NeuroLens: A tool to treat post-TBI visual hypersensitivity?

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Concussion/TBI: Why Vision?

- 80-90% of all information entering the brain is visual
- Over 50% of brain is involved in visual processing (over 30 brain regions and 8 cranial nerves)
- Visual processing alone consumes 44% of brain’s energy
- 90% of all concussions will have 1 or more ocular problems
- Hidden vision problems frequently persist 6-9 months after a concussion
Visual Symptoms Associated With TBI

Visual field deficits
Visual inattention/difficulty shifting attention
Difficulty following moving targets (pursuit dysfunction)
Poor spatial localization
Poor depth perception
Visual perception deficits
Poor tracking when reading (saccadic dysfunction)
Vertigo/disequilibrium (VOR dysfunction)
Eyestrain/double vision (Convergence insufficiency)
Blurred vision (Accommodative dysfunction)

Visual Hypersensitivity: Photophobia/Light Sensitivity (photosensitivity) and Motion Sensitivity
Parvocellular Pathway

• Focal vision
• What is it?
• Responds to high contrast targets
• Static, non-moving targets
• Slower processing speed
• Most vision tests assess this pathway
Magnocellular Pathway

• Spatial orientation (Where is it?)
• Posture/balance
• Movement
• Preconscious anticipation of change in environment
• Rapid speed in processing
• Autonomic nervous system—parasympathetic/sympathetic
• More prone to dysfunction after TBI
What is Visual Hypersensitivity?

• Brain injury can cause neurological disinhibition, increasing one’s sensitivity to **light** and **movement**, resulting in visual cortex hyperexcitability

• An increased magnitude of the hyperexcitability results in a corresponding increased susceptibility to visual stress symptoms

• Reduced filtering of extraneous information, background noise, and central-peripheral visual mismatch contribute to increased visual hypersensitivity
Photosensitivity

• 50% of those with mTBI (10% in non-concussed; 50-90% in migraineurs), time-course months to years after incident

• Cortical or subcortical regulation of response to changes in illumination and visual-spatial patterns, possibly mediated by the dorsal visual pathway, may be contributing to the perception of photosensitivity in those with TBI

• Altered pupillary light reflex dynamics (biomarker of mTBI PS)?

• Elevated critical flicker fusion frequency (CFF)—the minimum light flicker frequency for an individual to perceive a steady (non-flickering) presentation of light—may be related to discomfort with fluorescent lighting

• Dysfunction of suprachiasmal nucleus and hypothalamus in mTBI?

• Dense tints typically tried, but may not allow for neural adaptation. Tints of 30% transmission or less recommended

• Brimmed hats are helpful

• CL may also allow for neural adaptation in mTBI
Three ways the trigeminal nerve may be irritated by photosensitivity

1. Light could excite neurons in multiple regions of the caudal trigeminal brainstem complex and increased parasympathetic outflow to the eye, cause vasodilatation of choroidal blood vessels, activation of neural pain sensors in blood vessels, and finally an abnormal discomfort and pain.

2. Melanopsin specific ganglion cells of retina connect to pain regions in trigeminovascular thalamic neurons which then project to multiple cortical areas.

3. Light and melanopsin specific ganglion cells project directly to trigeminal nerve outside optic nerve pathway to triggering pain.
Where is light-modulated nociceptive information processed?

Melanopsin RGC innervates Po nociceptive neurons

Spinal trigeminal neuron innervates Po nociceptive neuron

Trigeminal innervation of dura mater

Trigeminal ganglion projects to spinal nucleus
Clinical Correlates of Photosensitivity

• Pupillometry: Pupillary diameters, constriction latency, average constriction velocity and peak dilation velocity

• Critical Flicker Fusion Frequency (CFF): Elevated thresholds, (over 50 Hz)
Visual Motion Sensitivity

• Intolerance to busy environments with changing light patterns, visual movement, or clutter
• Distinct entity from a VOR gain defect causing vertigo
• ? Self motion vs. object motion signals across visual field disrupted at V6/V6 resulting in visual instability (visual cortex) and disequilibrium
• Elevated Coherent Motion Thresholds
• VEP magnocellular latency delay: Where ➔ What becomes What ➔ Where
• Pattern Glare Provocative Test (striped patterns cause visual illusions or even vertigo)
• Stroboscopic Provocative Test (intolerance increases with increased strobe speed)
• OKN/Gibsonian Optic Flow Provocative Test
• Saccadic Fixation Disparity Provocative Test (disturbing response to dichoptic target)
• Binasal occlusion reduces symptoms
• Base-in prism reduces symptoms of pattern glare, disturbance on saccadic fixation disparity card
Coherent Motion Threshold Test

