflict.

Average Latency time	10-11-20	23-12-20	11-03-21	11-08-21	23-11-21	20-01-22
Red Left Looking Saccades	216	208	176	200	203	170
Green Right Looking Saccades	214	213	198	204	207	182

Table 1: N=15-200ms

Force Plate Posturography

Pre-treatment instrumented force plate posturography demonstrated impaired postural stability consistent with the persistent neuromotor control deficits documented in chronic PPCS. Force plate measures have been shown to detect ongoing functional impairment in PPCS populations (12) and to identify deficits not apparent on conventional clinical balance tests such as the Balance Error Scoring System. (11) Athletes and workers with mTBI history demonstrate significantly poorer stability than controls during balance perturbations and quiet stance, with differences accentuated in eyes-closed conditions, (11) a pattern consistent with the reliance on visual substitution for vestibular impairment observed clinically in PPCS. Pre- and post-treatment posturographic data are presented in Figure 2

Force Plate Analysis: Pre vs Post Treatment

P1 Pre-treatment

Test	Eyes Open, Firm Plat-	Eyes Closed, Firm Plat-	Eyes Open, Soft Plat-	Eyes Closed, Soft Plat-
Date	15 Jan 2018 – 10:10	15 Jan 2018 – 10:11	15 Jan 2018 – 10:12	15 Jan 2018 – 10:13
Mean Distance	4.5 (Poor 138%)	3.7 (Normal 91%)	6.9 (Normal 111%)	10.3 (Good 74%)
Mean Distance	4.2 (Very Poor 168%)	3.3 (Normal 102%)	5.4 (Normal 112%)	8.8 (Good 78%)
Mean Distance	1.0 (Good 67%)	1.0 (Very Good 58%)	3.3 (Normal 109%)	3.8 (Good 63%)
RMS Distance	5.2 (Very Poor 141%)	4.5 (Normal 98%)	7.9 (Normal 111%)	12.1 (Good 76%)
RMS Distance AP	5.0 (Very Poor 167%)	4.3 (Normal 109%)	6.8 (Normal 117%)	10.9 (Good 79%)
RMS Distance	1.3 (Good 66%)	1.4 (Very Good 59%)	4.1 (Normal 108%)	5.1 (Good 66%)
Range AP	25.4 (Very Poor 180%)	25.7 (Poor 138%)	38.3 (Very Poor 142%)	62.6 (Normal 95%)
Range ML	8.1 (Normal 86%)	9.5 (Normal 87%)	26.6 (Very Poor 153%)	38.8 (Normal 101%)
Mean Velocity	11.8 (Very Poor 158%)	15.9 (Very Poor 156%)	31.7 (Very Poor 225%)	60.4 (Very Poor 161%)
Mean Velocity	9.8 (Very Poor 178%)	13.6 (Very Poor 155%)	25.7 (Very Poor 246%)	51.7 (Very Poor 173%)
Mean Velocity	5.0 (Very Poor 145%)	5.7 (Poor 130%)	14.5 (Very Poor 223%)	22.2 (Poor 139%)

Test	Eyes Open, Firm Plat-	Eyes Closed, Firm Plat-	Eyes Open, Soft Plat-	Eyes Closed, Soft Plat-
95% Ellipse Area	120.7 (Normal 114%)	109.8 (Good 67%)	516.6 (Very Poor 248%)	1049.1 (Very Poor
Sway Area	11.9 (Very Poor 143%)	12.9 (Normal 111%)	57.4 (Very Poor 348%)	166.9 (Very Poor 391%)
Mean Frequency	0.4 (Normal 110%)	0.7 (Very High 169%)	0.7 (Normal 100%)	0.9 (Low 62%)
Mean Frequency	0.4 (Normal 101%)	0.7 (Very High 171%)	0.8 (Normal 107%)	1.0 (Low 66%)
Mean Frequency	0.9 (Very High 171%)	1.0 (Very High 173%)	0.8 (Low 76%)	1.0 (Very Low 51%)
Adaptation/Fa-	107.3% (Fatigue)	10.7% (Fatigue)	4.7% (Fatigue)	8.6% (Adaptation)
Balance Age	56.4	39.2	62.7	40.7
Falls Risk	15.6%	12.9%	17.8%	13.0%

P2 Post treatment

Test	Eyes Open, Firm Plat-	Eyes Closed, Firm Plat-	Eyes Open, Soft Plat-	Eyes Closed, Soft Plat-
Date	9 Jun 2022 – 10:15 am	9 Jun 2022 – 10:16 am	9 Jun 2022 – 10:20 am	9 Jun 2022 – 10:21 am
Mean Distance	2.3 (Good 71%)	2.6 (Good 65%)	4.8 (Good 77%)	6.6 (Very Good 47%)
Mean Distance	2.0 (Normal 82%)	2.4 (Good 76%)	4.2 (Normal 87%)	5.2 (Very Good 46%)
Mean Distance	0.7 (Very Good 46%)	0.6 (Very Good 35%)	1.6 (Very Good 52%)	2.9 (Very Good 49%)
RMS Distance	2.9 (Good 79%)	3.2 (Good 69%)	5.8 (Normal 81%)	7.7 (Very Good 48%)
RMS Distance AP	2.8 (Normal 92%)	3.1 (Good 79%)	5.4 (Normal 93%)	6.7 (Very Good 48%)
RMS Distance	1.0 (Very Good 51%)	0.7 (Very Good 33%)	2.0 (Very Good 54%)	3.8 (Very Good 49%)
Range AP	27.7 (Very Poor 197%)	15.3 (Normal 82%)	28.4 (Normal 105%)	40.1 (Good 61%)
Range ML	6.8 (Good 72%)	3.8 (Very Good 35%)	12.6 (Good 73%)	23.0 (Very Good 60%)
Mean Velocity	9.9 (Poor 133%)	9.2 (Normal 90%)	14.0 (Normal 99%)	29.9 (Good 80%)
Mean Velocity	8.5 (Very Poor 154%)	8.4 (Normal 96%)	11.7 (Normal 112%)	23.5 (Good 79%)
Mean Velocity	3.6 (Normal 105%)	2.6 (Very Good 60%)	5.6 (Normal 86%)	13.5 (Normal 85%)
95% Ellipse Area	51.8 (Very Good 49%)	43.4 (Very Good 27%)	204.7 (Normal 98%)	469.6 (Good 79%)
Sway Area	7.7 (Normal 93%)	4.2 (Very Good 36%)	16.2 (Normal 98%)	51.5 (Poor 121%)
Mean Frequency	0.7 (Very High 181%)	0.6 (High 136%)	0.5 (Low 64%)	0.7 (Very Low 48%)
Mean Frequency	0.7 (Very High 179%)	0.6 (Very High 141%)	0.5 (Low 62%)	0.8 (Very Low 51%)
Mean Frequency	0.9 (Very High 181%)	0.7 (High 131%)	0.6 (Low 61%)	0.8 (Very Low 40%)
Adaptation/Fa-	41.6% (Adaptation)	1.2% (Fatigue)	17.5% (Adaptation)	7.2% (Fatigue)
Balance Age	33.2	22.7	26.5	23.7
Falls Risk	12.5%	12.3%	12.3%	12.3%

