

Functional Neurology Treatment of Traumatic Brain Injury and Post-Concussion Syndrome

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One of the most important things that the brain does is help you determine where you are in space.

The brain uses several different sensory systems to do this.

Proprioception: stretch receptors in muscles and joints tell the brain where the body parts are in relation to gravity

Vestibular System: receptors in the inner ear tell the brain where the head is in relation to gravity, and how it is moving

Visual system: the eyes tell the brain where the body is in relation to the world

All of these inputs need to work together in order for the brain to make sense of the body's location in relation to the environment.

These systems all need to integrate, and this initially takes place in the brainstem.

The forces involved in TBI result in shearing of the long thin fibers that allow these systems to communicate and work together.

In essence, the symptoms of TBI are the result of a failure of these systems to integrate.

TBI is not just about impairments of vision, or of vestibular sense and balance, or of feedback from the body's receptors.

TBI is about a failure of all of these systems to properly work together and inform the brain of where the body is in space.

The harder the brain has to work to localize itself in the environment, the faster it fatigues, and the more likely that higher brain functions will fail.

When these inputs create a sensory mismatch, the result is dizziness and vertigo.

This is one of the most common consequences of traumatic brain injury. 50% of TBI patients experience this at some point in their recovery.

In order for the visual system to map the world, gaze must be stable.

In order for the eyes to be able to give you accurate information about where the world is, they need to be able to hold still.

Almost all TBI patients have some form of gaze stability problem.

The systems that integrate on the parts of the brainstem that promote gaze stability come from the inner ear, the cerebellum, the basal ganglia, the frontal lobe, the parietal lobe...

Almost the entire brain is involved in gaze stability.

This means that fixing gaze stability is not about just eyes. It is about identifying and fixing the parts of the brain that are creating the problem.

It is about stimulating the deficient parts of the brain, and engaging them during gaze stabilization exercises.

When the eyes drift around a target more than 5 degrees, the result is dizziness and vertigo.

When the eyes drift less than this, the result is brain fog with visual tasks.

The first thing the brain does to try to stabilize gaze is engage upper neck muscles as reflexes.

This is most commonly the driving force behind persistent post-traumatic headaches.

The job of the functional neurologist is to evaluate the functionality of all relevant systems, proprioceptive, vestibular, and visual.

Most importantly, we evaluate how all of these systems **work together**, as all of these systems are directly influenced by the functionality of the others.

We find out what does not work, what still does, and design rehabilitation programs that get all of these systems working together in harmony again.

THE POTENTIAL IMPACT OF VARIOUS PHYSIOLOGICAL MECHANISMS ON OUTCOMES IN TBI, MTBI, CONCUSSION AND PPCS

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Michael Powell^{2,6}, Steven Geanopoulos⁷, Steven Noseworthy⁸,
Datis Kharrazian⁹, Chris Turnpaugh^{3,11}, Albert Comey^{1,12},
and Glen Zielinski^{2,3,13}*

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Vestibular impairment is a common consequence of mTBI.

As many as 65% of mTBI patients will experience some form of vestibular dysfunction during their recovery.

Evidence of central and peripheral vestibular dysfunction was identified at a higher frequency in symptomatic patients.

Abnormal nystagmus or oculomotor findings indicative of central vestibulopathy were present in 50% of symptomatic subjects.

Rotational chair testing found evidence of peripheral vestibulopathy in 25% of symptomatic patients, and central vestibular pathology in 17% of symptomatic subjects.

Computerized dynamic posturography (CDP) has been proposed as a means to monitor the status of vestibular function.

CDP testing assesses the patient's ability to maintain postural stability against a standardized series of balance and postural challenges.

Stability has been shown to decrease with mTBI, with shifts in stabilization strategies toward greater anterior-posterior and decreased medial-lateral control.

This raises the likelihood that concussed patients have a chronic risk of falls without appropriate neurorehabilitation.

Visual dysfunction has been shown to be extremely prevalent in the mTBI population.

The frequency of occurrence of oculomotor dysfunction in TBI, considering accommodation, version, vergence, strabismus, and cranial nerve palsy has been assessed.

The results indicate that oculomotor dysfunction is the norm in TBI.

The neural substrates of eye movements involve diffuse networks including the frontal lobes, parietal lobes, and cerebellar cortices.

These systems also serve within networks that facilitate executive functions and higher cognitive experience.

Impairments of eye movements have thus been demonstrated to have considerable clinical utility in both diagnosing and rehabilitating the mTBI patient.

TBI patients demonstrate aberrancies in predictive components of smooth pursuit eye movements.

These deficits in average target prediction, eye position error, and eye position variability positively correlated with executive function and attentional measures.

Impairment of predictive smooth pursuit eye movements thus may be a sensitive indicator of impaired attention processing.

Variability of gaze position errors during predictive smooth pursuit proved to be a sensitive measure that correlated with neurocognitive testing data and DTI findings.

The degree of variability correlated with the mTBI spectrum severity.

Performance variability during predictive visual tracking is a valid and useful indicator of damage to frontal white matter tracts, and is similarly indicative of impaired cognitive function.

Eye movement function has been shown to be impaired in post-concussion syndrome, with eye movement deficits found on measures relating to motor functions executed under both conscious and semi-conscious control, as well as on several eye movement functions that are beyond conscious control and indicative of sub-cortical brain function.

The literature demonstrates specific deficits in antisaccade performance, a task that in non-mTBI individuals that requires attentional focus, inhibitory control, working memory, and the ability to generate voluntary goal-directed behavior.

Computerized eye movement testing should be utilized with post-concussion syndrome patients to evaluate for incomplete recovery of function.

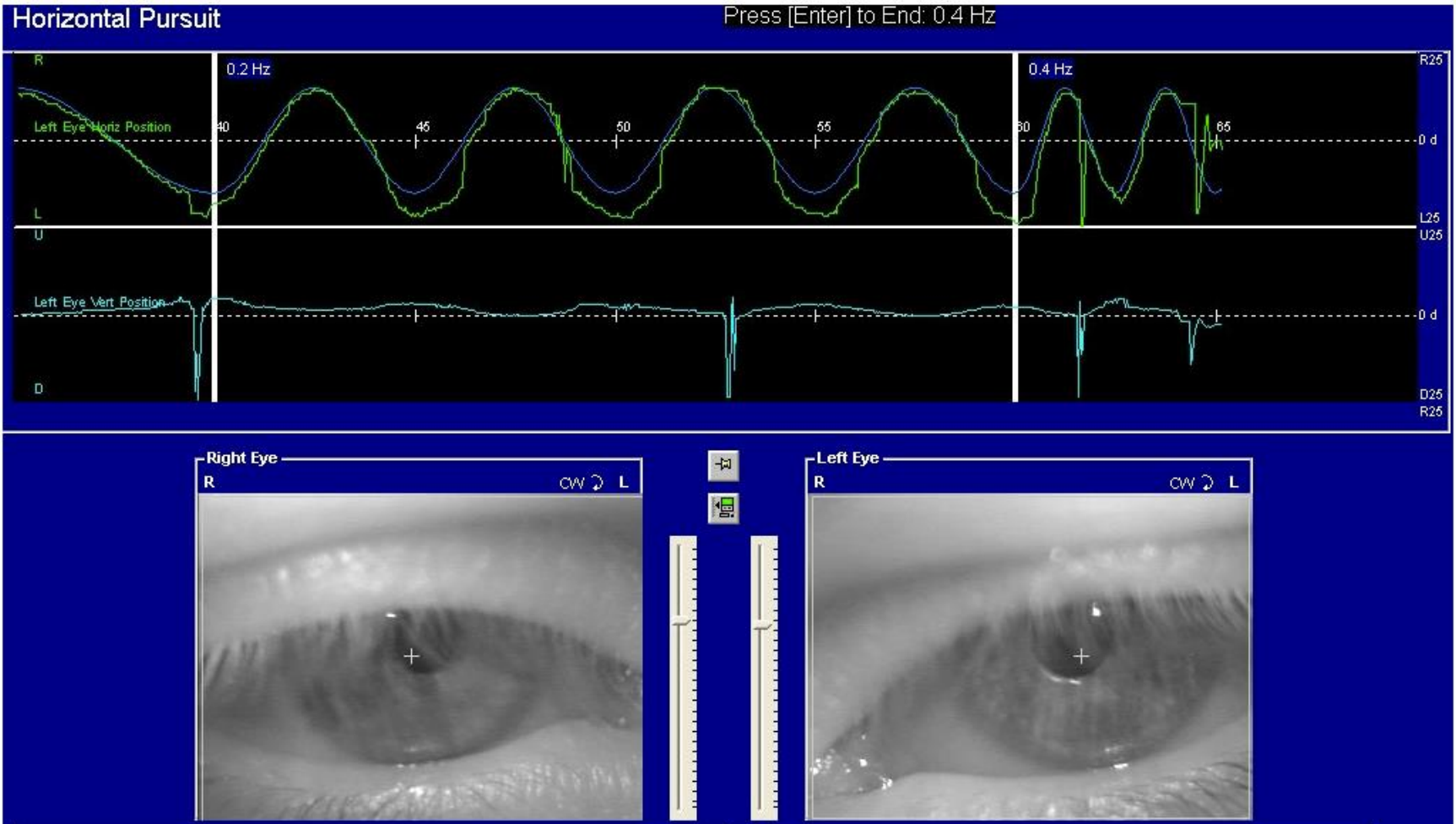
Rehabilitation of eye movement dysfunction has been shown to be effective in mTBI patients.

90% rate of improvement from oculomotor rehabilitation, with reduction in both symptoms and objective findings that persisted at a 2- to 3-month follow-up,

