

Some Thoughts on Oscillopsia: A Perspective

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Oscillopsia refers to the “illusory movement of the world”. It is found in a number of neurological conditions, such as stroke,¹⁻³ superior oblique myokymia,⁴ and multiple sclerosis.⁵ Oscillopsia occurs when there is a mismatch, or disconnect, between the underlying neurological control/command signal to the brain to move the eyes (i.e., corollary discharge) and the actual eye movement and related retinal-image motion.

Patients have described the perception of oscillopsia as “shaking of the world,” “like having a non-functional ‘vertical hold’ on a television” (with vertical oscillopsia in stroke), or having an “oscillating” visual world. It

sometimes produces nausea, dizziness, difficulty navigating busy surrounds, and imbalance. It can also cause general frustration and considerable annoyance, as well as safety issues, for example its occurrence while ambulating or driving.

As disturbing as oscillopsia is, it is possible to use it clinically in a positive manner. Oscillopsia provides a source of additional and immediate “visual feedback” related to the underlying aberrant ocular movements that can be harnessed in a beneficial way.

We have used oscillopsia to assist positively in many patient’s oculomotor control. We would like to share four interesting cases.

In the first case, there was acquired pendular, horizontal, nystagmus, and oscillopsia, secondary to a moderate traumatic brain injury with extended coma.⁶ He reported difficulty fixating objects, problems with visual scanning, and annoyance with the constantly moving environment. Oculomotor-based auditory feedback was the primary therapeutic approach, in which one ‘hears’ their abnormal eye movements. The adult patient was instructed to ‘do something’ to reduce the oscillopsia amplitude and frequency, as well as the correlated tonal modulation (“reduce the jerkiness of the tone, smooth it out”) during the training sessions. This ‘something’ was the following: under binocular-viewing conditions, he ‘imagined’ that he was ‘looking’ with his right (dominant) eye only, and this became his “higher-level control trigger mechanism”. Thus, in this unusual case, the patient had ‘normal’ visual feedback, superimposed perceptual oscillopsia-based abnormal visual feedback, and the veridical oculomotor-based auditory feedback, all providing both visual and auditory perceptual information that had the potential to be used to control his nystagmus volitionally. He learned to do so in a matter of a few short (30-45 minutes) but intense sessions in our eye movement biofeedback clinic. He had correlated tonal and visual information available that could be used to develop and

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exert higher-level control processes to reduce his nystagmus, and in turn his oscillopsia, which he could also readily do and appreciate visually at home. In addition, oculomotor-based vision therapy techniques were performed at home, including fixation and saccade procedures, as well as relaxation training to reduce the nystagmus intensity.

The second case involved a 40-year-old woman restricted to a wheelchair with multiple sclerosis (MS) and bilateral rotary nystagmus, with rotary oscillopsia, and ambulatory and typing difficulties, as her main symptoms. Her goal was to type once again. This was a more complicated case, as the oculomotor effects and the correlated oscillopsia related to the MS waxed and waned over the several month period that she was trained in our clinic. Training included fixation and saccade procedures, as well as relaxation approaches to reduce the nystagmus intensity, along with lid-based tactile feedback related to the nystagmus movement. At times, the oscillopsia was reported reduced, but a few weeks later, it worsened, in a repeated cycle. Therefore, success varied.

The third case involved superior oblique myokymia,⁴ which is believed to be due to a peripheral irritation of the trochlear nerve in one eye. The patient reported intermittent (10-20 times per day for a few minutes duration), unilateral, oblique oscillopsia throughout the visual field. Her primary symptom was unsteadiness walking during a transient attack of oscillopsia. Here, the unaffected eye had normal vision and visual feedback, whereas the affected eye had reduced vision and abnormal visual feedback, namely the oscillopsia. In this scenario, there was the superimposition of normal and abnormal vision, and normal and abnormal visual feedback, both obliquely and normally-oriented – certainly a confusing visual-perceptual state for the individual! The technique that worked best was the use of peripheral awareness during such an attack. The patient was instructed to try to direct/bias her general/visual attention into the periphery, so that the more troublesome central

oscillopsia was less noticeable, during an attack. The patient found this approach to be helpful in her normal work and home environment. Other training techniques included oculomotor-based vision therapy, such as fixation, pursuit, and saccade procedures, as well as relaxation to reduce the nystagmus. In addition, she used a strategy of remaining still during the actual attack, if possible.

The last case involved oscillopsia secondary to Wallenberg's syndrome (i.e., stroke) in an older individual¹⁻³ with the objectively-recorded horizontal, saw-tooth nystagmus typical of a vestibular origin, and related oscillopsia, lasting a few months after the incident. No oculomotor/vision training was implemented for a range of reasons, but it likely would have been helpful. The natural history of the visually-related symptoms and signs was followed over a 13-year period. The patient expressed having trouble navigating sidewalks, especially curbs, due to the oscillatory movements. Interestingly, although the oscillopsia and nystagmus were no longer evident months later under normal viewing conditions with typical ambient illumination, the nystagmus reappeared immediately and consistently, with repetition, when tested in near total darkness! This demonstrates the power and effectiveness of visual feedback as a natural, self-'training' tool to *totally* dampen the nystagmus. We speculate that this may also be the case in both acquired *and* in some cases of congenital nystagmus, when visual feedback is reduced/degraded. For example, we have tested a few patients with documented congenital nystagmus where oscillopsia is not expected to be present. However, in a reduced cue environment, with a reduced visual frame-of-reference (such as a very dark movie theater or dark roadway), intermittent oscillopsia is reported.

We believe that all cases of nystagmus with oscillopsia should be assessed and trained with the full armamentarium of clinical tools at one's optometric command:⁷ auditory and visual feedback approaches (e.g., afterimages),

oculomotor-based vision therapy, base-out vergence prisms to reduce the nystagmus, yoked prisms to reduce the head turn, contact lenses to provide proprioceptive feedback via additional trigeminal nerve stimulation, tactile feedback using the patient's index finger held gently against the eyelid to 'feel' the nystagmus intensity, relaxation techniques to reduce the nystagmus, and perhaps even extending medically to acupuncture and pharmacotherapy.⁸

These are challenging patients, but the rewards are great.

REFERENCES:

1. Werner DL, Ciuffreda KJ, Tannen B. Wallenberg's syndrome: a first-person account. *J Am Optom Assoc* 1989; 60(10): 745-747.
2. Tannen B, Ciuffreda KJ, Werner DL. A case of Wallenberg's syndrome: ocular motor abnormalities. *J Am Optom Assoc* 1989; 60(10): 748-752.
3. Clavero M, Ciuffreda KJ, Werner DL, Tannen B. A 13-year follow-up of a case of Wallenberg's syndrome: first-person account and ocular motor abnormalities. *Optometry* 2002; 73(11): 694-699.

4. Birnbaum MH, Ciuffreda KJ, Tannen B. Peripheral awareness in the treatment of an unusual neuromotor vision disorder. *J Optom Vis Dev* 1994; 25: 253-258.
5. Costello F. Vision disturbances in multiple sclerosis. *Semin Neurol* 2016; 36(2): 185-195.
6. Ciuffreda KJ, Goldrich SG, Neary C. Use of eye movement auditory biofeedback in the control of nystagmus. *Am J Optom Physiol Opt* 1982; 59(5): 396-409.
7. Ciuffreda KJ, Tannen B, Rutner D. Multisensory feedback therapy for oculomotor dysfunction. In: *Models of the Visual System*, (eds.) Hung GK, Ciuffreda KJ. Kluwer Academic/Plenum Publishers, New York, NY, 2002, 741-769.
8. Thurtell MJ, Leigh RJ. Treatment of nystagmus. *Curr Treat Options Neurol* 2012; 14(1): 60-72.



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