Patient 2

Spinal and Neurological Examination

Cervical examination revealed restricted range of motion and segmental dysfunction probably contributing to the cervicogenic headache and dizziness components of P2's presentation. Chiropractic management of craniocervical junction dysfunction in post-concussion syndrome has been reported to produce substantive improvement in dizziness, headache, and related symptoms across a case series of patients with PPCS duration of at least six weeks, (15) with evidence that cervical dysfunction acts as a significant contributing factor to symptom perpetuation in a proportion of PPCS patients. (15)

Spinal adjustments targeting identified segmental dysfunction were incorporated into the treatment program on this basis. Raglans Test produced a diastolic change of 20mmHg from supine to standing at 3mins suggesting Orthostatic Tachycardia as a confounding condition. The occulocarotid reflex was slow to respond taking 15s as was the carotid body reflex taking 11s (N<5s)

Convergence and Oculomotor Assessment

Patient 2 presented with convergence insufficiency (CI) of 15cms (N=6). CI is a binocular vision disorder characterised by receded near point of convergence, reduced positive fusional vergence, and impaired vergence facility. Symptomatic CI has a reported prevalence of ~40% in adults with PPCS two to six months post-injury, (18) with convergence-insufficient patients demonstrating significantly higher Rivermead Post Concussion Symptoms Questionnaire scores than those without CI. (18) Military and civilian studies report CI prevalence rates of 23–42% following mTBI, (19) and office-based vergence and accommodative rehabilitation has been shown to improve clinical signs of CI and produce measurable neuroplastic changes in the vergence oculomotor network, specifically in the frontal eye fields, supplemental eye fields, parietal eye fields, cerebellar vermis, and visual cortex, as demonstrated in the CONCUSS clinical trial. (20) P2's convergence deficit produced direct functional limitations in near-vision tasks including reading, fine-detail work, and any near-field visual demand critical to vocational performance and to the online retraining subsequently undertaken. Maddox Rod assessment at 1m showed convergence of 50mm. Horizontal Smooth Pursuit showed multiple saccadic inclusions.

Saccadometry

Pre-treatment saccadometry demonstrated elevated latency and disordered trajectory profiles in both overlap paradigms, consistent with the frontal-cerebellar circuit impairment documented in chronic PPCS. (8, 9) Post-treatment saccadometry demonstrated objective improvement from baseline. Pre- and post-treatment saccadometric data are presented in Figure 3.

As mentioned saccadometry produces a huge volume of interpretive information and these latency plots are a small representation of the quantity of data that saccadometry can produce. Of note here is, that, in the pre-treatment latency plot there is a collection of green right looking saccades at around 100ms which indicate dysfunction of voluntary saccadic system. The post treatment plots show a shift of these 'express' saccades into the normal range, indicating improved eye movement control function which implies improved neurological activity. The difference between the sides was 35ms reduced to 18ms (N= 15-20ms).

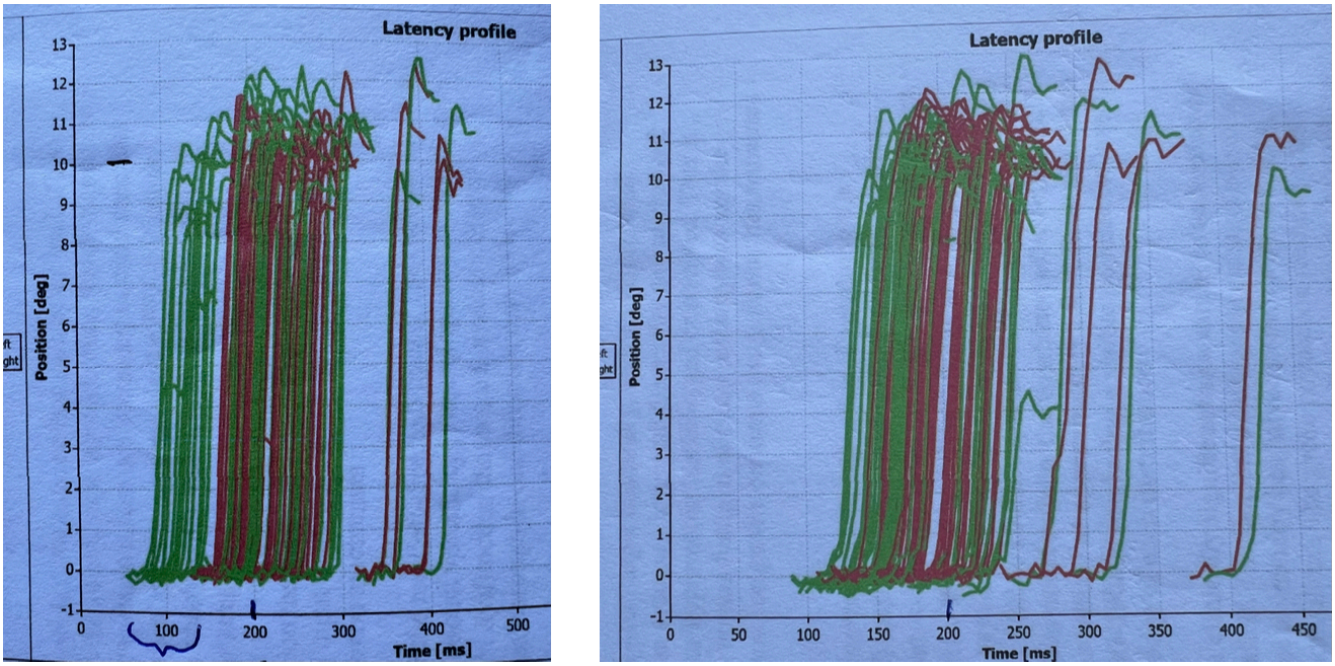

Figure 3: Patient 2 Saccadometry Latency Plots

LATENCY AVERAGES	Pre Tx	Post Tx
Red Left Looking Saccades	235	201
Green Right Looking Saccades	200	183

Table 2.

Force Plate Posturography

Pre-treatment force plate posturography confirmed objective postural instability consistent with persistent balance impairment in chronic PPCS. (11, 12) Post-treatment force plate posturography demonstrated objective improvement from baseline.

This force plate analysis represents severe multi system postural control failure, characterised by critical visual dependency with near total postural collapse without vision on a firm surface. It indicates that proprioception alone on a firm surface is completely inadequate to maintain upright stability. He is entirely dependent on visual input for posture control on a firm surface. When vision is removed the postural control system fails rather than successfully switching to proprioceptive and/or vestibular references, as would normally occur. Recall that this chap, on initial presentation, had to lean against a wall in order to talk!

The paradoxical relative preservation of performance on a soft platform compared to a firm platform without vision indicates that the patient cannot effectively use firm surface proprioceptive input as a postural reference when visual input is unavailable. This suggests disruption of the central integration of proprioceptive and vestibular signals, likely at the level of the vestibular nuclei and their cerebella connections, which are responsible for weighting and combining these inputs for postural control.

The absence of within-trial adaption in each of the 4 testing conditions confirms that this patient has no postural adaptive reserve, every condition taxes the system to its absolute limit.