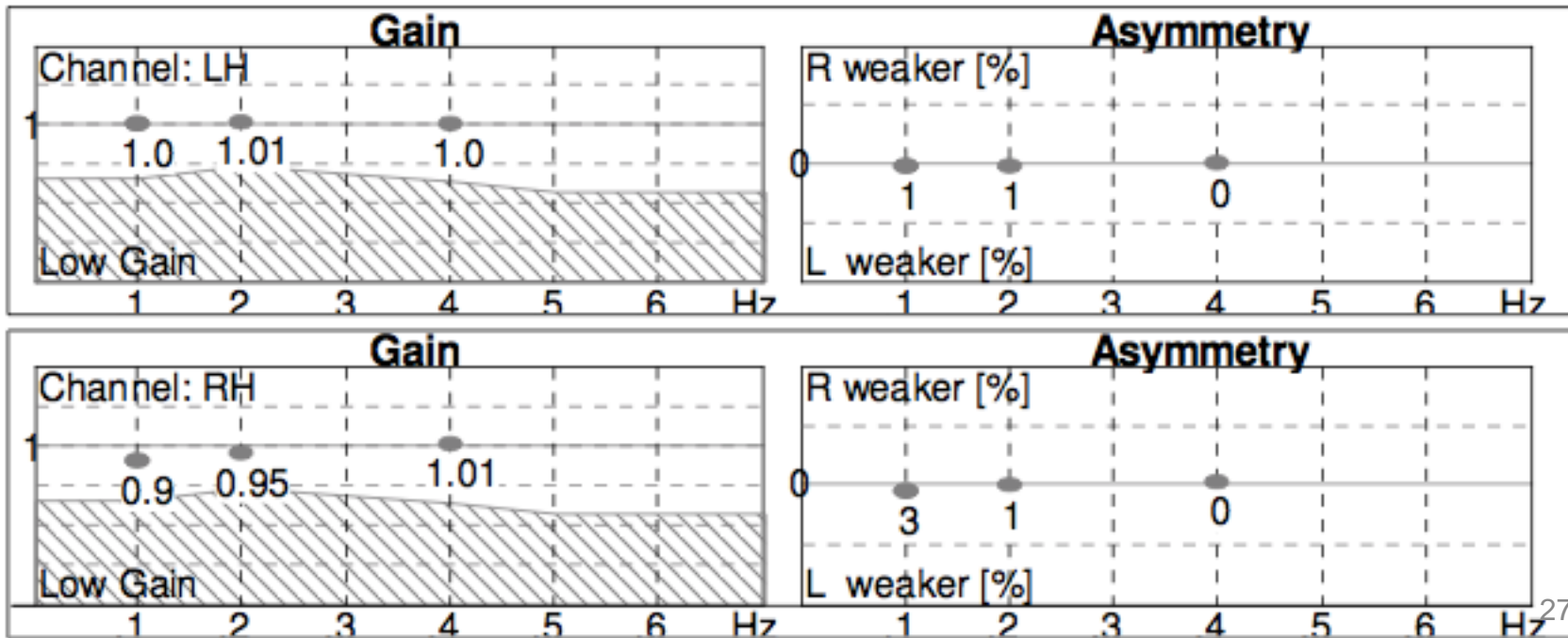
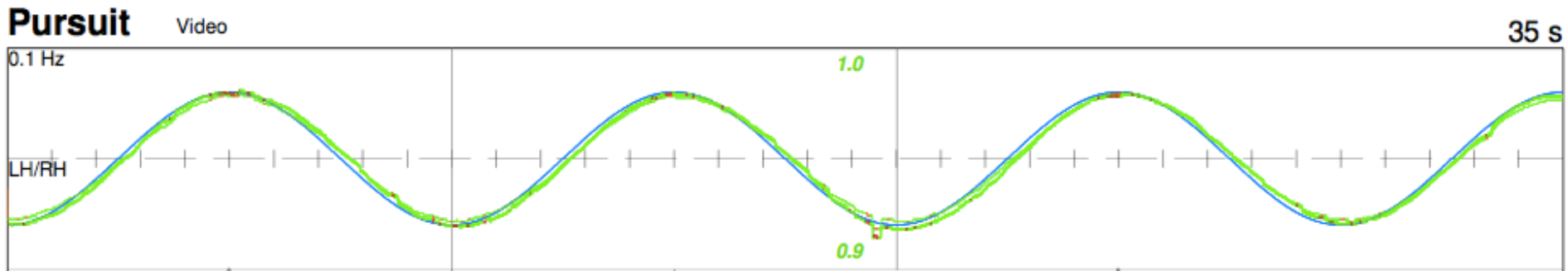
demonstrating both the efficacy of optometric rehabilitation and significant neural plasticity in mTBI patients.

Given the interaction between the visual and vestibular systems, the frequency of impairment noted in these systems, and their potential influence on recovery, thorough and sensitive evaluation of these systems is a prerequisite for effective management of mTBI.

Video Nystagmography



Video Nystagmography

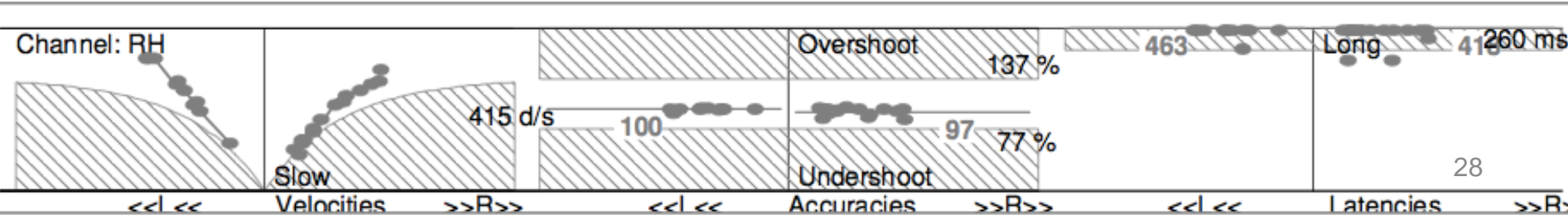
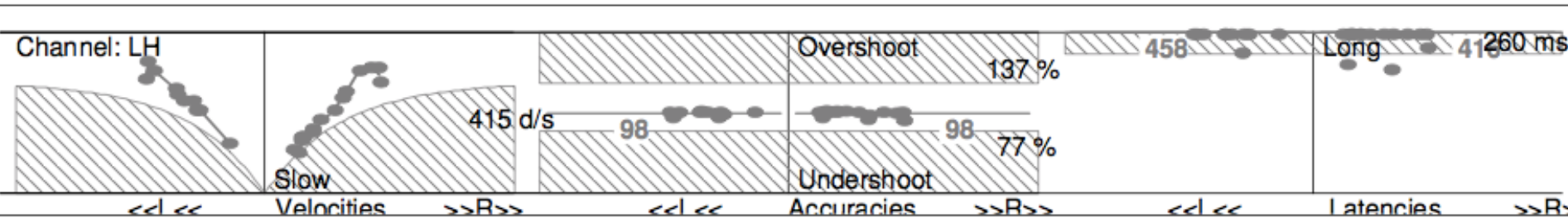
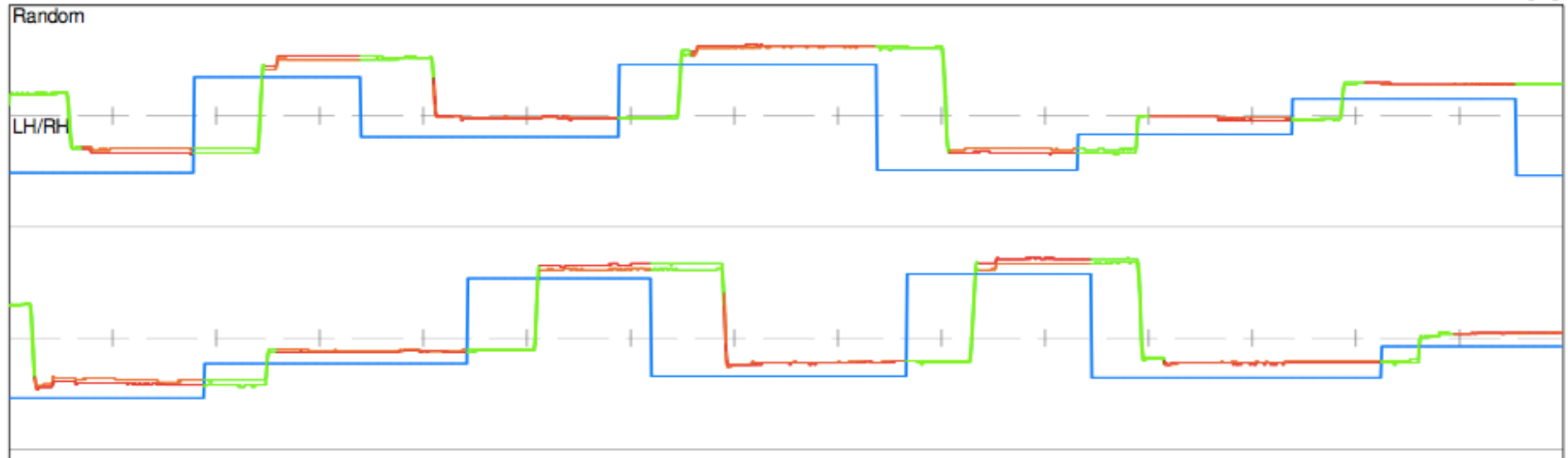


Video Nystagmography

Saccade-Random

Video

15 s

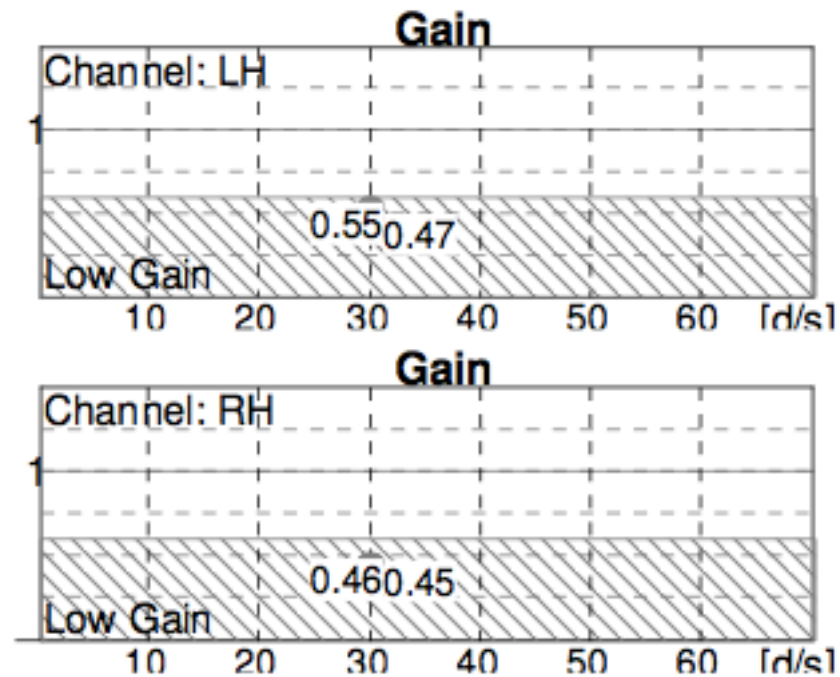
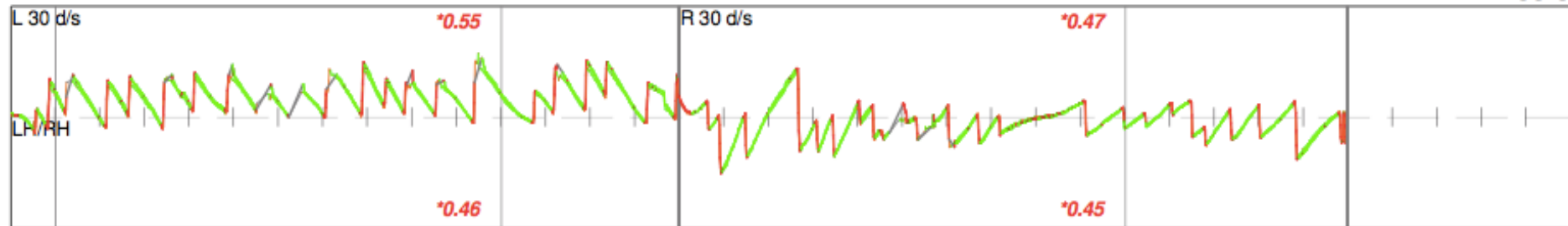


