



NeuroVision Associates
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When Your Brain Is Inflamed, Vision Pays the Price

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When Your Brain Is Inflamed, Vision Pays the Price

The Symptom Nobody Can Explain

You've been to the emergency room. You've had an MRI, and it came back "normal." Your eye doctor checked your eyes and told you they're healthy – 20/20, in fact. And yet:

Reading makes you nauseous after ten minutes. The grocery store aisle feels like it's tilting. Screens leave you exhausted in a way that sleep doesn't fix. Overhead fluorescent lights make you want to close your eyes and sit down. You've started avoiding the highway at night.

Nothing on the standard tests is wrong. But something is very clearly wrong.

For a large number of our patients, the missing piece is neuro-inflammation – inflammation inside the brain and nervous system itself. It doesn't show up on a routine eye exam, and it rarely shows up on a standard MRI. But it has a profound effect on how the visual system works, and understanding it changes what we can do about it.

Part 1 of a series on neuro-inflammation and the visual system:
trauma and immune-driven inflammation.

What Neuro-Inflammation Actually Is

Your brain has its own dedicated immune system. The main players are cells called microglia – roughly ten percent of the cells in your brain. In their resting state, they're housekeepers: they prune unused connections, clear out cellular debris, and support the neurons around them.

When the brain is injured, infected, or under metabolic stress, microglia switch into defense mode. They change shape, multiply, and release inflammatory signaling molecules called cytokines. In the short term this is exactly what you want – it's the brain's version of the swelling and redness around a cut. The problem is when they don't switch back off.

This is the finding that reframed how we think about brain injury. In a landmark imaging study, researchers used PET scanning to look directly at microglial activity in people who'd had a traumatic brain injury months to years earlier. They found active, ongoing inflammation **up to 17 years after the original injury** – and the degree of inflammation tracked with how impaired patients were cognitively, independent of how much visible structural damage they had. [1]

Read that again: independent of visible structural damage. The scan can look fine while the process is still running.



Why the Visual System Takes the Hit First

Vision is not a small corner of the brain. Depending on how you count, somewhere between half and two-thirds of the brain's pathways are involved in processing or acting on visual information. That makes the visual system uniquely exposed to anything that disrupts brain function broadly.

Three features make it especially vulnerable:

- **It runs on long, myelinated wiring.** Signals travel from the retina to the back of the head and then forward again to areas that control eye movement, balance integration, and attention. Those long tracts are insulated with myelin, and myelin is one of the first things inflammation degrades. Damage the insulation and the signal doesn't stop – it slows.
- **It is metabolically expensive.** Visual processing burns an enormous amount of energy. Inflammation impairs how efficiently brain cells produce energy, and the highest-demand systems fail first, especially under sustained load. This is why symptoms so often appear after twenty minutes of reading rather than in the first two.
- **It requires precise timing between two eyes and many brain regions.** Binocular vision, eye tracking, and the constant handshake between your visual and balance systems all depend on signals arriving on schedule. Slow one stream relative to the other and the brain receives a mismatch. Your nervous system interprets mismatch as danger – which is why the downstream symptoms are dizziness, nausea, anxiety in visually busy spaces, and a powerful urge to get out of the store.

That last point is the heart of it. Most inflammation-related visual symptoms are timing problems, not clarity problems. That's precisely why a 20/20 eye chart misses them. The chart measures the sharpness of a static letter. It says nothing about speed, synchrony, endurance, or integration.

Where We Can Actually See It

This is where objective testing matters, because it turns "I feel awful and no one can find anything" into a measurable finding.

The **Visual Evoked Potential (VEP)** measures the electrical response your visual cortex produces when your eye sees a patterned stimulus, and – critically – how many milliseconds it takes to get there. Experimental work has established that VEP latency (the delay) tracks closely with the amount of demyelination along the pathway, while VEP amplitude (the size of the response) tracks with loss of nerve fibers themselves. [2] More recent work has shown that the latency delay reflects myelin health not just in the optic nerve but along the deeper visual pathway into the visual cortex. [3]

Translated: the test can show us that the signal is arriving late, and give us a reasonable read on why.