Force Plate Analysis: Pre vs Post Treatment

P2 Pre-treatment

Test	Eyes Open, Firm Plat-	Eyes Closed, Firm Plat-	Eyes Open, Soft Plat-	Eyes Closed, Soft Plat-
Date	11 Aug 2020 – 11:16	11 Aug 2020 – 11:17	11 Aug 2020 – 11:19	11 Aug 2020 – 11:19
Mean Distance	6.7 (Very Poor 204%)	19.1 (Very Poor 468%)	7.5 (Normal 116%)	21.4 (Very Poor 150%)
Mean Distance	6.1 (Very Poor 239%)	18.3 (Very Poor 561%)	5.0 (Normal 100%)	15.4 (Poor 134%)
Mean Distance	1.8 (Normal 117%)	3.4 (Very Poor 192%)	4.5 (Very Poor 145%)	11.0 (Very Poor 179%)
RMS Distance	8.6 (Very Poor 228%)	22.2 (Very Poor 476%)	8.6 (Normal 116%)	24.7 (Very Poor 152%)
RMS Distance AP	8.3 (Very Poor 271%)	21.8 (Very Poor 550%)	6.3 (Normal 105%)	20.2 (Very Poor 143%)
RMS Distance	2.3 (Normal 116%)	4.4 (Very Poor 187%)	5.8 (Very Poor 149%)	14.2 (Very Poor 168%)
Range AP	55.3 (Very Poor 389%)	100.6 (Very Poor 525%)	30.5 (Normal 108%)	101.3 (Very Poor 148%)
Range ML	11.5 (Poor 125%)	22.0 (Very Poor 190%)	36.6 (Very Poor 194%)	78.3 (Very Poor 184%)
Mean Velocity	18.0 (Very Poor 234%)	81.7 (Very Poor 678%)	25.4 (Very Poor 174%)	88.5 (Very Poor 213%)
Mean Velocity	14.5 (Very Poor 262%)	77.4 (Very Poor 735%)	14.8 (Poor 135%)	67.4 (Very Poor 186%)
Mean Velocity	7.4 (Very Poor 214%)	16.5 (Very Poor 356%)	17.1 (Very Poor 248%)	43.9 (Very Poor 274%)
95% Ellipse Area	355.5 (Very Poor 324%)	1783.4 (Very Poor	692.3 (Very Poor 328%)	5365.7 (Very Poor
Sway Area	27.4 (Very Poor 314%)	225.9 (Very Poor	54.6 (Very Poor 329%)	543.1 (Very Poor
Mean Frequency	0.4 (Normal 107%)	0.7 (Very High 155%)	0.5 (Low 72%)	0.7 (Very Low 44%)
Mean Frequency	0.4 (Normal 102%)	0.7 (Very High 153%)	0.5 (Low 66%)	0.8 (Very Low 46%)
Mean Frequency	0.7 (High 138%)	0.9 (Very High 148%)	0.7 (Low 65%)	0.7 (Very Low 34%)
Adaptation/Fa-	36.9% (Fatigue)	7.8% (Fatigue)	27.6% (Fatigue)	12.9% (Fatigue)
Balance Age	81.2	106.8	66.7	79.6
Falls Risk	31.1%	80.5%	19.7%	29.4%

P2 Post treatment

Test	Eyes Open, Firm Plat-	Eyes Closed, Firm Plat-	Eyes Open, Soft Plat-	Eyes Closed, Soft Plat-
Date	4 May 2021 – 10:58 am	4 May 2021 – 10:59 am	4 May 2021 – 11:01 am	4 May 2021 – 11:01 am
Mean Distance	4.1 (Poor 124%)	4.3 (Normal 106%)	4.3 (Good 67%)	11.9 (Normal 83%)
Mean Distance	2.0 (Good 79%)	3.9 (Normal 118%)	2.9 (Very Good 59%)	9.4 (Normal 81%)
Mean Distance	3.1 (Very Poor 196%)	1.3 (Good 75%)	2.4 (Good 78%)	6.1 (Normal 99%)
RMS Distance	4.5 (Normal 120%)	5.4 (Normal 115%)	5.1 (Good 69%)	13.8 (Normal 85%)
RMS Distance AP	2.5 (Normal 83%)	5.1 (Poor 129%)	4.1 (Good 68%)	11.7 (Normal 82%)
RMS Distance	3.7 (Very Poor 188%)	1.7 (Good 75%)	3.0 (Good 78%)	7.4 (Normal 88%)
Range AP	13.5 (Normal 95%)	31.4 (Very Poor 164%)	29.3 (Normal 104%)	55.7 (Normal 81%)
Range ML	17.5 (Very Poor 190%)	11.1 (Normal 96%)	15.1 (Good 80%)	37.9 (Normal 89%)
Mean Velocity	9.9 (Poor 128%)	17.1 (Very Poor 142%)	14.4 (Normal 99%)	40.9 (Normal 99%)
Mean Velocity	7.4 (Poor 133%)	15.2 (Very Poor 144%)	12.4 (Normal 113%)	35.3 (Normal 97%)
Mean Velocity	5.0 (Very Poor 144%)	5.2 (Normal 111%)	5.4 (Good 78%)	14.8 (Normal 92%)
95% Ellipse Area	169.6 (Very Poor 154%)	166.4 (Normal 97%)	226.7 (Normal 107%)	1488.9 (Very Poor
Sway Area	13.5 (Very Poor 155%)	16.7 (Normal 96%)	17.7 (Normal 106%)	142.5 (Very Poor 286%)
Mean Frequency	0.4 (Normal 96%)	0.6 (Very High 143%)	0.5 (Low 71%)	0.5 (Very Low 36%)
Mean Frequency	0.6 (Very High 158%)	0.7 (Very High 142%)	0.7 (Normal 95%)	0.7 (Very Low 40%)
Mean Frequency	0.3 (Very Low 55%)	0.7 (Normal 118%)	0.4 (Very Low 38%)	0.4 (Very Low 21%)
Adaptation/Fa-	15.5% (Adaptation)	55.2% (Fatigue)	23.4% (Fatigue)	6.2% (Adaptation)
Balance Age	63.4	47.8	29.3	37.7
Falls Risk	18.2%	13.8%	12.4%	12.8%

Diagnostic Assessment

Overview of Assessment Battery

Both patients underwent comprehensive assessment structured across the objective domains: autonomic, saccadic, postural stability, cervical, neurological and physical examination with oculomotor assessment for convergence insufficiency (P2 only). The assessment battery reflects the multi-domain presentation of PPCS, encompassing vestibular, oculomotor, cognitive, autonomic, cervicogenic, and postural systems, (5, 7) and the recognised need for objective rather than purely symptom-based diagnostic approaches to this population given the absence of a consensus diagnostic standard. (21)

Saccadometry

Saccadometry was performed in darkened conditions, consistent with published protocols. (9) The overlap (OVL) paradigm was assessed. Parameters recorded included bilateral latency (ms),

amplitude (degrees), peak velocity (deg/s), and main sequence ratio (Q value) at each session. Latency distribution profiles and phase plots were generated for each paradigm to characterise trajectory variability.

Pre-treatment saccadometry for P1 demonstrated elevated latency with bilateral asymmetry and high inter-trial trajectory variability, consistent with frontal-cerebellar circuit disruption. The latency was elevated relative to published normative data8 with wide latency scatter consistent with poor control of saccade initiation, a pattern that has been shown to correlate with corpus callosum white matter diffusivity, reduced executive function performance, and PPCS symptom burden. (9)

Post-treatment saccadometry demonstrated improvement from baseline. Pre-treatment saccadometry for P2 demonstrated elevated latency, reduced peak velocity, and disordered trajectory profiles consistent with the oculomotor circuit disruption characteristic of chronic PPCS.8,10 Post-treatment saccadometry demonstrated objective improvement.