Video Nystagmography

OPK-Fixed

Video

35 s

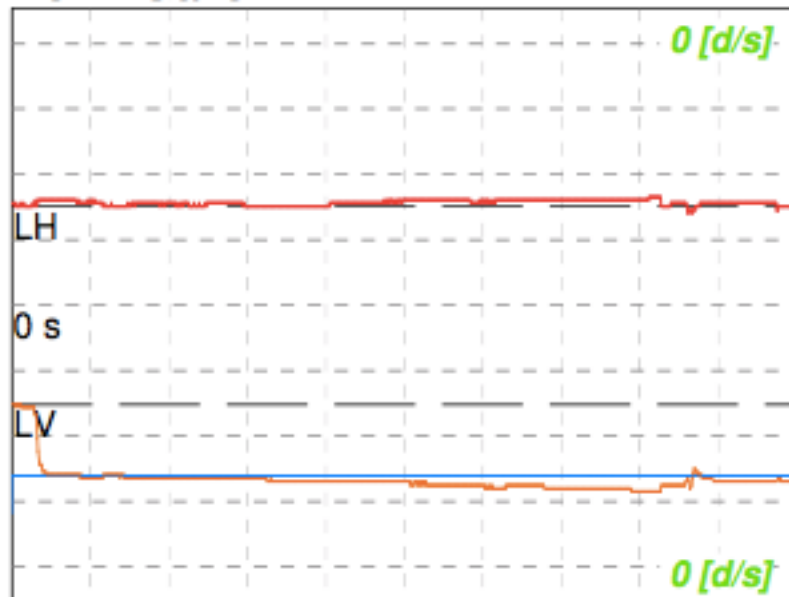


Video Nystagmography

Gaze-Vertical

Video

Down Gaze



Up Gaze

10 s

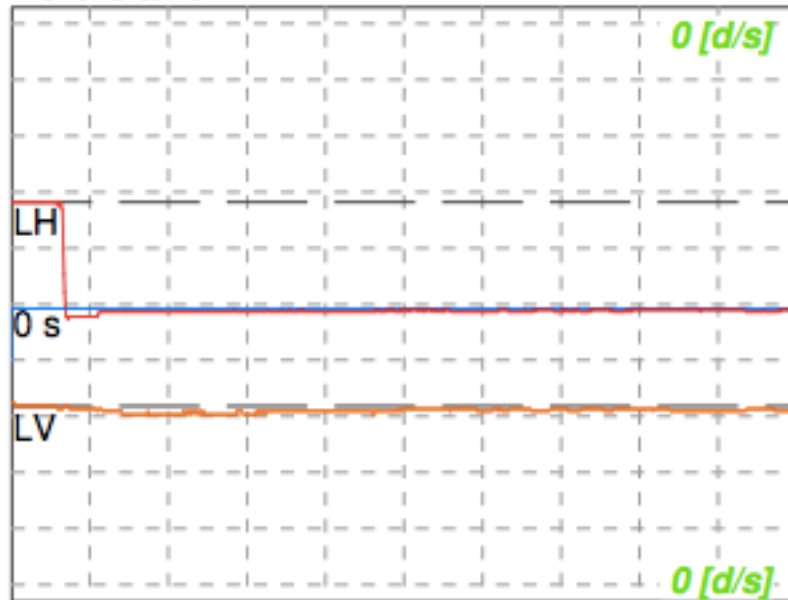


Video Nystagmography

Gaze-Horizontal

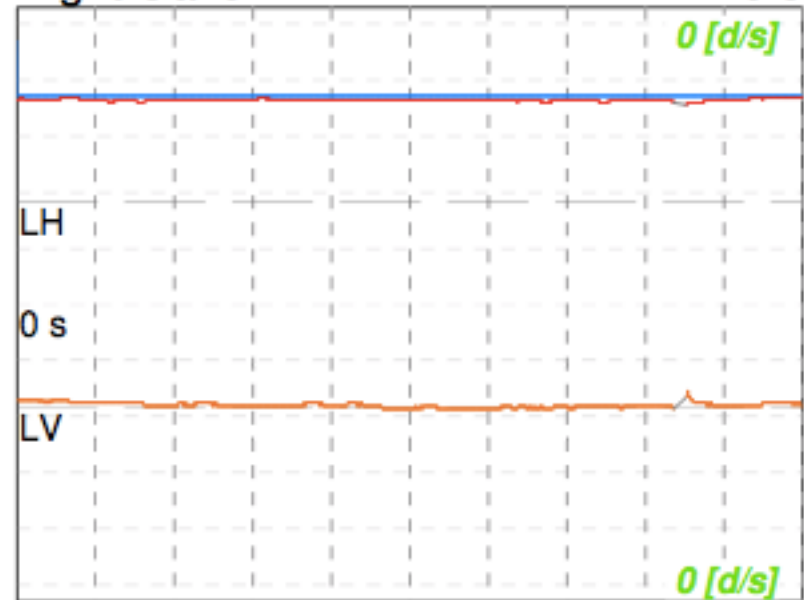
Video

Left Gaze



Right Gaze

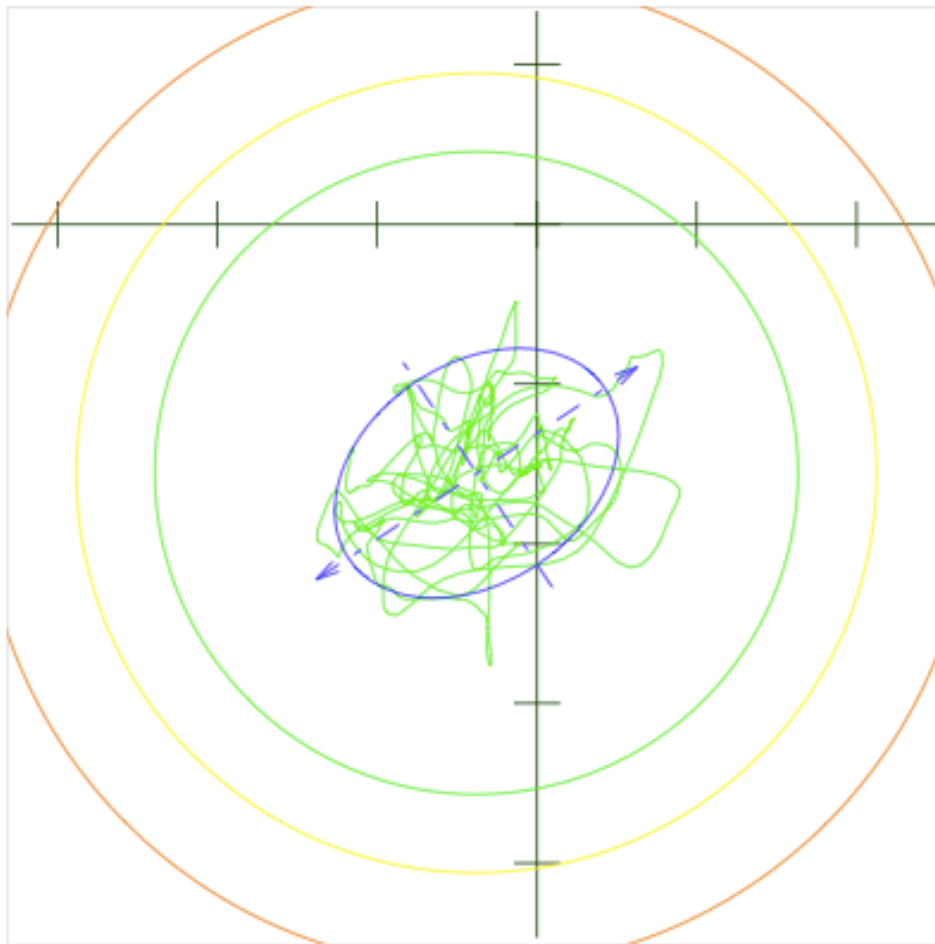
10 s



Computerized Dynamic Posturography



Computerized Dynamic Posturography



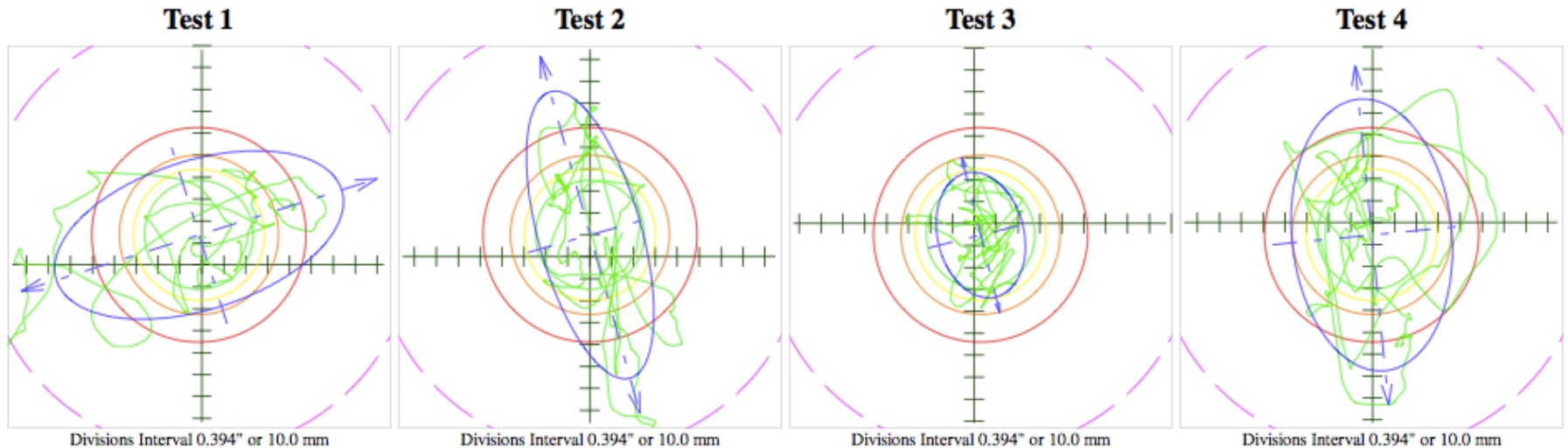
Divisions Interval 0.394" or 10.0 mm

| | |
|--------------------------------------|---|
| Test Name | Perturbed Stability Eyes Open |
| Test Type | PSEO |
| Test Date | 11/13/2013 01:10 PM |
| Test Parameters | 19.997s @ 64.011 Hz |
| Samples: Total & Valid | 1280 & 1280 |
| Test Status | Completed |
| Sway Center [Xo, Yo] | [-0.0038 m, -0.0156 m] |
| Max Theoretical Sway (LoS) | 0.2132 m |
| ML Sway: Max & 95% Conf. | 0.0009 m & 0.0177 m |
| AP Sway: Max & 95% Conf. | 0.0048 m & 0.0156 m |
| 95% Conf. Sway: Max & Min | 0.0193 m & 0.0137 m |
| Actual Sway: Max & Ave | 0.0138 m & 0.0054 m |
| Sway Velocity: Max & Ave | 0.094 m/s & 0.023 m/s |
| Sway Acceler.: Max & Ave | 1.693 m/s ² & 0.316 m/s ² |
| Sway Path Length | 0.4644 m |
| Area 95% Conf. Ellipse | 0.0002078 m ² |
| Stability Score | 91.0% |
| Stability Class | Healthy Balance |
| Pred. Direction of Sway | 33.6 deg |
| Directionality | 29% |
| Fatigue Ratio | 0.0% |
| Adaptation Ratio | 23.9% |
| Parameter p | 3.1 |
| Height | 1.78 m |
| Weight | 88.9 kg |
| Body Mass Index | 28.1 |
| Body Mass Index Class | Overweight |

| | | | | | |
|--------------------------|---------------------------|-----------------------|---------------------------|-------------------------|---------------------------|
| Stability Level: | Healthy Balance | Mildly Reduced | Moderately Reduced | Severely Reduced | Profoundly Reduced |
| Reference Values: | <u>>= 81.1%</u> | 76.5-81.0% | 70.9-76.4% | 60.0-70.8% | <= 59.9% |