• CMT: 10% or above significant
Magnocellular latencies greater than 119 milliseconds to 0.25-cycle/degree stimuli (or mean vertical sinusoidal latencies >113 milliseconds to 0.50-cycle/degree stimuli) and mean vertical sinusoidal grating amplitudes of less than 14.75 mV to 0.50-cycle/degree stimuli were classified as having had a history of concussion.
Limitations of this study included the subjects in the youngest age group 8 to 20 years did not return for retest (Session 2) due to lack of time and interest in completing the follow-up visit. Thus, the test and test–retest analysis was not available for this age group. The young subjects had no difficulties in completing the VPT test and the VLSQ-8, but more data are needed to determine the validity of the results in children. The participants reflected the racial and ethnic population in our locale, and most have dark irises, which prevented an adequate analysis with respect to factors, such as iris color. While data from our sample size are helpful to establish test re-test reliability, further studies to include a larger number of persons in each age category and across with other racial and ethnic makeup would be helpful to further the establishment of normative data.
Stroboscopic Provocative Test
OKN/Gibsonian Optic Flow Provocative Test
Is Binocularity a factor in visual hypersensitivity?
Common binocular dysfunctions associated with TBI include:

- Tendency toward exophoria (outward resting state of the eyes)
- Tendency toward reduced fusional vergences and convergence insufficiency
- Tendency toward exo fixation disparity
Exophoria

Angle of heterophoria
What is Fixation Disparity?

- When single vision is maintained during fusion, retinal slippage can occur so that bifixation can be lost within Panum’s Fusional Area and no double vision is noted; 5-10 min of arc in magnitude
- Thought to be associated with asthenopia (e.g. convergence insufficiency) especially when engaged in near work
Convergence Insufficiency can make text look double when trying to read.

Some people with Convergence Insufficiency experience a “halo effect” instead of double vision.
Saccadic Fixation Disparity Provocative Test
Evidence that binocularity is involved in visual hypersensitivity:

• Closing/covering one eye reduces the pattern glare disturbance
• Closing/covering one eye reduces moving text or halo effect when reading
• Base-in prism reduces symptoms by reducing convergence demand and exo fixation disparity
• Binasal occlusion reduces symptoms possibly by reduced overlap of nasal binocular fields
• Binocular vision therapy reduces symptoms
Binasal Occlusion is thought to reduce symptoms by eliminating some of the binocular field overlap.
Binasals block some visual field overlap
Prescribing small amounts of base-in prism artificially reduces exophoria and exo FD
NeuroLenses work by artificially reducing exophoria at far and another 1.7 prism diopters at near using a progressive prism, thus improving alignment and reducing convergence stress more than traditional base-in lenses.
Graphical Analysis: BI prism reduces phoria at far and near; NeuroLenses reduce phoria even more for near Base-In NeuroLens
Empirical Method of Measuring Prism for NeuroLenses
Evidence that trigeminal nerve is related to spatial localization and visually guided movements

• Disturbance of visually guided movements and spatial localization often occurs after H. Zoster infection

• Surgical ablation of the trigeminal nerve disrupts cortical spatial organization during development
EOM proprioceptive fibers connect to trigeminal nerve to aid in spatial localization.
Theory of why NeuroLenses improve TBI-related visual hypersensitivity and asthenopic symptoms

TBI causes greater exophoria (outward eye resting posture), convergence insufficiency and exo fixation disparity/unstable fixation.

Along with binocular misalignment, there is poor synchronization of peripheral vision tracking system, controlled primarily by magnocellular driven saccadic eye movements, and central vision tracking, controlled primarily by parvocellular driven pursuit system occurs.

The proprioceptive fibers of the extra-ocular muscles are activated by constant attempts to rectify a binocular misalignment and/or perceptual magno/parvo timing imbalance.

This lack of coordination results in an over stimulation of the trigeminal nerve which in turn overactivates the trigeminal nucleus caudalis. The trigeminovascular system links the trigeminal nerve territory and the upper cervical region via the trigeminal nucleus caudalis which explain the pain referred from the head and the neck.

NeuroLenses reduce the binocular stress and may alter the perceptual magno/parvo timing mismatch. Because the visual system doesn’t need to work as hard, there is a relief of symptoms.
Case Study

• 30 y/o female 5 year post-concussion: daily eyestrain, blur, headaches into the neck/shoulder area, nausea, diplopia, light sensitivity, motion sensitivity, reading difficulties
• Mild astigmatism, low exophoria, exo fixation disparity: convergence insufficiency
• Unable to do coherent motion threshold test due to severe nausea
• Magnocellular latency lag on VEP, nausea felt during test
• Pt prescribed 1.5 BI NeuroLenses and noted immediate relief of eyestrain, headaches, motion sensitivity and photophobia
• Pt was able to begin active vision therapy after two months of wear
• Three months after initial eval, mild symptoms gradually returned
• Pt prescribed additional 1 prism diopter BI which neutralized distance FD
• “The world has stopped swimming!” “I can now read, shop, socialize, and function all day without symptoms. . .Thank You!”
Typical NeuroLens Rx and Responses

- Single vision lenses about $650.00
- Multifocal (progressive) lenses about $850.00
- First few days may have increase in symptoms
- May need to increase prism after 3-6 months or more
- Non-concussed patient responses show improved symptoms in about 78% of users
- May become a good bridge to vision therapy and/or an adjunctive treatment tool for patients with post-TBI visual hypersensitivity
Questions remain:

• What is the exact mechanism of action?
• Will regular base-in prism lenses perform just as well as NeuroLenses?
• Will NeuroLenses have customizable progressive base-in prescription powers available?
• Do NeuroLenses improve VEP magnocellular processing, coherent motion processing thresholds, or critical flicker fusion measures?