Force Plate Posturography

Instrumented force plate posturography was used to quantify postural stability through measurement of centre-of-pressure displacement during standardised standing tasks. Force plate-based posturography has been shown to detect ongoing neuromotor control impairment in PPCS populations (12) and to identify deficits not apparent on clinical balance tests alone, with instrumented measures sensitive to postural abnormalities persisting well beyond symptomatic resolution in both acute and chronic mTBI. (11)

Pre-treatment posturography for both P1 and P2 demonstrated impaired postural stability. Post-treatment posturography demonstrated objective improvement from baseline in both patients.

Oculomotor Assessment

Both patients underwent assessment of Near Point Convergence (NPC). Convergence insufficiency was diagnosed on the basis of a receded NPC, consistent with the diagnostic criteria used in published PPCS convergence insufficiency studies. (18) The prevalence of symptomatic convergence insufficiency in adults with PPCS has been reported at 20.4%18 and at 23-42% in military populations, (19) placing the finding within an expected range for this diagnostic context.

Physical and Neurological Examination

Both patients underwent physical examination including cervical range of motion assessment, segmental motion palpation, upper and lower limb neurological examination, and cranial nerve screening.

Both patients demonstrated cervical segmental restriction and dysfunction consistent with cervicogenic contribution to headache and dizziness. Neurological examination was otherwise consistent with multi-domain PPCS without focal lateralising deficit.

Diagnostic Challenges

Several diagnostic challenges were present in both cases. There is currently no global consensus on the objective measures that establish a definitive diagnosis of PPCS in any age group, (21) and the diagnosis remains clinical rather than biomarker-confirmed. The objective assessment tools used in this series, quantitative saccadometry, and force plate posturography, are not universally available in primary or secondary care settings, creating inequity in access to this level of diagnostic evaluation for PPCS patients.

Second, both patients presented beyond twelve months post-injury, at which point the neurological deficit profile reflects the compound effects of the initial injury, maladaptive neuroplasticity during the intervening period, physical deconditioning, and possible learned postural and behavioural compensatory strategies. Disentangling these contributions from the primary injury deficit was not possible from assessment data alone and was not required for treatment planning.

Third, neuro-imaging was not performed at the time of assessment. Both patients had presented beyond the acute phase at which structural neuro-imaging was performed, clinical assessment did not reveal red flags warranting urgent imaging. The absence of neuro-imaging is a recognised limitation of clinical case series in this population and precludes characterisation of any structural correlates of the objective neurophysiological findings.

Diagnosis

Both patients were diagnosed with persisting post-concussive syndrome (PPCS) on the basis of a documented history of mTBI, symptom duration exceeding three months, and the presence of three or more persistent post-concussive symptoms across multiple domains at the time of presentation. The diagnosis is consistent with the 2023 American Congress of Rehabilitation Medicine diagnostic criteria for concussion and mTBI, (21) which define mTBI as a traumatically induced physiological disruption of brain function with Glasgow Coma Score 13-15, loss of consciousness under 30 minutes where present, and post-traumatic amnesia under 24 hours.

The multi-domain objective abnormalities documented across the assessment battery, saccadometric frontal-cerebellar disruption (both), postural instability (both), and convergence insufficiency (both), were interpreted as consistent with the distributed neurophysiological consequences of chronic mTBI affecting brainstem, cerebellar, oculomotor, and autonomic pathways, and as representing the objective substrate of the presenting symptom profile.

Alternative diagnoses considered and excluded included: primary vestibular disorder (no unilateral peripheral pattern on examination, no BPPV features, no Menière's disease features); functional neurological disorder (objective findings on quantitative instrumented assessment inconsistent with a purely functional aetiology); primary mood disorder driving somatic amplification (possible contributory factor not formally excluded but not the primary diagnosis given the objective neurophysiological profile); and cervicogenic syndrome as the primary diagnosis (cervicogenic pathology was identified in both patients and managed as a contributing factor, but the distributed multi-domain objective findings were not consistent with cervicogenic aetiology as the sole explanation).

Prognosis

At the time of first assessment, the prognosis for each patient was guarded given the chronicity of their presentations, the multi-domain objective neurophysiological deficit profiles, and the documented difficulty of achieving recovery in PPCS beyond twelve months. (6, 7) The absence of a prior structured multimodal rehabilitation programme for either patient was noted as a prognostically relevant factor, as both had exhausted standard management without benefit prior to referral.

For P1, the prognosis for functional improvement was considered reasonable given the absence of neurological red flags, the absence of a prior structured rehabilitation attempt targeting the documented objective deficit profile, and the patient's motivation to return to his trade. The saccadometric dysmetria was a significant finding but was interpreted as reflecting a potentially reversible adaptive state rather than irreversible structural damage, on the basis of the absence of a focal deficit.

For P2, the prognosis was also considered reasonable for functional improvement, though complicated by the orthostatic tachycardia, which required specific rehabilitation addressing the autonomic system, (19, 20) and the cervicogenic component requiring physical management. (15, 16)

Both patients were counselled that the rehabilitation program would be individualised, protracted, and progressively dosed, and that the primary target was functional recovery and occupational reintegration rather than normalisation of all objective neurophysiological parameters.

Therapeutic Intervention

Shared Intervention Principles

Both patients received an individualised, assessment-driven, multimodal neurorehabilitation program. Interventions were selected and dosed on the basis of each patient's specific objective deficit profile as identified at baseline assessment, consistent with the approach described by Ross et al (10) in which neuroplasticity and sensory entrainment created by rigorous, diagnostic- and problem-focused, structured neurorehabilitation are proposed as the mechanisms of efficacy in chronic PPCS.

The principle of progressive loading, beginning at a level within the patient's tolerance and advancing difficulty systematically with demonstrated capacity, was applied across all components. No adverse events were reported during either patient's treatment course.

Patient 1: Clinic-Based interventions

Vestibular stimulation via Orbital multi-axis rotational chair. The Orbital device delivers controlled rotation in sagittal roll, yaw, and pitch planes, providing vestibular stimulation patterns not reproducible through any natural daily activity. Multi-axis rotational therapy in the PPCS population has been associated with significant reductions in symptom severity, enhanced balance improvement, and faster recovery trajectories when added to standard neurorehabilitation protocols.

Saccade and oculomotor training using a head-mounted weighted helmet with laser targeting computer-generated randomly selected LARP and RALP diagonal plane target panels. The weighted helmet increases the proprioceptive and motor demand of head movement, while the laser-to-target task requires active VOR engagement with immediate visual feedback on accuracy, a prerequisite for cerebellar VOR adaptation. (22, 23, 24, 25, 26)

Sensorimotor integration as backwards treadmill walking with weighted ankles (0.5 kg per ankle) and concurrent juggling for five minutes per session. This dual-task paradigm diverts attentional resources from conscious postural control, training automatic vestibular-postural responses while the visual system is engaged in the juggling task. The backwards direction specifically challenges the vestibular system by removing the predictive visual flow available during forward locomotion.

Lighted target panel tapping using random illuminated targets presented on a panel requiring rapid manual responses, beginning at slow presentation rates and progressively increasing in speed with demonstrated performance capacity. This trains upper limb reaction time, visuomotor processing speed, and hand-eye coordination.