Computerized Dynamic Posturography

| | | | | |
|--------------------------------|---------------------------|---------------------------|-----------------------|---------------------------|
| Stability Score | 0.0% | 31.5% | 70.6% | 0.0% |
| Stability Class | Profoundly Reduced | Profoundly Reduced | Mildly Reduced | Profoundly Reduced |
| Pred. Direction of Sway | N/A | -74.4 deg | -76.3 deg | N/A |
| Directionality | N/A | 65% | 32% | N/A |
| Fatigue Ratio | N/A | 40.7% | 0.0% | N/A |
| Adaptation Ratio | N/A | 0.0% | 10.8% | N/A |
| Parameter p | N/A | 5.1 | 4.0 | N/A |
| Height | 1.65 m | 1.65 m | 1.65 m | 1.65 m |
| Weight | 64.4 kg | 64.4 kg | 64.4 kg | 64.4 kg |
| Body Mass Index | 23.6 | 23.6 | 23.6 | 23.6 |
| Body Mass Index Class | Normal | Normal | Normal | Normal |



Visual Dep. (0% = no dependency): Romberg Ratio: N/A

Perturb. Dep. (0% = no dependency): Romberg Ratio: N/A

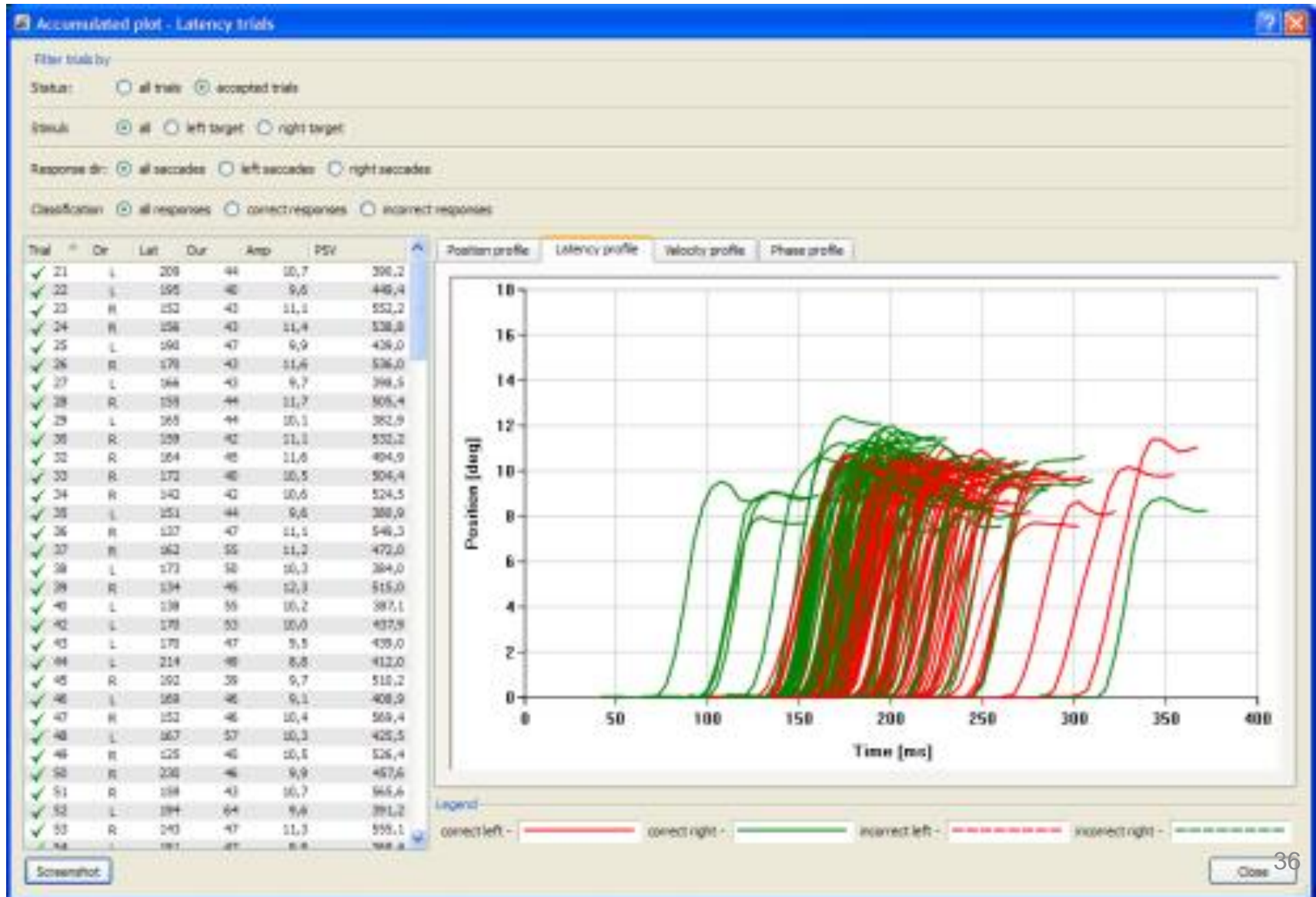
Stability Ratio: N/A

Stability Ratio: N/A

Saccadometry



Saccadometry



Vestibular Head Impulse Testing

Report Date: 10/22/2015

Report Operator: Default Administrator

Head Impulse

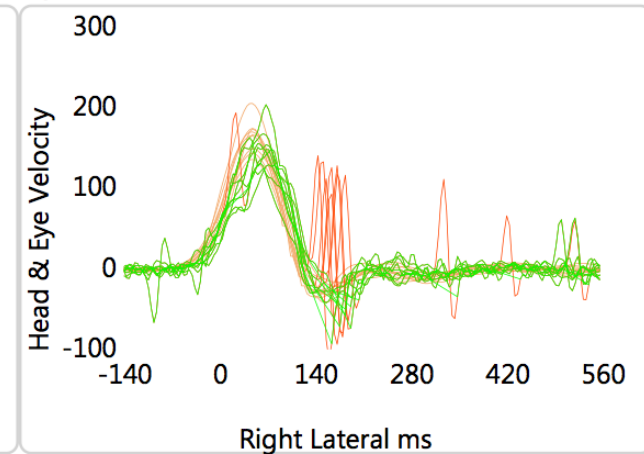
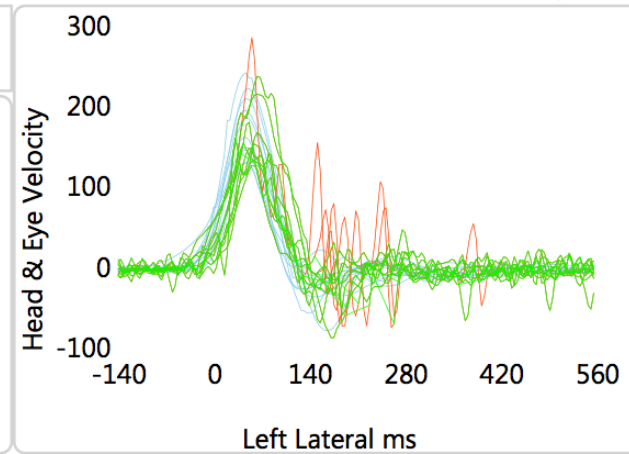
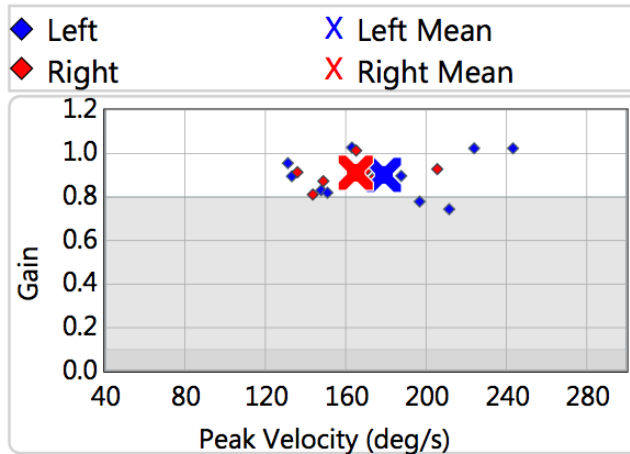
Lateral Impulse Test: 10/22/2015 3:48 PM

Test Operator: Default Administrator

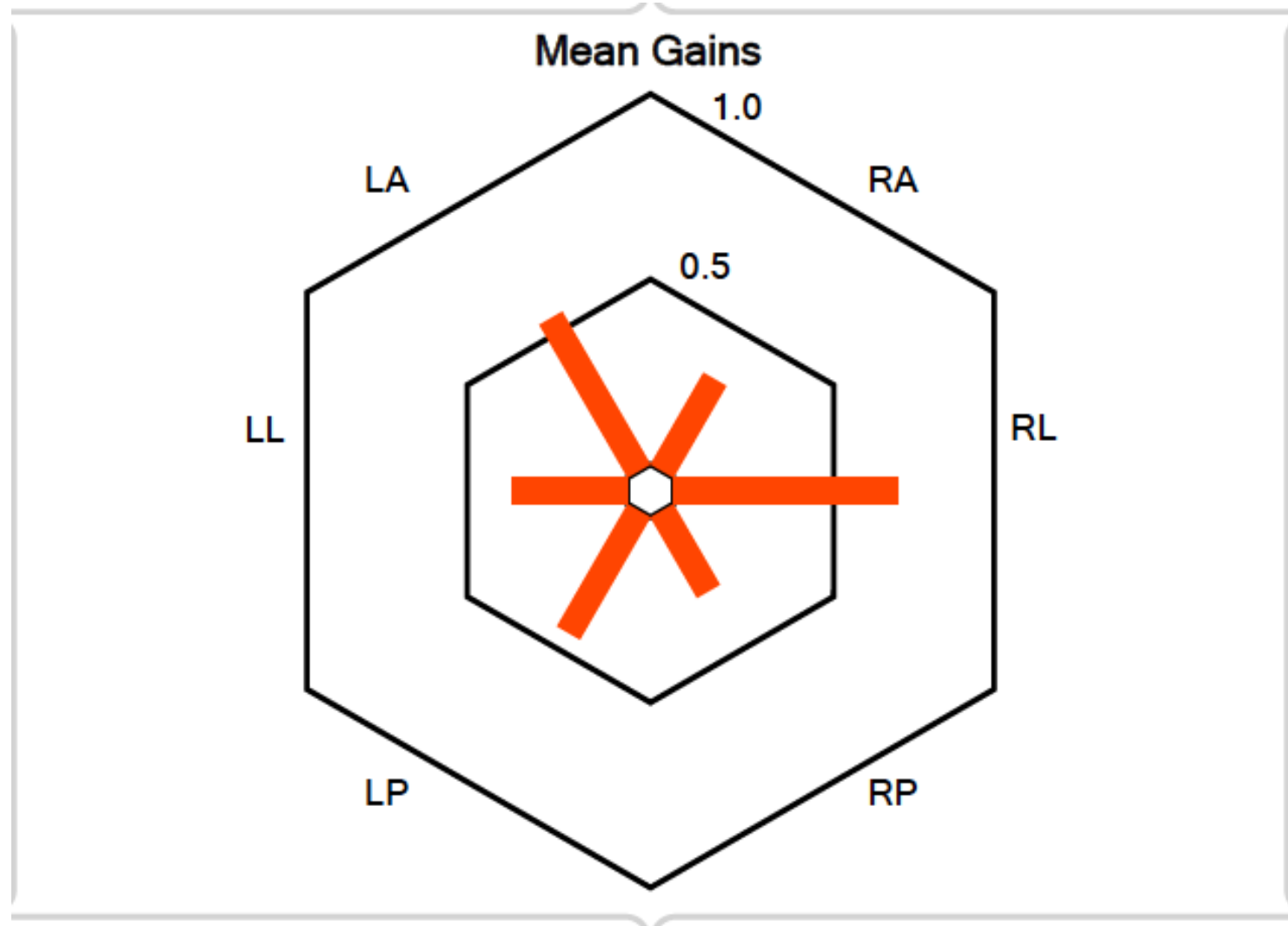
Left Mean: 0.9, σ : 0.1

Right Mean: 0.91, σ : 0.05

Asymmetry: 1%



Vestibular Head Impulse Testing



Case 1: Perry

- 64 YOM
- Received several concussions while in military service
- Suffers from severe vertigo
- Suffers from severe PTSD