Paired with EEG/ERP testing, which looks at how efficiently the brain processes and responds to information, we can document a pattern that a routine exam simply cannot capture – and we can retest to see whether it's moving.



We'd Love To See You on Your Next Visit

Vision Rehabilitation and Therapy In Plano, Texas.



Weekly White Paper

When Your Brain Is Inflamed, Vision Pays the Price

Trauma: The Most Common Trigger We See

Concussion and mild traumatic brain injury are the most frequent entry point into this picture at our practice. The mechanical injury happens in an instant. The inflammatory cascade it sets off can run for months or years.

Animal work has demonstrated that persistent microglial activation after brain trauma drives progressive neuronal loss and myelin breakdown for as long as a year after a single injury [4], and that when microglia are blocked from mounting that chronic response, the cognitive impairments don't develop at all [5].

This matters clinically for one reason above all: **it explains the patient who was “fine” for a while and then got worse.** Families interpret that as psychological. It isn't. It's a biological process with its own timeline, and it's often still active – and therefore still addressable – long after everyone has been told to just wait it out.

Common visual presentations after trauma include convergence insufficiency (eyes struggling to work together at near), accommodative dysfunction (focusing fatigue), unstable eye tracking during reading, light sensitivity, and visual-vestibular mismatch in motion-rich environments.

Immune-Driven Inflammation: When the System Turns Inward

The second category is inflammation that isn't triggered by an impact at all, but by the immune system targeting nervous tissue directly – or by an immune response that started elsewhere and never stood down.

Multiple sclerosis is the classic example, and optic neuritis – inflammation of the optic nerve – is frequently its first symptom, often presenting as pain with eye movement and a washing-out of color and contrast in one eye. But the broader group is larger than MS: lupus, Sjögren's, thyroid eye disease, rheumatoid disease, and post-viral immune activation can all produce visual symptoms through inflammatory mechanisms.

These patients often describe something subtly different from trauma patients: colors look faded or “dirty,” contrast is poor even though acuity tests fine, and symptoms fluctuate – worse during a flare, worse with heat, worse when they're run down. Fluctuation is a hallmark, and it's another reason these patients get told it's in their head.

IMPORTANT: new visual loss, pain with eye movement, or significant color desaturation in one eye need same-day medical evaluation. Some of these conditions have a treatment window measured in days.

What This Means for You

If you've been handed a normal MRI and a normal eye exam while still living with visual symptoms, you have not been given a clean bill of health. You've been given the results of tests that were not designed to measure the thing that's wrong.



Neuro-optometric rehabilitation works on the functional side of this equation. We can't switch off inflammation with lenses and therapy – but we can measure what the visual system is actually doing, reduce the load it's carrying with therapeutic lenses and prism, and retrain the timing and integration that inflammation disrupted. In our experience, that combination is frequently what finally lets patients read, drive, work, and walk through a grocery store again.

Coming in this series:

- **Part 2:** Metabolic inflammation – how diabetes and blood sugar instability inflame the visual system long before diabetic retinopathy appears.
 - **Part 3:** The neurodegenerative conditions – where visual changes in dementia and Parkinson's often precede the diagnosis by years.
 - **Part 4:** Lyme disease, chronic infection, and dysautonomia/POTS – the patients most often dismissed, and often the ones with the most to gain.
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Still Having Visual Symptoms After a “Normal” Exam?

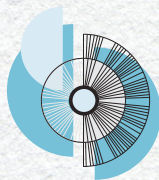
A neuro-optometric evaluation at NVANT looks at the parts of your visual system a routine exam doesn't test – eye teaming, focusing, tracking, visual-vestibular integration, and, where indicated, objective VEP and EEG/ERP electrophysiology. If what you've read here sounds like your experience, we'd welcome the chance to take a closer look.



REFERENCES

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This document does not contain medical advice.



Have A Question? Let Us Know!
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