Rhythmic entrainment therapy using audiovisual feedback signal synchronised with bilateral hand clapping on cue. Entrainment-based timing training, consistent in principle with the Interactive Metronome paradigm, has been shown in a randomised controlled pilot trial to significantly improve attention, immediate memory, and delayed memory in patients with persistent cognitive complaints following TBI when added to standard rehabilitation care, with effect sizes in the medium-to-large range. (27)

Spinal adjustments targeting identified cervical segmental dysfunction. Spinal manipulative therapy for cervicogenic headache has been supported by systematic review and meta-analysis with significant moderate-to-large short-term effects for headache frequency and intensity. (16)

Home program

One leg teeth balance exercise: a standing single-leg balance task while teeth cleaning to engage the trigemino-cerebellar proprioceptive pathway.

Diaphragmatic breathing in a five-second rhythm for one minute, three times per day, slow respiratory rate breathing to modulate autonomic tone and reduce sympathetic hyperactivation consistent with the post-concussive autonomic dysregulation. (28) This was documented with pupillometry (29, 30) to assist with patient compliance.

Vergence card: home vergence rehabilitation targeting the oculomotor convergence-divergence system.

iPad App saccade and pursuit training: horizontal anti-saccade training and diagonal pursuit exercises (up-right and down-left) for three minutes twice daily.

Breath-pause exercise: sudden full exhalation followed by maximal apnoea, progressively building toward one-minute sustained breath pause. This exercise modulates CO_2/O_2 balance and trains respiratory and autonomic regulation.

Head-turn walking: rapid side-to-side yaw head movements while walking, within symptom tolerance, providing a low-level continuous horizontal VOR stimulus during functional activity. (31)

Administration of therapeutic intervention

P1 completed 45 treatment sessions over 16 months. Clinic sessions were structured to deliver the full intervention battery with progressive task difficulty. The treatment table for P1 is presented in the Timeline section.

Changes in therapeutic intervention with rationale

Treatment intensity and task difficulty were progressively advanced throughout the 16-month course as P1 demonstrated improved performance capacity. The primary adaptive change across the treatment period was systematic progression of all tasks toward greater speed, complexity, and cognitive-motor demand, in line with the principle of progressive neurological loading. No abrupt changes in treatment type or direction were required. The 16-month duration reflects the protracted nature of recovery in chronic PPCS beyond twelve months post-injury and the extended timeline required to produce cumulative neuroplastic change in the context of the documented objective deficit profile.

Patient 2: Clinic-Based interventions

Vestibular stimulation via Orbital multi-axis rotational chair, as described for P1, with axis selection and dosing determined by P2's specific vestibular deficit profile on assessment. Beginning with Yaw and progressing to Yaw with Roll, P2 progressively tolerated the stimulation without adverse effects.

Rhythmic entrainment therapy: bilateral hand clapping synchronised to a 54 Hz audiovisual beat. The 54 Hz frequency corresponds to the Interactive Metronome standard beat rate. Rhythmic entrainment therapy at this frequency trains sensorimotor synchronisation, neural timing, and frontal-cerebellar timing circuits implicated in cognitive processing speed, deficits consistently documented in PPCS. A randomised controlled pilot trial found that Interactive Metronome therapy added to standard rehabilitation care significantly improved attention, immediate memory, and delayed memory in soldiers with persistent cognitive complaints following blast-related TBI, with neuroplastic changes accounting for large proportions of the variance in neuropsychological outcomes. (27)

Sensorimotor Integration Reaction Timer panel training with the 'gap scenario'. The gap paradigm presents a temporal gap (400ms) between target offset and the next stimulus, reducing fixation-related suppression of the superior colliculus and producing shorter saccadic latencies. This specifically targets the saccadic latency elevation documented on saccadometry, addressing the neural timing deficit at the subcortical level.

Head-mounted laser: tracing a wall map and navigating a maze at varying distances. This task requires sustained active head-eye coordination across spatial targets, engaging the VOR across the full range of head movement and demanding convergence and divergence across changing target distances, directly addressing P2's convergence insufficiency in a functional movement context.

Diagonal saccade and pursuit exercises: saccades and smooth pursuits performed diagonally across a wall target at arm's length. Diagonal plane exercises engage the LARP and RALP canal and oculomotor planes simultaneously, combining vestibular and vergence rehabilitation demands.

Repetitive Peripheral Somatosensory Evoked Potential (RPSEP) stimulation of the Trigeminal nerve (CN V) using a peripheral nerve stimulator applied at four locations along the Trigeminal distribution for one minute each. Trigeminal nerve stimulation produces somatosensory evoked potentials with cortical projections to the primary somatosensory cortex via brainstem relay, (32) and Trigeminal SEP provides an objective test of Trigeminal nerve function and its central connections through the brain-stem. (33) The Trigeminal nuclei in the pons are not anatomically contiguous with the vestibular nuclei but have direct functional interactions relevant to vestibular, autonomic, and oculomotor integration.

Repetitive stimulation at peripheral Trigeminal locations was applied to drive afferent brainstem input through these interconnected circuits as a component of the broader neurorehabilitation strategy.

Spinal adjustments targeting identified cervical segmental dysfunction, as described for P1.

Home program

Cervical stretching exercises: addressing identified range of motion restriction and muscle tension.

X2 VOR exercise performed slowly: active head movement while maintaining visual fixation on a moving target was performed at reduced speed consistent with P2's tolerance, targeting gaze stabilisation through VOR adaptation.

One leg teeth balance exercise: as described for P1.

Isometric exercises five minutes daily to promote Myokine production while maintaining strength and joint loading without dynamic movement stress, appropriate for a patient with exercise intolerance in the early treatment phase.

Vergence card: home vergence rehabilitation for the convergence insufficiency directly identified on assessment.

Infinity Thumb exercise: looking firstly to the horizon then the tip of the outstretched thumb, followed by bringing the thumb to 6cm in front of the eyes and reversing, this trains convergence and divergence across the full range of binocular gaze, specifically addressing P2's convergence deficit.

Deep knee bends (squats) 25 repetitions three times daily: lower limb strengthening and repeated postural loading providing an orthostatic and proprioceptive training stimulus.

Administration of therapeutic intervention

P2 completed 42 treatment sessions over 10 months. The treatment table for P2 is presented in the Timeline section.

Changes in therapeutic intervention with rationale

As P2's convergence insufficiency improved with directed oculomotor rehabilitation, the visual demands of the laser map and maze exercise were progressively increased to maintain challenge at the convergence and divergence system. The reaction timer panel speed was systematically advanced with demonstrated improvement in response latency and accuracy. The vestibular

rotation components were adjusted across sessions in response to P2's tolerance and symptomatic response. The vocational reorientation that occurred during the latter portion of treatment, P2 undertaking online distillery manufacturing courses, was not a treatment modification but an adaptive patient-led functional outcome that emerged from improving capacity during treatment, and was supported as a purposeful and motivationally significant recovery milestone.

The 10-month treatment duration reflects P2's rate of clinical progression within the constraints of his functional tolerance and the complexity of his multi-domain deficit profile.

Follow-up and outcomes

Patient 1

Clinician-assessed outcomes were documented through serial objective re-assessment across the treatment period. Progressive improvement was noted throughout the 16 months of treatment across all assessed domains.

Saccadometry: post-treatment data demonstrated improvement from baseline in latency, bilateral latency symmetry, and trajectory profile consistency in the overlap paradigm. Quantitative pre- and post-treatment saccadometric data are presented.