- Suffers from severe agoraphobia and acrophobia
- Anxiety and panic develops in social situations involving multiple people or multiple conversations
- Is easily overstimulated by complicated visual and auditory environments

- Reports a constant sensation of motion and very poor balance
- Vertigo and instability are frequently debilitating

- Has constant pain in his parascapular and upper cervical regions
- Regular and ongoing physical therapy has proven to be only palliative
- Pain is a variable 3-7/10 VAS, primarily R sided

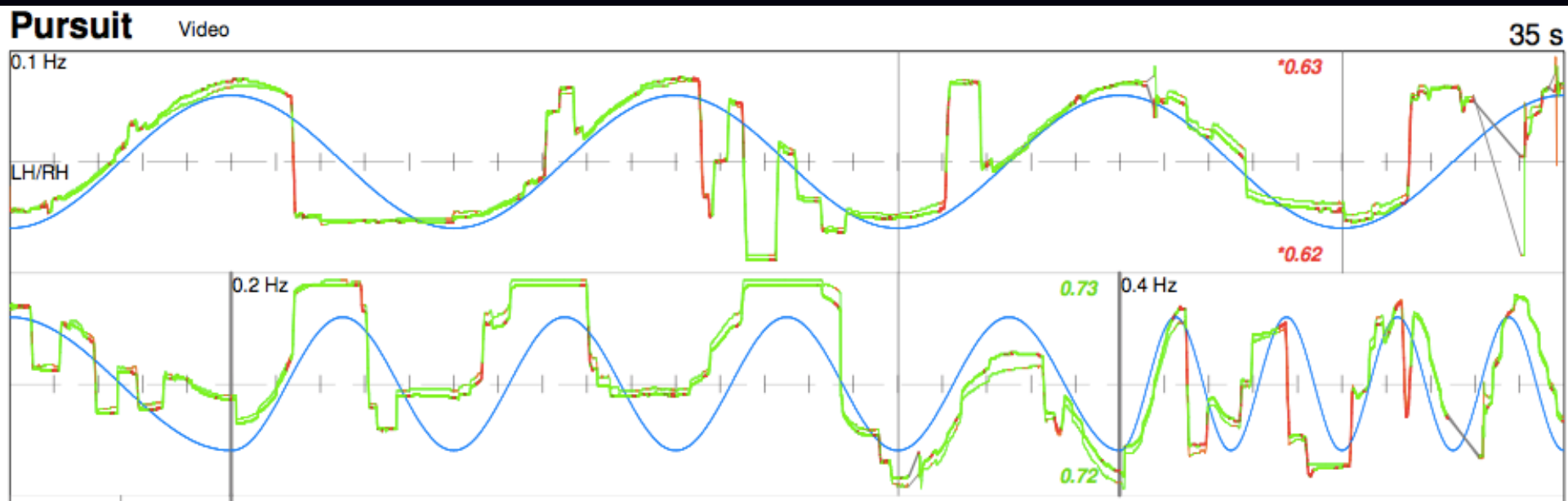
- Has moderate cognitive difficulties
 - Impaired processing speed
 - Impaired working memory to short-term memory conversion
 - Intermittent receptive aphasia

- Suffers from severe social withdrawal
- Is frequently unable to leave the house for months at a time, as vertigo triggers severe panic attacks
- Has no contact with anyone other than his spouse and healthcare providers

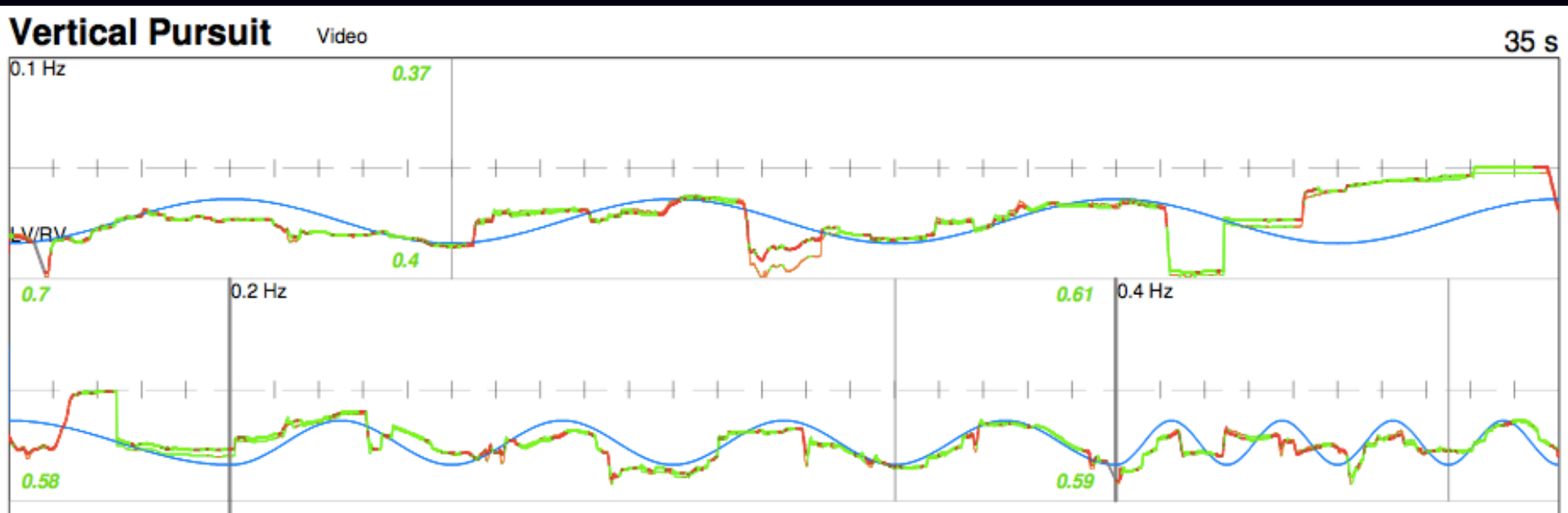
- Veterans Administration has determined **100% disability** for PPCS and PTSD

- Has received every therapy indicated, by extremely qualified people, without success

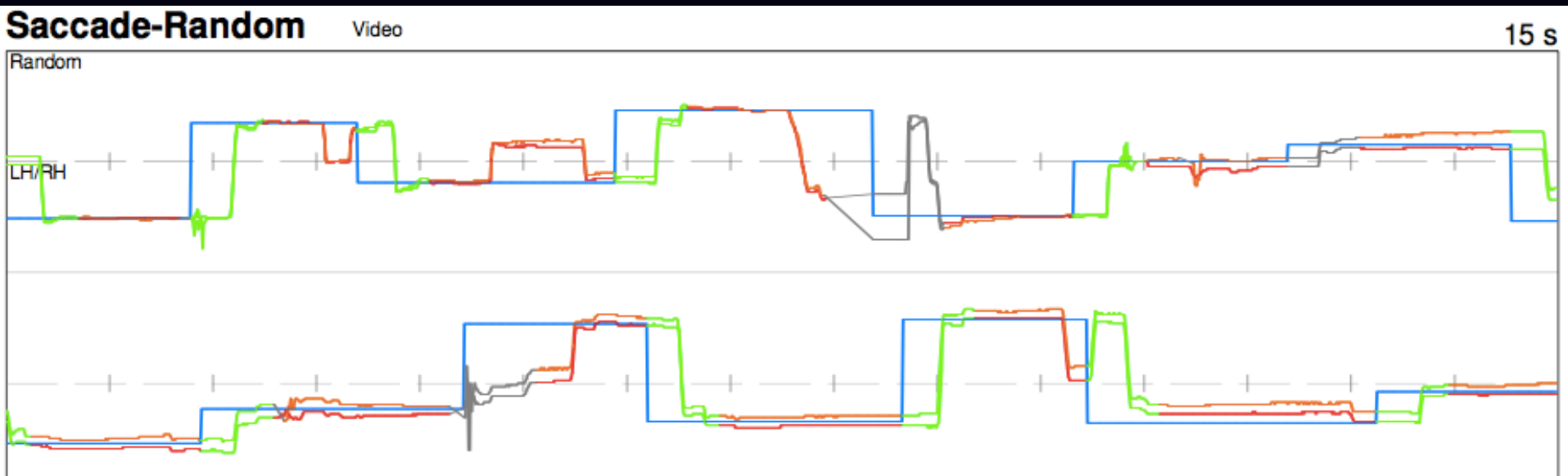
Video Nystagmography Findings



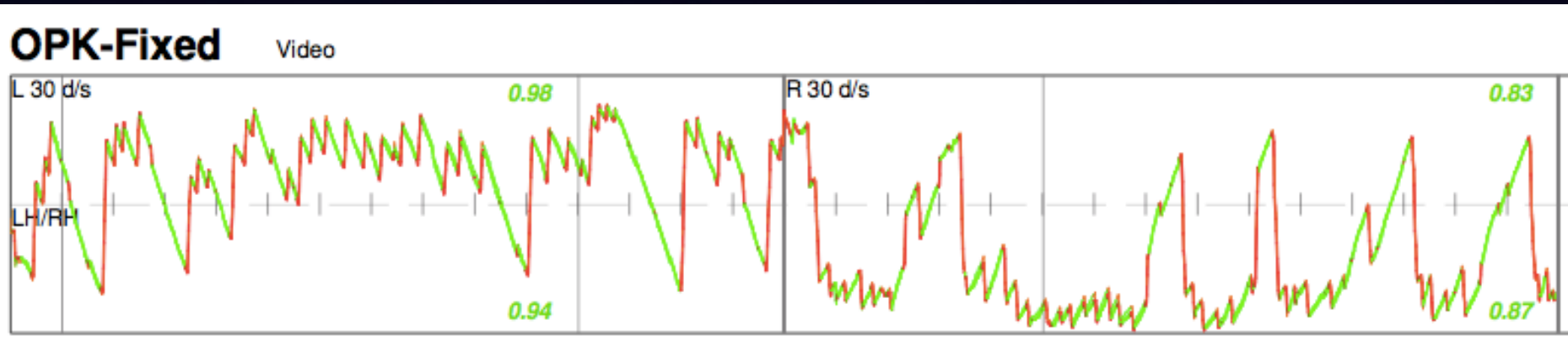
Video Nystagmography Findings



Video Nystagmography Findings



Video Nystagmography Findings



Treatment Parameters

- Therapy included:
 - Trigeminal nerve stimulation with a combination of low volt galvanic and somatosensory evoked potential stimulus
 - specific to his lesion

Treatment Parameters

- Yaw plane rotational chair stimulation with simultaneous vertical optokinetic stimulus
- specific to his lesion

Treatment Parameters

- Contraphasic hand-foot complicated movement exercises
- specific to his lesion

Treatment Parameters

- Extraocular pursuit-microsaccade exercises utilizing gap stimulus for R frontostriatal stimulus
- specific to his lesion

Treatment Parameters

- Gaze stabilization exercises utilizing combined pursuit-saccade-Halmagyi head thrust parameters
 - specific to his lesion

