Eye movements, visual acuity and spatial constancy

L. F. Dell'Osso, Ph. D.

Ocular Motor Neurophysiology Laboratory, Veterans Affairs Medical Center,
and the Departments of Neurology, Case Western Reserve University
and University Hospitals of Cleveland, Cleveland, Ohio

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Abstract

How are eye movements related to acuity or to the lack of spatial constancy, oscillopsia (OSOP)? How do subjects with congenital nystagmus (CN) suppress OSOP? Can we apply their strategies to cases of acquired nystagmus?

In normals, the maintenance of target foveation with low retinal slip is thought to be necessary for good visual acuity. Retinal slip velocities below 1.67-4 deg/sec have been given as upper bounds for good vision. Subjects with CN do not usually have OSOP and can have good (even normal) visual acuity. CN subjects can maintain target position (SD = 0.21 deg) and low retinal slip velocities (SD = 1.97 deg/sec). Previously, we identified two possible mechanisms for the suppression of OSOP in subjects with CN : 1) efference copy of the CN waveform to negate the effects of the oscillation or 2) the stable vision available during foveation periods.

A 48 year-old man with hereditary CN lost consciousness; when he came to, he had troublesome, intermittent OSOP. Recordings revealed a jerk left with extended foveation (JLef) waveform when OSOP was absent (SD of eye position was 0.24 deg and SD of retinal slip velocity was 1.87 deg/sec). However, with the onset of OSOP, his CN waveform abruptly changed to jerk right (JR) and was biased several degrees to the right of the target. Phase-plane analysis revealed that, during the periods of OSOP, the JR waveform did not enter the foveation window defined by the limits, 0 ± 0.5 deg and 0 ± 4.0 deg/sec. CN direction shifts, common in some CN subjects, do not normally result in loss of foveation periods or OSOP.

During electronic retinal image stabilization (RIS) transient OSOP persisted with the same CN waveform changes. RIS usually results in OSOP that can be suppressed by the CN subject. This subject also suppressed OSOP during his normal JLef waveform but was unable to do so when the biased, JR waveform appeared.

The efference-copy mechanism is tenable only if the man's pathology occurred at a point beyond where the efference-copy signal is fed back.

Stable vision by itself is insufficient since RIS causes OSOP in CN. We conclude that the mechanism that prevents OSOP requires the motor stability provided by the CN foveation periods. Without them, OSOP is not suppressed even during RIS.

If we presume that CN subjects make use of existing mechanisms for preserving the sense of spatial constancy (even allowing that they may have optimized these mechanisms), the suppression of OSOP in cases of acquired nystagmus should be possible if we can provide a foveation window during each nystagmus cycle.

Key words : acuity, congenital nystagmus, fixation, oscillopsia.

Introduction

Eye movements are intimately related to both visual acuity and the sense of spatial constancy or, the absence of oscillopsia (OSOP). The exploration of this relationship is the subject of this paper. Specifically, how do individuals with congenital nystagmus (CN) achieve good acuity ? What mechanism(s) are used in CN to suppress OSOP ? Finally, can the mechanism(s) used in CN be used by those with acquired nystagmus ? In studies of normals, Westheimer & McKee found that retinal image motion greater than 2.5 to 3.5 degrees per second resulted in a decrement in visual acuity. Murphy found motion greater than 1.67 degrees per second resulted in a decrement in contrast sensitivity and Barnes & Smith found motion greater than 3 to 4 degrees per second caused a 10% decrement in acuity.

These figures for normals raise the question, what are the foveation dynamics in CN and how are they related to visual acuity ? If one looks at the position tracing of a cycle of CN, the foveation period can be identified as that period of time during which the eye position is within 30 minutes of arc of the target. In the velocity tracing, the foveation period is that period of time during which eye velocity is less than 240 minutes of arc per second. Plotting eye position versus eye velocity yields a phase-plane portrait. On the phase plane, a foveation period appears as a cusp about the zero-position, zero-velocity point.

We examined the foveation periods in five-second intervals of fixation for a subject with CN by using phase-plane analysis. The analysis of several such five-second records of fixation produced a standard deviation (SD) for mean foveation position of 0.21 degrees and for retinal slip velocity, 1.97 degrees per second. There were repeatable one-second intervals during which the foveation position accuracy was within *one minute of arc* and the phase planes revealed perfectly superimposed cusps. We concluded from this that the fixation mechanism in individuals with CN was not only active but

also accurate. Position and velocity histograms for five-second records revealed the majority of the data clustered about zero minutes of arc and zero minutes of arc per second respectively.