Force plate posturography: assessment demonstrated improved postural stability from the pre-treatment baseline. Pre- and post-treatment data are presented.

Patient-assessed outcome: at the completion of treatment, P1 reported feeling markedly recovered. This subjective assessment of recovery was consistent with the objective neurophysiological findings and directly preceded P1's decision to seek re-employment in his trade. P1 subsequently returned to full-time employment as a qualified electrician. At the time of preparing this manuscript, P1 provided verbal confirmation that no exacerbations or recurrence of the initial signs or symptoms have occurred since resuming full-time work.

The post-treatment assessments described above constitute the follow-up diagnostic data for P1. No further formal follow-up assessment was scheduled beyond the treatment completion assessment. Ongoing monitoring was not conducted through additional neurophysiological testing at the time of manuscript preparation, as P1 reported full functional recovery.

P1 completed 45 sessions across a 16-month treatment period, indicating satisfactory adherence to the clinic-based program across an extended course. The sagittal roll component of the Orbital rotational chair, described as an atypical movement not reproducible in daily activity, was tolerated without vestibular exacerbation across the treatment period. Home program adherence was not formally measured by objective means; P1's progressive improvement on all clinical and objective measures across sessions is consistent with adequate engagement with home exercise components between clinic visits. No tolerability concerns were documented during the treatment period.

No adverse events attributable to the treatment program were reported or documented for P1. No unanticipated events affected the treatment course. The absence of symptom exacerbation beyond the typical post-session fatigue expected in neurological rehabilitation is consistent with the progressive loading approach applied across all interventions.

Patient 2

Saccadometry: post-treatment assessment demonstrated improvement from the pre-treatment baseline across latency, peak velocity, and trajectory profile metrics in the overlap paradigms. Post-treatment data are presented in Figures 1 & 3.

Force plate posturography: post-treatment force plate assessment demonstrated improved postural stability from the pre-treatment baseline. Post-treatment data are presented in Figure 2.

Convergence: clinical convergence assessment at the completion of treatment demonstrated improvement in near point convergence (NPC=6cms) from the presenting deficit.

Patient-assessed outcome: P2 reported a remarkable improvement in his ability to progress through life across the 10-month treatment period. Towards the treatment completion P2 was feeling well enough to undertake online courses and to pursue vocational reorientation. He was not entirely pain-free at treatment completion and acknowledged the potential benefit of further treatment; however, he reported sufficient functional capacity to sustain his new vocational activities independently.

The unanticipated functional endpoint of P2's rehabilitation, transition from spray painting to founding, operating, and staffing a distillery manufacturing business, was not the originally intended outcome of treatment but represents a remarkable and clinically meaningful vocational reintegration in a patient who entered treatment unable to resume his prior occupation and unable to engage in any employment or retraining.

The post-treatment assessments described above constitute the follow-up diagnostic data for P2. No further neurophysiological follow-up assessment was undertaken at the time of manuscript preparation. P2 remained engaged in his new vocational activities without scheduled clinical attendance, though he acknowledged the potential value of further treatment.

P2 completed 42 sessions across a 10-month treatment period, indicating satisfactory adherence across a protracted outpatient course. The vestibular rotation, RPSEP stimulation, sensorimotor integration components including spinal manipulation, were tolerated across the treatment period without documented intolerance. Home program adherence was not formally measured by objective means.

Progressive improvement of objective measures across sessions is consistent with sufficient engagement with home exercises to support the clinical gains documented. No significant tolerability concerns were documented during the treatment period.

No adverse events attributable to the treatment program were reported or documented for P2.

Discussion

Strengths

The primary strength of this case series is the use of serial objective neurophysiological assessment to document treatment response in parallel with the primary functional outcome of interest, occupational reintegration. The combination of quantitative saccadometry, and instrumented force plate posturography across the treatment period provides a multi-domain objective evidence base that is rarely reported alongside vocational outcomes in the PPCS literature.

Most published PPCS case series and retrospective studies rely on subjective symptom scales as primary outcome measures; the objective neurophysiological data presented here address a recognised gap in the evidence base. (5, 7)

Both patients represent a chronicity of presentation, PPCS exceeding twelve months, that is associated with poor prognosis in the broader literature and that is underrepresented in rehabilitation trial populations. (6, 7) The occupational outcomes are clinically meaningful beyond the neurophysiological data: P1's full return to a safety-critical skilled trade and P2's novel vocational self-employment represent outcomes that carry direct economic, psychological, and social significance for the patients and their communities.

A further strength is the explicit documentation of every intervention modality, its dosage, and its progression rationale, in compliance with CARE reporting standards, (34) which allows readers and future researchers to evaluate and potentially replicate the treatment approach.

Limitations

The absence of a control condition is the primary methodological limitation inherent to case series design. It is not possible to determine from two cases whether the observed improvements reflect the specific treatment program, the passage of time, regression to the mean, placebo response, or some combination of these. The CARE guidelines acknowledge this limitation as intrinsic to case report methodology (34) and the cases are presented as clinical observations rather than as evidence of treatment efficacy.

Neuro-imaging was not performed at either presentation. The absence of MRI data means that the structural correlates of the objective neurophysiological findings, including any white matter abnormalities consistent with diffuse axonal injury, or focal lesions that might explain the asymmetric components of the saccadometric data, cannot be characterised. This limits the mechanistic interpretation of the findings.

Formal standardised symptom questionnaires, such as the Rivermead Post Concussion Symptoms Questionnaire or the Post-Concussion Symptom Scale, were not administered. The absence of validated symptom severity scoring precludes direct comparison of symptom trajectories with published normative data and with outcomes reported in other PPCS rehabilitation studies using these instruments.

The contribution of individual treatment components to the overall outcome cannot be determined from the data available. Both patients received multimodal programs in which all interventions were delivered concurrently and progressively. The specific therapeutic mechanism responsible for the measured improvements cannot be isolated.

Home program adherence was not objectively monitored. The assumption that home exercises contributed to outcomes rests on clinical inference rather than documented compliance.

Finally, the asymmetry in available data across the two patients means that the full objective assessment battery is not directly comparable between cases.

Discussion of relevant literature

PPCS in working-age adults and occupational outcomes

Persisting post-concussive symptoms affect between 24% and 43% of those who sustain a concussion (5) and constitute a significant source of occupational disability. Silverberg et al. (35) demonstrated that even among mTBI patients who achieve a formal return to work, a substantial

proportion experience ongoing productivity loss, with 60% reporting affected work productivity at six to eight months post-injury, and residual symptoms, depression, anxiety, and bodily pain continuing to impair work performance. This highlights that the boundary between 'returned to work' and 'fully recovered occupationally' is not clearly defined by RTW status alone, a distinction directly illustrated by the contrasting outcomes of P1 and P2 in the present series, where P1's full return to a skilled and physically demanding trade represents a more complete occupational recovery than the term 'RTW' alone would convey.

Post-concussive symptom burden is the strongest predictor of failure to return to work at six months post-mTBI. (36) In a prospective cohort study (UPFRONT, n=1,151), rates of complete return to work increased from 34% at two weeks to 77% at twelve months, but occupational factors influenced short-term RTW while symptom burden and psychological distress were the dominant predictors of long-term RTW.37 Both P1 and P2 in the present series presented with symptom burdens exceeding twelve months and failed to achieve RTW through prior standard management, placing them in the high-risk category described in the literature. Their subsequent successful occupational reintegration following individualised multimodal neurorehabilitation is therefore a clinically noteworthy departure from the expected trajectory.