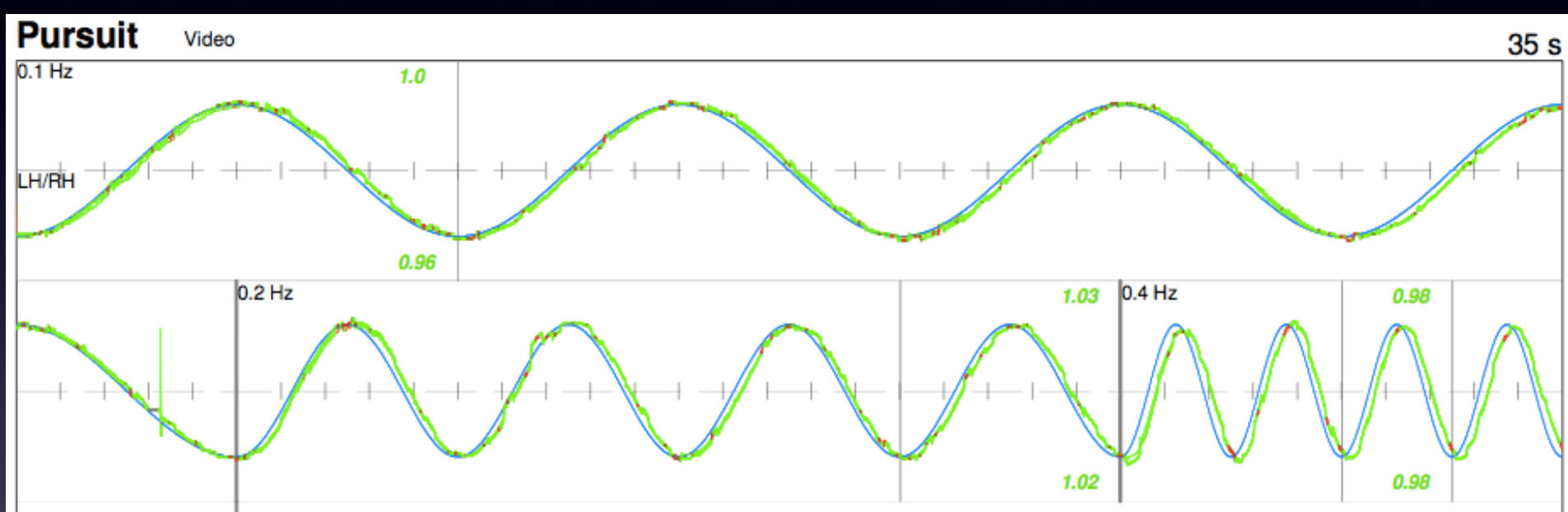
Treatment Parameters

- Visual pursuit exercises with simultaneous in-phase background optokinetic stimulus exceeding target velocity
- specific to his lesion

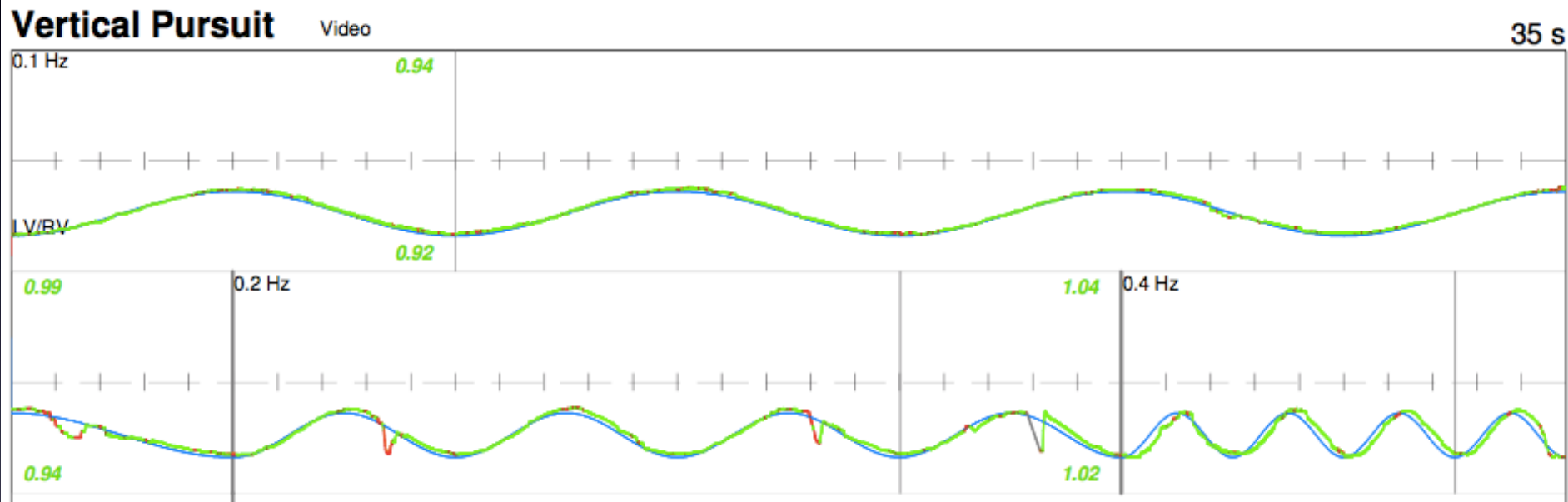
Results

- After 16 treatments over 8 weeks:

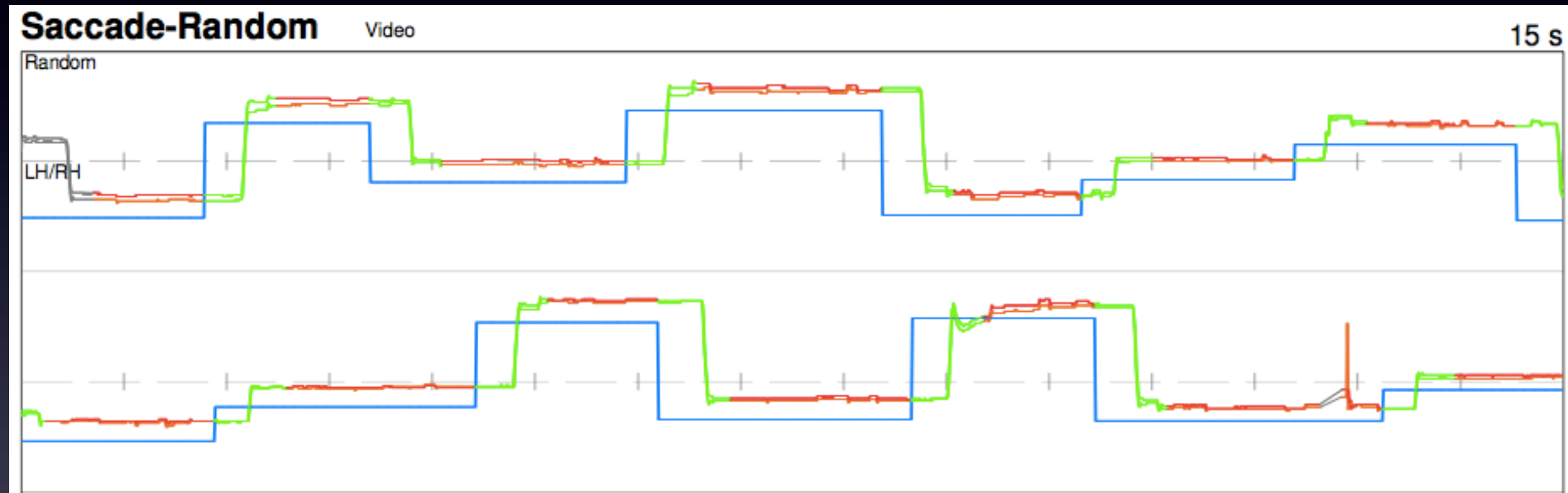
Video Nystagmography Findings



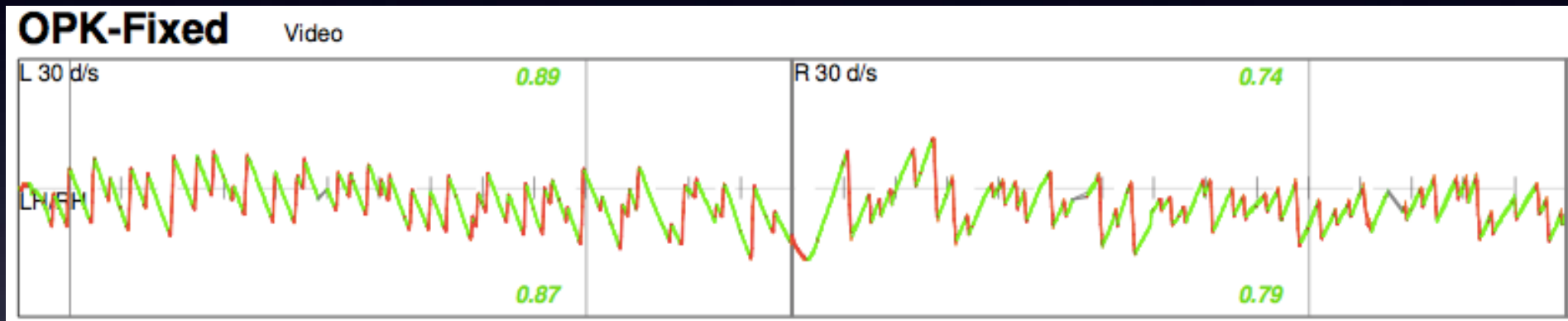
Video Nystagmography Findings



Video Nystagmography Findings



Video Nystagmography Findings



Results

- One month into care, PT was able to take his son to a Portland Trailblazers game, without any overstimulation, panic, vertigo, or other negative symptoms
- He reported having the best day he has experienced in ten years

- 6 weeks into care, agoraphobia and vertigo fully resolved:



CASE 2: ROSIE

- ▶ 27 YOF
- ▶ July 2015, falls attempting backflip on trampoline
- ▶ strikes occiput

- ▶ constant headache
- ▶ constant dizziness
- ▶ photophobia
- ▶ brain fog
- ▶ poor working memory
- ▶ poor spatial awareness

- ▶ seen by a comprehensive team of medical professionals
- ▶ MD neurologist
- ▶ MD PMR-sports medicine, hospital concussion clinic
- ▶ PT vestibular therapist
- ▶ DO
- ▶ LMT
- ▶ ND
- ▶ RDT
- ▶ personal trainer

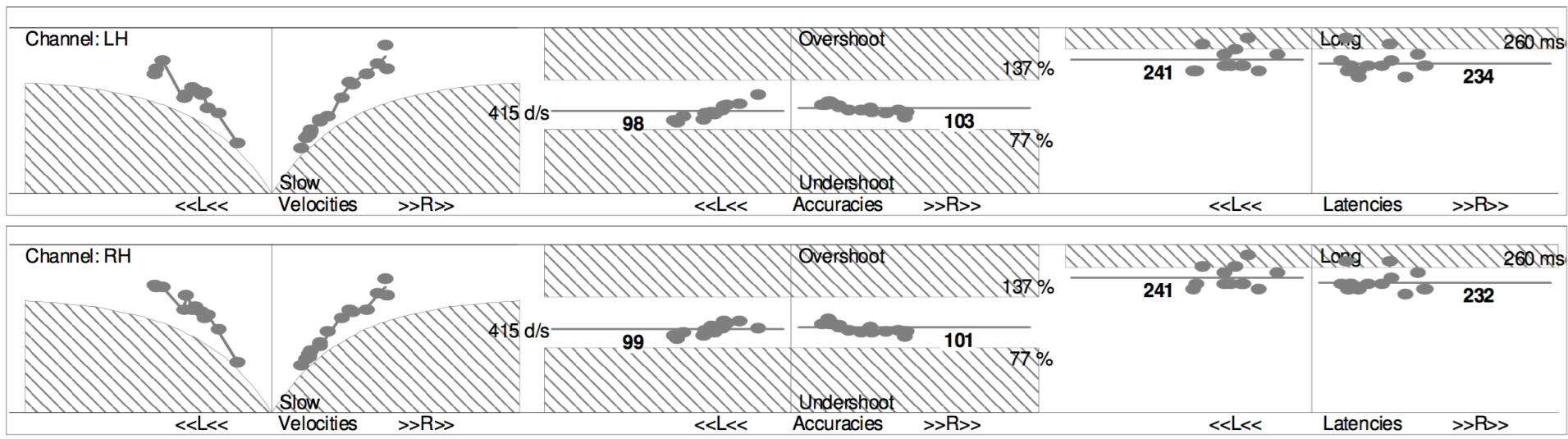
- ▶ began treatment including gaze stabilization and vestibular habituation
- ▶ gradually improved
- ▶ at 3 week evaluation, continued to demonstrate photophobia, phonophobia, impaired spatial awareness, and mild headaches