Examination of eye movement data taken while the subject was wearing compound prisms revealed a significant decrease in the amplitude of the CN. The position histogram without prisms showed data clustered between + 30 and – 90 minutes of arc whereas, with prisms the histogram showed virtually all the data between ± 30 minutes of arc. The SD of the mean of the foveation periods while wearing compound prisms was 12.73 minutes of arc. Despite the horizontal CN, the data in the vertical plane revealed a SD of approximately five minutes of arc, which is comparable to normal data. Thus, an individual with CN can have normal vertical fixation stability and very accurate and repeatable horizontal target foveations on a beat-to-beat basis.

In an individual with an oscillation such as CN, visual acuity is proportional to three things : 1) the foveation time per cycle ; 2) the retinal slip velocity during foveation ; and 3) the beat-to-beat stability of foveation or "jitter". Using the data gathered from a subject with CN at various gaze angles, we devised a nystagmus foveation function (NFF) and plotted it versus gaze angle. This NFF was equal to the foveation period per cycle (in msec) multiplied by the nystagmus frequency (in Herz) to give the fraction of foveation time (unitless) divided by product of the standard deviations of the mean foveation-period position (in deg) and mean foveation-period velocity (in deg/sec). The resulting NFF (in sec/deg^2) is a function with a much sharper peak in the null region than the classically used nystagmus intensity function. Furthermore, the NFF reached even greater values when plotted for the data taken during the use of compound base-out prisms. The NFF is a better predictor of the gaze angle of highest visual acuity than the intensity function is for a given subject and furthermore, should be able to predict acuity on an intersubject basis whereas, the intensity function cannot.

The next question we ask of CN subjects is, why do individuals with CN not complain of OSOP ? In subjects with acquired nystagmus the moving retinal images result in a perception of OSOP. In individuals with CN, the same moving retinal images result in a perceptually stable world. What is the difference and how is it related to eye movements ?

We identified five possible mechanisms that might be employed by the individual with CN to suppress OSOP. They are : 1) a decreased threshold for OSOP due to afferent defects ; 2) the acquisition of visual information only during foveation periods ; 3) an extra-retinal signal that cancels out the nystagmus oscillation ; 4) an elevated threshold for motion detection due to the oscillations ; and

5) suppression from the quick phases that reduces the motion detection threshold. To better understand the suppression mechanism operable in CN we asked, what would be the perception of an individual with CN to retinally stabilized images ?

We studied four subjects (two female, two male) in the age range of 23 to 45 years old whose best corrected visual acuity was 20/30 or better. They did not have strabismus. Their waveforms were, one pendular and three jerk. Their foveation periods were well developed. We used three different methods to stabilize images : 1) retinal afterimage ; 2) optically stabilized retinal images ; and 3) electronically stabilized retinal images.

The optical method made use of a high-minus contact lens that focused all images at the center of rotation of the eyeball (making them insensitive to eye motion) combined with a high-plus spectacle lens that focused the images on the retina. Electronic stabilization was accomplished by making use of the eye motion signal picked up by a retinal scleral search coil and using it to drive a mirror galvanometer that moved the target image in synchrony with the eye movements. Afterimages were placed on the subjects' retinas both in the dark and with an LED target visible. Optical stabilization was tested with both an LED target and in a structured background. Electronic stabilization was of an Amsler grid subtending a 20 by 20 degree square. Subjects were asked if the afterimage was moving or if the stationary target was moving and if so, were their directions the same or different.

Of the four subjects in this study, two reported occasional OSOP present in real-world situations. Two subjects reported OSOP of an afterimage in the dark and all four subjects reported OSOP of the afterimage when simultaneously viewing a stationary LED. During optical stabilization of an LED, two of three subjects reported OSOP and two of four reported OSOP of a real-world background. During electronic stabilization, both of the subjects tested reported OSOP.

In several of these situations, one subject found he could suppress the OSOP of the stabilized retinal image but, when this was done, the non-stabilized portion of his visual field suddenly appeared to oscillate. The subject could freely switch between the stabilized image and the non-stabilized surround and impose perceptual stability on either but not both.

Kommerell has shown that individuals with CN had normal ability to estimate velocities of moving targets. However, Brandt and Dieterich have shown that the threshold for motion detection is higher in the periphery than in the central retina and that this elevation is greater in CN subjects than in normals.