The vocational reorientation of P2, from spray painter to distillery proprietor, raises a question not directly addressed in the existing RTW literature: whether novel vocational self-employment constitutes a categorically different form of recovery from returning to a prior occupation, and whether conventional RTW metrics capture its significance adequately. Brandt et al. (14) reported that most employees with PPCS returned to work but only a minority worked under similar conditions to pre-injury, with employees and managers alike struggling to assess workload accurately. P2's case suggests that for some patients with PPCS, vocational reorientation toward a different occupation better matched to the post-injury functional state may represent a more sustainable and ultimately more successful outcome than forced return to an occupation whose physical and visual demands may no longer be compatible with the residual deficit profile.

Multimodal neurorehabilitation in chronic PPCS

The evidence base for multimodal neurorehabilitation in chronic PPCS beyond twelve months post-injury is limited but encouraging. Ross et al. (7) reported significant pre-post improvements in both subjective symptom scales and objective measures of trail making, processing speed, reaction time, and visual acuity in a retrospective chart review of patients with PPCS at a mean of approximately two years post-injury, using an intensive five-day multimodal outpatient program. The authors concluded that late-stage PPCS patients may have substantive benefits from a patient-centred, diagnostic- and problem-focused neurorehabilitation approach, and specifically hypothesised that neuroplasticity and sensory entrainment created by rigorous structured neurorehabilitation are efficacious even when patients have clinically plateaued. (7) The present two cases are consistent with this hypothesis, with both patients demonstrating objective neurophysiological improvement and meaningful functional reintegration despite presentations exceeding twelve months post-injury.

The multi-domain objective deficit profiles of both patients, spanning autonomic dysregulation, oculomotor and saccadic circuit impairment, postural instability, and cervicogenic pathology, are consistent with the distributed neurophysiological disruption that characterises PPCS7 and explain why unimodal approaches had not produced functional recovery in either patient prior to referral.

The pathway of care described by Corwin et al. (13) emphasises that visual and vestibular rehabilitation in mTBI should be driven by individual assessment findings and delivered through coordinated multi-modal programs rather than through single-modality protocols, a principle reflected throughout both treatment courses described here.

Saccadometry as a biomarker

Saccadometric findings in both patients were consistent with published characterisations of mTBI-related oculomotor impairment. Heitger et al. (8) demonstrated that latency distribution measurement discriminates between concussed patients and healthy controls, and that saccadometric abnormalities persist well beyond symptomatic resolution in a proportion of PPCS patients. The present cases extend this observation by documenting saccadometric improvement following targeted multimodal rehabilitation, a finding with implications for the use of saccadometry not only as a diagnostic biomarker but also as a treatment response measure. Corwin et al. (9) demonstrated correlations between saccade performance and corpus callosum white matter diffusivity, Stroop performance, and symptom burden, providing a neuroanatomical substrate for the oculomotor deficits measured at baseline in both patients.

The persistence of some saccadometric abnormality at treatment completion in both patients, most notably trajectory scatter, is consistent with the known difficulty of fully normalising frontal-cerebellar circuit timing in established PPCS,7 and is interpreted as reflecting the structural rather than purely functional nature of the underlying circuit impairment.

Oculomotor rehabilitation

P1 and P2's presenting convergence insufficiency is consistent with the 20.4% prevalence of CI reported by Nordfalk et al. (18) in adults with PPCS and the 23–42% rates reported in military mTBI populations. (19) The CONCUSS clinical trial demonstrated measurable neuroplastic changes in the frontal eye fields, supplemental eye fields, parietal eye fields, cerebellar vermis, and visual cortex following office-based vergence and accommodative rehabilitation for concussion-related CI, (20) consistent with the plasticity of the vergence oculomotor system in the PPCS population and supporting the rationale for targeted vergence rehabilitation as incorporated in the treatment program.

Rhythmic entrainment and timing-based rehabilitation

The entrainment therapy component of both treatment programs, synchronised audiovisual hand clapping with the 54 Hz, is consistent in principle with the Interactive Metronome paradigm, which operates at the same 54-beat-per-minute frequency. Nelson et al. (27) reported in a randomised controlled pilot trial that Interactive Metronome therapy added to standard rehabilitation care for soldiers with persisting cognitive complaints following blast-related TBI produced significant improvements in attention, immediate memory, and delayed memory, with effect sizes in the medium-to-large range and indicators of neuroplastic change accounting for large proportions of the variance in neuropsychological outcomes. The cognitive gains observed in both P1 and P2 across the treatment period, including the cognitive capacity that enabled P2 to undertake online vocational retraining, are consistent with the cognitive rehabilitation mechanism proposed by Nelson et al. (27)

Vestibular rehabilitation and rotational therapy

Vestibular stimulation through the Orbital multi-axis rotational chair provided the primary peripheral vestibular rehabilitation component of both programs. The sagittal roll plane, described as an atypical movement not reproducible in activities of daily living, is consistent with the novel vestibular stimulation paradigms discussed by Sadeghi et al, (18) in the context of unidirectional rotational rebalancing of vestibular asymmetry. Multi-axis rotational therapy has been incorporated as a component of intensive multimodal PPCS programs in the published literature, (7) and audio biofeedback augmentation of vestibular rehabilitation therapy has been shown to produce larger improvements than standard vestibular rehabilitation alone in patients with chronic mTBI and persistent imbalance, with significant effects on both subjective symptom severity and objective sensory organisation testing composite scores. (39) It is the author's opinion that full body multi axis targeted vestibular stimulation is a driver of enhanced homeostasis. (40, 41, 42, 43)

Cervicogenic management

Cervicogenic pathology was identified and managed in both patients through spinal adjustments targeting documented segmental dysfunction. Nusholtz et al. (15) reported substantive improvement in post-concussive dizziness, headache, and related symptoms following chiropractic management of craniocervical junction dysfunction in patients with PPCS of at least six weeks duration. Bini et al. (16) confirmed in a systematic review and meta-analysis that spinal manipulative therapy produces significant short-term reductions in cervicogenic headache frequency and intensity. The cervicogenic components of both patients' presentations, headache and restricted range of motion, are likely to have contributed to their overall symptom burden and are appropriately included as targets within the multimodal framework.

Scientific rationale for conclusions

The neurophysiological improvements documented across both cases, and the functional and occupational outcomes that followed, are most parsimoniously explained by neuroplastic reorganisation of the sensory-cognitive-motor networks disrupted by the original mTBI, facilitated by the individualised multimodal rehabilitation program. Ross et al. (7) specifically hypothesised that neuroplasticity and sensory entrainment created by rigorous, diagnostic- and problem-focused structured neurorehabilitation are the mechanisms of efficacy in chronic PPCS, a hypothesis consistent with the findings of the present two cases.

The saccadometric improvements in both patients are consistent with progressive rehabilitation of the frontal-cerebellar circuits mediating response inhibition and spatial remapping. (8, 9) The persistence of some trajectory scatter at treatment completion in both patients is expected given the structural rather than purely functional nature of mTBI-related white matter disruption, and is consistent with the known ceiling of neuroplastic recovery in established PPCS.

The convergence deficit improvement is consistent with the neuroplastic changes in the vergence oculomotor network documented by Alvarez et al.[2]0 following targeted vergence rehabilitation, supporting a rehabilitation-driven rather than spontaneous mechanism for this component of recovery.

The occupational outcomes of both patients, full return to skilled trade employment for P1 and new vocational self-employment for P2, most likely reflect the aggregate functional recovery across all treated domains rather than improvement in any single domain, consistent with the distributed multidomain nature of the initial neurophysiological deficit profiles. The prior failure of both patients to achieve RTW through standard management argues against spontaneous recovery as the primary explanation for the occupational outcomes.

Conclusions

Primary take-away lessons

These two cases demonstrate that working-age adults with persisting post-concussive symptoms ex- tending beyond twelve months, a population for whom standard management has proven insufficient and whose prognosis for recovery is generally considered poor, can achieve clinically meaningful neurophysiological improvement and meaningful occupational reintegration following individualised, assessment-driven, multimodal neurorehabilitation.

The serial objective neurophysiological data from quantitative pupillometry, saccadometry, and force plate posturography provide documented evidence of measurable change across the treatment period, and the occupational outcomes demonstrate that rehabilitation impact extends to the domains of greatest practical significance for working-age patients.

The contrast between P2's prior inability to work and his subsequent ability to establish and staff a new commercial enterprise illustrates that in some cases of chronic PPCS the most meaningful vocational outcome is not a return to the prior occupation but an adaptive reorientation that better fits the post-recovery functional state, a nuance not well captured by standard return-to-work metrics.

The cases collectively support the clinical case for a comprehensive, objective assessment-guided, multimodal approach in chronic PPCS, and underscore the importance of treating occupational reintegration, in whatever form it takes, as a primary outcome measure rather than a secondary observation.

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Note:

Written informed consent from both patients for the publication of these case reports and the accompanying images is held by the author.

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Timeline

Patient 1

Time Point	Event — Findings — Interventions — Outcomes
Pre-injury	Employed full-time as a qualified electrician. No neurological, vestibular, or oculomotor history. Functionally independent.
Index event — October 2018	Sustained mTBI from a motor vehicle accident. Standard medical management commenced.
2 years post-injury	Failed to return to work following standard management. Persistent PPCS across cognitive, oculomotor, vestibular, autonomic, and postural domains at presentation.
Baseline assessment — November 2020	Saccadometry: elevated latency with bilateral asymmetry; high inter-trial trajectory variability. Force plate posturography: impaired postural stability. Cervical examination: segmental dysfunction identified. Orthostatic Tachycardia. Occupational status: unable to work as electrician.
Treatment commencement — November 2020	Individualised multimodal programme initiated: — Vestibular stimulation: Orbital multi-axis rotational chair (sagittal roll, yaw, pitch as indicated by assessment) — Saccade training: weighted helmet with head-mounted laser to computer-generated random LARP/RALP target panels — Sensorimotor integration: backwards treadmill walking with weighted ankles (0.5 kg each) and concurrent juggling — Random-target lighted panel tapping (speed progressed with demonstrated capacity) — Entrainment therapy: audiovisual feedback signal synchronised with bilateral hand clapping — Spinal adjustments to identified spinal segmental dysfunction
Home programme	One-leg teeth balance exercise; diaphragmatic breathing 5-second rhythm 1 min 3× daily; vergence card; iPad App horizontal anti-saccade and pursuit training 3 min 2× daily (up-right / down-left diagonal); breath-pause exercise progressing to 1-minute sustained apnoea; VOR stimulus — walking with rapid side-to-side head rotation within tolerance.
Months 1–16 (45 sessions total)	Progressive improvement documented across all domains throughout the 16-month treatment period. Treatment sessions delivered at patient-specific intensity with difficulty progressed in line with demonstrated capacity. Saccadometric trajectory variability and latency improved. Postural stability improved on force plate.
Post-treatment assessment	Saccadometry: improved latency symmetry and reduced trajectory variability from baseline. Force plate: improved postural stability scores from baseline.
Treatment completion — March 2022	P1 reported feeling markedly recovered. Decision taken to pursue return to work. Full-time employment resumed as a qualified electrician.
Follow-up (manuscript preparation) — March 2026	P1 provided verbal confirmation that no exacerbations or recurrence of initial signs or symptoms have occurred since returning to full-time employment as an electrician.

Timeline

Patient 2

Time Point	Event — Findings — Interventions — Outcomes
Pre-injury	Employed full-time as a spray painter. Occupation involving sustained close-range visual demand, postural loading, and exposure to physical environmental conditions. No neurological, vestibular, or oculomotor history.
Index event — September 2016	Sustained mTBI by falling from a child's scooter and hitting his head on concrete paving. Standard medical and allied health management initiated.
August 2020 post-injury	Failed to return to work following prior management. Persistent PPCS across vestibular, oculomotor, cervicogenic, cognitive, and postural domains at presentation. Convergence insufficiency identified as a direct vocational barrier. Referred to Brain Based Therapies Pty Ltd, Eden NSW.
Baseline assessment — August 2020	Convergence insufficiency; Orthostatic Tachycardia; Saccadometry: elevated latency, reduced peak velocity, disordered trajectory profiles. Force plate posturography: objective postural instability. Cervical examination: restricted range of motion, segmental dysfunction. Occupational status: Disability Pension.
Treatment commencement — August 2020	Individualised multimodal programme initiated: — Vestibular stimulation: Orbital multi-axis rotational chair — Entrainment therapy: bilateral hand clapping synchronised to 54 Hz audiovisual beat — Sensorimotor integration: Reaction Timer panel with 'gap scenario' — Head-mounted laser: tracing a wall map and navigating a maze at varying distances — Oculomotor exercises: diagonal saccades and pursuits at arm's length on wall target — Repetitive Peripheral Somatosensory Evoked Potential (RPSEP) stimulation of the Trigeminal nerve (CN V) at 4 locations, 1 minute per location, using a peripheral nerve stimulator — Spinal adjustments to identified cervical segmental dysfunction
Home programme	Cervical stretching exercises; zero-gain VOR exercise performed slowly; one-leg teeth balance exercise; isometric exercises 5 min daily; vergence card with instructions; Infinity-Thumb exercise targeting convergence deficit; deep knee bends (squats) 25 repetitions × 3 daily. Dietary and supplemental adjuncts discussed: ketogenic diet as neuroinflammatory mitigation, CoQ10, magnesium supplementation.
Months 1–10 (42 sessions total)	Remarkable functional improvement documented progressively across the 10-month treatment period. Oculomotor, vestibular, postural, and cervicogenic domains all improved. Headache frequency and cervicogenic symptoms reduced. P2 reported increasing capacity for sustained near-visual tasks and cognitive activity.
Vocational reorientation (late treatment phase)	P2 enrolled in online courses in distillery manufacturing. Commenced vocational reorientation from automotive spray painter to distillery manufacturer. Commenced production of alcoholic retail products for sale at markets and through a retail outlet. Employed staff.
Post-treatment assessment — June 2021	Saccadometry: improvement from baseline in latency, peak velocity, and trajectory profiles. Force plate: objective improvement in postural stability from baseline.
Treatment completion — June 2021	P2 reported sufficient functional capacity to sustain vocational activities without further scheduled treatment. Business operational: distillery manufacturing, retail market sales, retail outlet, employed staff.
Follow-up (manuscript preparation) — April 2026	P2 acknowledged residual symptoms and the potential benefit of further treatment; however, reported managing adequately to sustain his new vocational role and business activities independently and without scheduled clinical attendance.