- ▶ August 2015, strikes her head on car door
- ▶ all sx return and dramatically increase
- ▶ requires 2 weeks off from all activity

- ▶ Continues to receive care from her entire team of professionals, but symptoms get progressively worse

- ▶ can engage in simple physical activity, but anything complex causes her to feel disoriented and “foggy”
- ▶ activity provokes her light and sound sensitivity
- ▶ Severe headaches provoked by exertion that persist throughout the day
- ▶ Only feels comfortable turning to her L
- ▶ struggles with falling and staying asleep
- ▶ anxious and labile

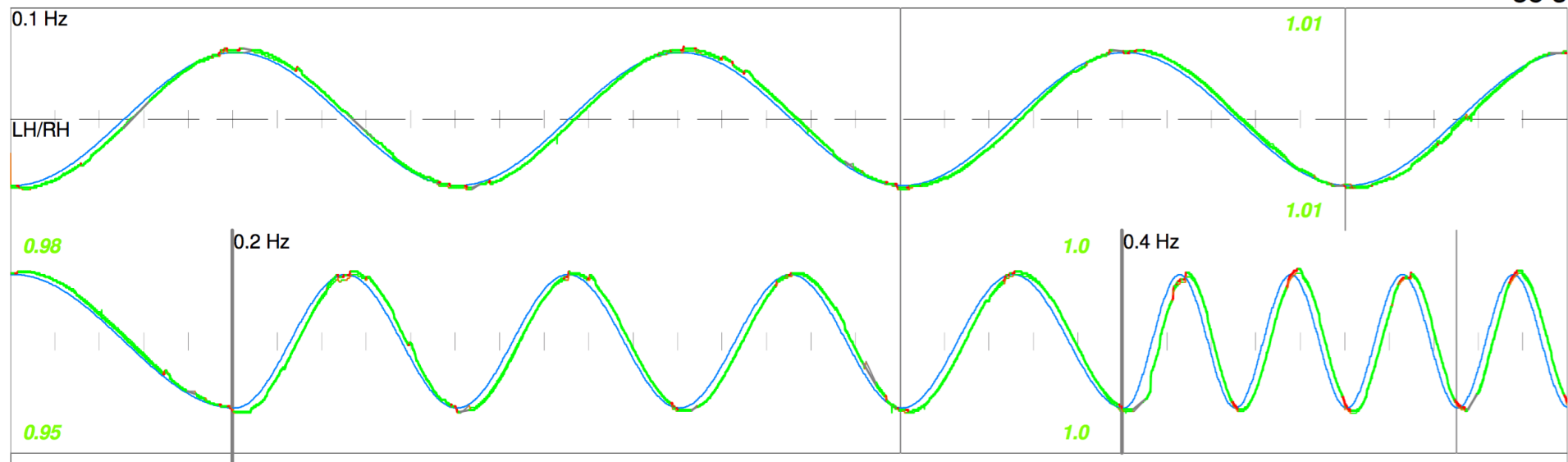
- ▶ Has been planning a trip to South America for years, unable to travel in this condition



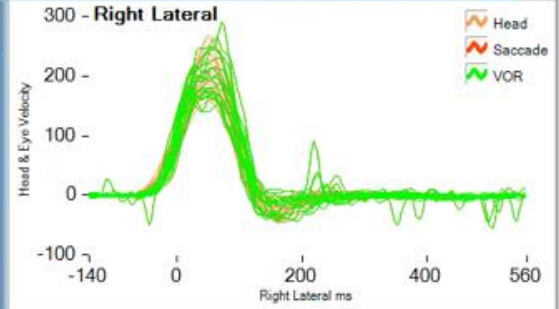
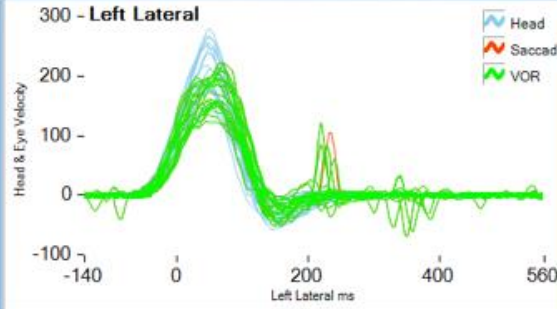
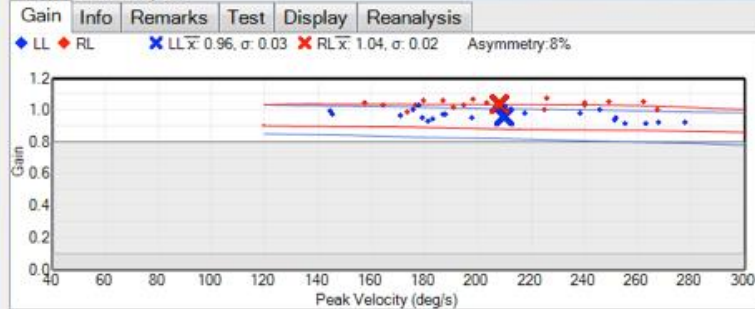
Horizontal Pursuit

Video

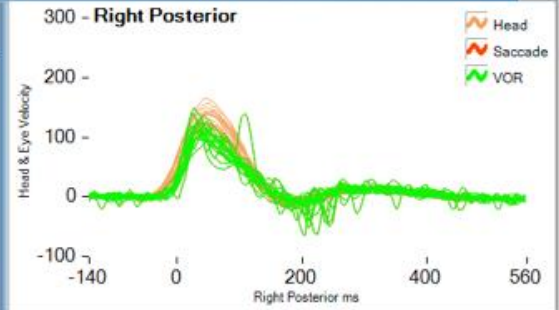
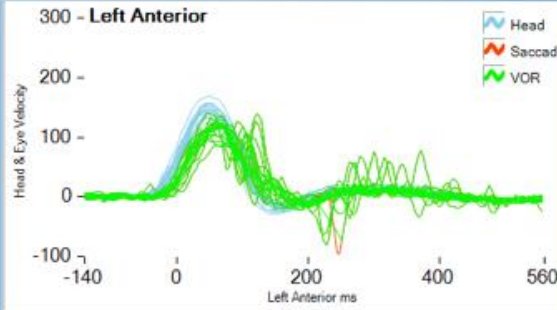
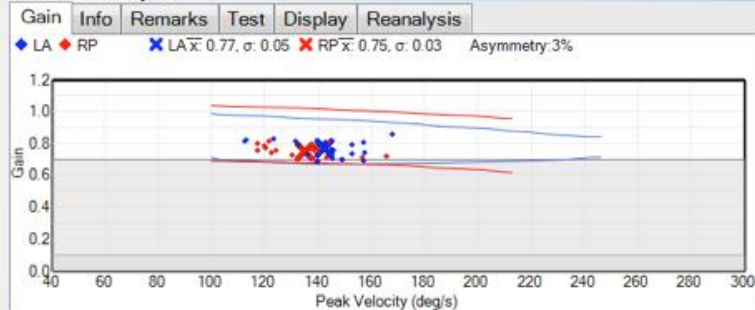
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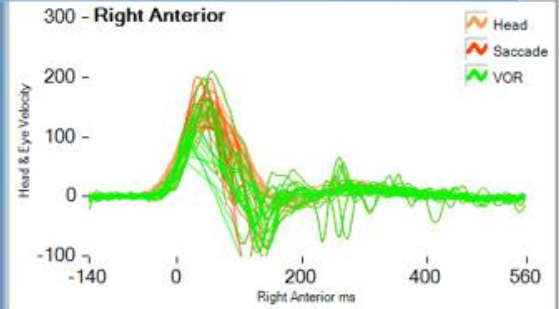
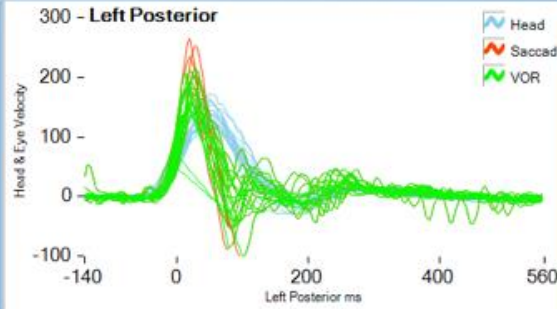
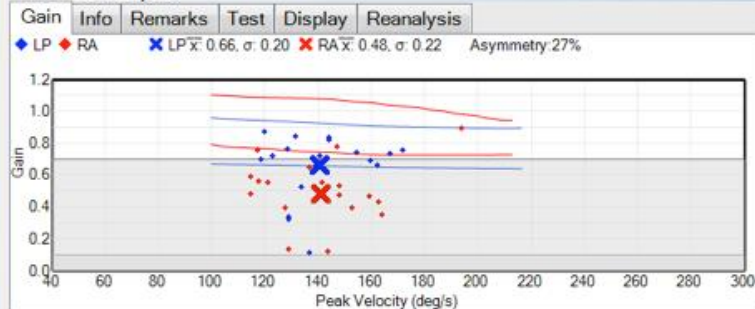
Lateral 2D Analysis