With regard to the possible mechanisms for maintaining stability in CN, our data allowed us to eliminate three. We found no afferent

defects in these patients, eliminating the first mechanism. Since the afterimage oscillated and the stationary LED did not, visual information must have been present throughout the CN waveform, ruling out the second possible mechanism. Since one of our subjects had pendular CN, suppression from quick phases was not a possible mechanism. The most likely mechanism for the suppression of OSOP of stationary targets and inducing OSOP of stabilized images is one that uses an extra-retinal signal to cancel the CN oscillation. This same mechanism has been hypothesized to be necessary for extracting true target motion and using that information for smooth pursuit. This model of efference copy of the CN waveform that I postulated in the mid-1960's was recently summarized in a block diagram where the efference copy was fed back in an internal feedback loop to cancel out that retinal motion induced by eye movement while allowing retinal motion produced by target motion to be detected. Our recent studies led us to conclude that, in addition to an extra-retinal signal that cancels the effects of the nystagmus oscillation, other mechanisms may also play a role in suppressing OSOP in some patients.

subjects with CN can accurately foveate targets during foveation periods with a SD of approximately .21 degrees and can maintain low retinal slip velocities during these foveation periods with a SD of 1.97 degrees per second for five seconds of fixation. We found that a variety of mechanisms operate to maintain spatial constancy in CN and some individuals appear to utilize one mechanism more than others. The possible mechanisms include the utilization of extra-retinal signals (efference copy, proprioception), elevated threshold for motion detection and possible suppression of visual input if images drift off the fovea.

To further understand how OSOP is suppressed in CN we studied a subject who had transient OSOP and hereditary CN. He was a 48 year old male with strabismus who lost consciousness six to seven years prior to our seeing him during a fasting attempt to lose weight. Upon regaining consciousness, he noted periods of horizontal OSOP and vertical diplopia. Artane was said to slow the OSOP at some point during his treatment.

Our examination revealed CN with jerk and jerk with extended foveation waveforms. He preferred right-eye fixation ; his left eye was exotropic. With left-eye fixation, his right eye assumed an exotropic and hypertropic position. He had spontaneous changes in his horizontal CN from jerk left with extended foveation to jerk right and the jerk right always correspond to the periods of OSOP. Spontaneous changes in CN direction (called bias reversals) does not usually produce OSOP in the CN subject. We studied ten-second intervals of fixation in this subject.

We recorded the horizontal and vertical movements of both eyes using scleral search coils. Analysis consisted of constructing phase portraits of the final jerk right cycle in a run of OSOP followed by the first three jerk left with extended foveation cycles during which time no OSOP was present. The right eye was the fixing eye and the eye that we analyzed.

The phase portrait revealed that during all three jerk left with extended foveation cycles, the foveation period fell within the foveation window defined by $\pm$ 30 minutes of arc position and $\pm$ 240 minutes of arc per second velocity limits. However, the jerk right waveform never entered the foveation window.

We repeated this analysis for ten seconds of fixation during retinal image stabilization. The phase portrait of a similar four cycles (one jerk right followed by three jerk left with extended foveation) revealed the same picture. That is, all three jerk left with extended foveation cycles entered the foveation window and the jerk right cycle did not.

When we looked at the phase portraits of the full ten seconds of fixation under conditions of both normal fixation and of retinal image stabilization we found that all of the jerk left with extended foveation waveforms entered the foveation window whereas, none of the jerk right waveforms did so. Thus, the horizontal OSOP was directly related to whether or not the waveform allowed a foveation period to fall within the foveation window.

When we looked at phase portraits of vertical motion we found that during both ten-second intervals (normal fixation and retinal image stabilization) both waveforms produced foveation periods well within the foveation window. No vertical OSOP was reported during either the jerk right or jerk left waveforms. Looking at the left eye data revealed a transient vertical tropia during the jerk right intervals ; this accounted for his vertical diplopia during the horizontal OSOP.

Summarizing, the foveation periods of the jerk left with extended foveation waveforms during ten seconds of fixation produced a position accuracy with a SD of 0.24 degrees and a velocity accuracy with a SD of 1.87 degrees per second. These values are comparable to those of the subject we previously studied who did not have OSOP. We concluded that efference copy alone cannot prevent OSOP unless the additional pathology in this case occurred at a point after the efference copy signal is fed back (the CN waveform changes should not have mattered if efference copy was the sole mechanism involved). Stable vision alone cannot prevent oscillopsia since retinal image stabilization did not abolish it. Motor stability is required to prevent OSOP since only with foveation periods that fell within the position and velocity limits for good vision was it possible for this subject to perceive a stable world.

I conclude the presentation with the following question, can we provide those with acquired nystagmus the same mechanism(s) used to suppress OSOP in CN and can they be successfully employed ?

ACKNOWLEDGEMENTS

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L. F. DELL'OSSO,
Departments of Neurology,
Case Western Reserve University,
University Hospitals of Cleveland,
Cleveland, Ohio (USA).