LARP 2D Analysis



RALP 2D Analysis

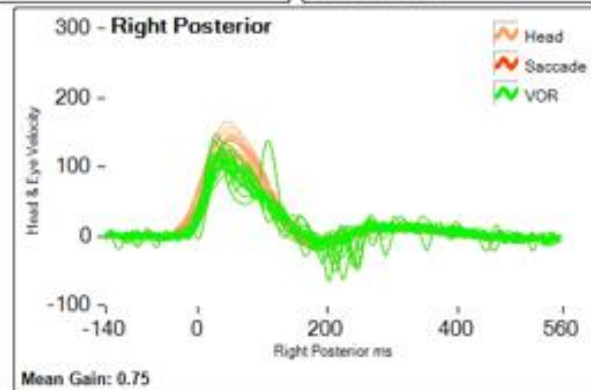
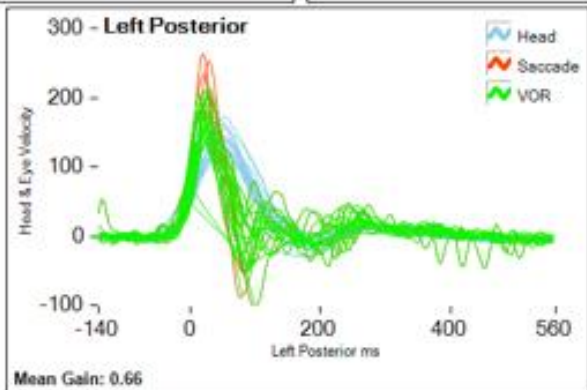
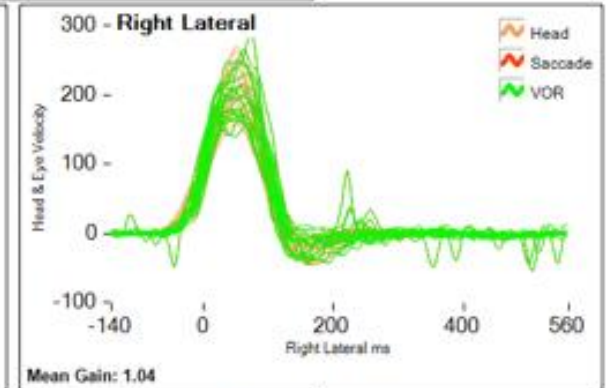
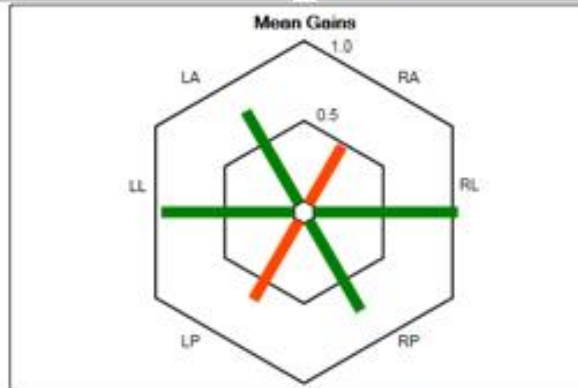
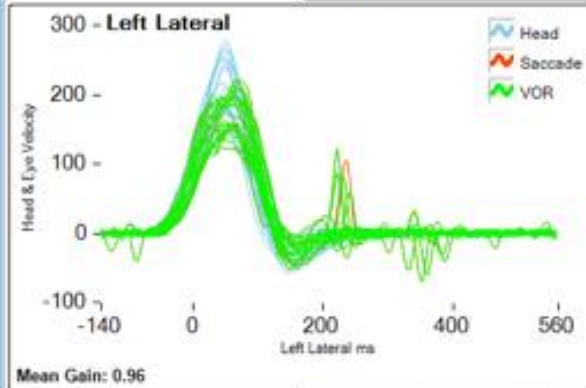
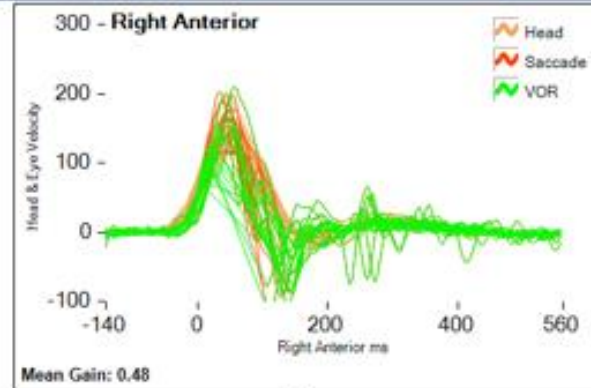
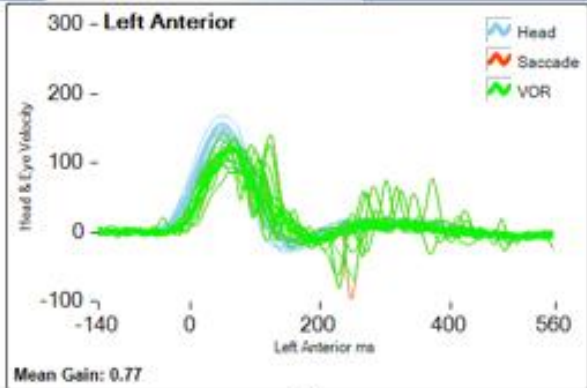


Asymmetry

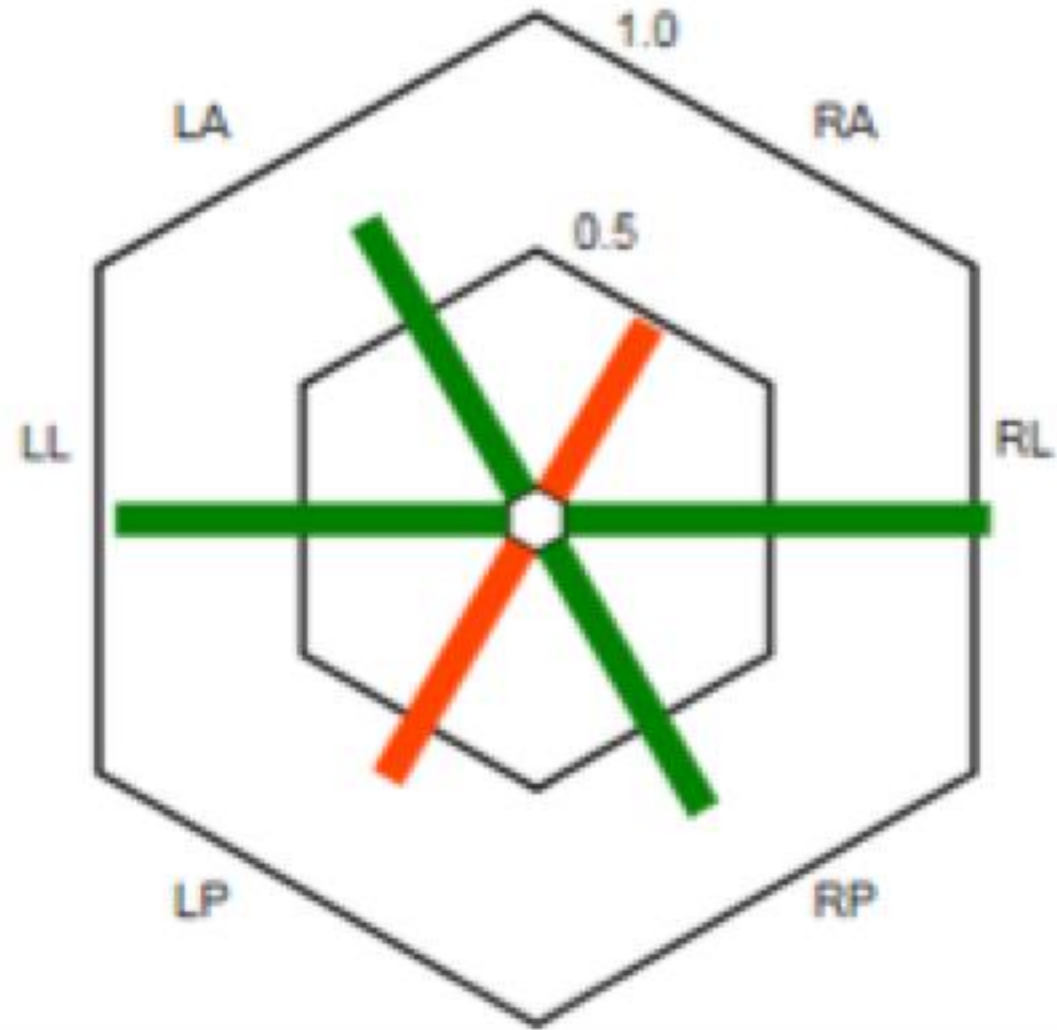
Anterior: 38%

Lateral: 8%

Posterior: 12%



Mean Gains



► Exercises:

1. Saccade from L-R into RALP plane gaze stabilization
2. R upper and lower extremity contraphasic complex movement exercises
3. R Halmagyi into 0.1 Hz pursuit R, with in-phase faster optokinetic background
4. R anterior canal stimulation exercises

► Follow up:

1. 5 reps R hand and foot opposite figure 8s.
2. 2 sets L inferior rectus saccades.
3. 5 reps R hand and foot opposite figure 8s.
4. 2 sets X2 upward pursuits.
5. 5 reps R hand and foot opposite figure 8s.
6. Vertical gaze stabilization.

- ▶ All symptoms resolved after 2 weeks of care, and Rosie was able to go on her trip.







Case 3:

Severe TBI with Locked In Syndrome

Locked-in syndrome is a condition in which a patient is aware but cannot move or communicate verbally due to complete paralysis of nearly all voluntary muscles in the body except for the eyes.

Total locked-in syndrome is a version of locked-in syndrome wherein the eyes are paralyzed as well.

Locked-in syndrome usually results in quadriplegia and the inability to speak in otherwise potentially cognitively intact individuals.

Those with locked-in syndrome may be able to communicate with others through coded messages by blinking, sniffing, or moving their eyes.

Patients who have locked-in syndrome are conscious and aware, with no loss of cognitive function.

They can sometimes retain sensation throughout their bodies.

Neither a standard treatment nor a cure is available.

Level of independence is directly related to motor recovery.

Very few reach a level of motor recovery enabling them to feed and perform activities of daily living on their own.

The prognosis for patients that suffer both brainstem and higher brain injury is extremely poor.

Stephanie

- 21 YOF
- Premed student, on her way to class in at Boston University

HPI

- Involved in MVA
- Vehicle crushed by semi-trailer
- Airlifted to hospital

HPI

- Broken ribs
- Pneumothorax
- Lacerated liver
- Internal bleeding

HPI

- Fractured pelvis
- Fractured R arm multisite
- Fractured L arm multisite
- Fractured mandible

HPI

- Multiple skull fractures – L temporal, L occipital
- Severe multisite intracranial hemorrhage
- Massive multisite internal bleeding

HPI

- Stabilized with induced coma for 2 weeks
- Multiple reconstructive surgeries

HPI

- ICU for 2 months
- IVP shunt placement for hydrocephalus

HPI

- Initial examination:

HPI

- Nonambulatory
- Bilateral decorticate posturing
- Marked L roll head tilt
- Marked R hypertropia
- Fully locked in

Examination Findings

- Unable to generate any volitional eye movements
- Unresponsive to any commands
- Only movement was what appeared to be random truncal myoclonus

Examination Findings

- Lesion presentation:
 - bilateral ventral pons
 - R cerebellum
 - L temporoparietal
 - Bilateral frontal

Examination Findings

- **Worst prognosis possible**
- leaned forward and generated sustained eye contact when her prognosis was being discussed

Treatment Parameters

- R rotational chair exercises with downward optokinetic exercises
- Passive R hand and foot contraphasic complicated movement exercises
- R Posterior Interosseous nerve 128 Hz vibratory stimulus.

Treatment Parameters

- Following R hand vibratory stimulus, Stephanie begins to saccade horizontally to targets on command.
- After 2nd set she develops spontaneous eye movements that become significantly distinct.
- After 15 minutes of treatment these are clearly responses to commands.

Treatment Parameters

- Performed 2 week intensive inpatient treatment protocol
- Treatment consisted primarily of:
 - 2:1 R:L warm caloric stimulus
 - Passive R hand and foot contraphasic complicated movement exercises
 - R rotational chair exercises with downward optokinetic exercises

Treatment Parameters

- Attended somatosensory evoked potential stimulus to
 - R posterior tibial
 - R posterior interosseous
 - bilateral V1-V3 Trigeminal nerves

Treatment Parameters

- After and of first week, had developed ability to stand with support, with frequent asterixis.
- Developed capacity to respond to yes-no questions with horizontal eye movements.

Treatment Parameters

- Treatment plan modified to include:
 - R 100Hz and L 500Hz auditory stimulus
 - L olfactory stimulus
 - R superior quadrant visual stimulus
 - R high volt galvanic vestibular stimulus

Results

- After 2 weeks of treatment:



Results

- After 1 month of care:

