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Oscillopsia, Retinal Image Stabilization and Congenital Nystagmus

R. J. Leigh,*†§ L. F. Dell'Osso,*§ S. S. Yaniglos,‡ and S. E. Thurston*§||

Most individuals with congenital nystagmus (CN) do not complain of oscillopsia (visual inconstancy) even though the amount of retinal image slip varies considerably according to gaze angle and CN waveform. We induced oscillopsia in four subjects with CN by artificially stabilizing images upon the retina under several conditions. Every subject reported oscillopsia during retinal image stabilization, but the condition of stabilization varied from one individual to another. Our results indicate that a variety of mechanisms operate to maintain spatial constancy in congenital nystagmus; some individuals appear to use one mechanism more than another. Possible mechanisms include use of extra-retinal signals, elevated threshold for motion detection and "suppression" of visual input except during foveation periods. Invest Ophthalmol Vis Sci 29:279–282, 1988

Ordinarily, eye movements prevent slip of images upon the retina from exceeding about 4 degrees per second. If retinal image velocity (RIV), commonly called, "retinal slip," exceeds 4 degrees per second, then visual acuity begins to decline[1,2] and oscillopsia —an illusory movement of the stationary world— may result.[3] Impaired vision and oscillopsia are common consequences of acquired nystagmus. While the relationship between RIV and visual acuity is a direct one, this is not the case for retinal slip and oscillopsia. Thus, the magnitude of oscillopsia cannot be directly related to RIV; for example, in acquired downbeat nystagmus the magnitude of oscillopsia was found to be 0.37 of the nystagmus magnitude.[4] Another example of this is congenital nystagmus (CN) in which oscillopsia is seldom a complaint despite slow phase RIV that may exceed 100° per second. By studying the mechanisms that maintain visual constancy in CN it may be possible to elucidate the relationship between RIV and oscillopsia in acquired nystagmus.

Several possible mechanisms may account for the absence of oscillopsia in association with CN. First, coexistent afferent visual defects might decrease the threshold for oscillopsia. Second, visual information might only be accessed during foveation periods (when there is minimal retinal image motion) and at other times would be "suppressed." Third, an extra-retinal signal could be used by the brain to cancel out those effects on vision due to the oscillations (see Howard for a review).[5] Fourth, an elevated central threshold for the detection of motion might help to suppress oscillopsia.[6] Fifth, the presence of quick phases may, like voluntary saccades, reduce the perceptual threshold for detecting target movement.[7]

We sought to determine which of these possible mechanisms was operating in CN by artifically stabilizing images upon the retina. Of the hypothetical mechanisms summarized above, only the explanation which entails use of an extra-retinal signal would predict that retinal image stabilization should produce oscillopsia in CN. We found that subjects with CN did experience oscillopsia when images were artificially stabilized upon the retinas, but that the conditions for this varied from individual to individual.

Materials and Methods

We studied four individuals with CN, all of whom had given informed consent; their ages ranged from 23 to 45 years. All subjects had visual acuity of 20/30 or better, none had strabismus, one had a pendular form of nystagmus and the other three had jerk nystagmus. All showed waveforms with well-developed foveation periods. One subject (S1) was knowledgeable about eye movements and the purpose of the experiments. No normal subjects were systematically studied although we stabilized normals using both the optical and electronic methods described below.

From the *Ocular Motor Neurophysiology Laboratory and †Neurology and ‡Optometry Services, Veterans Administration Medical Center Cleveland, Ohio; and the §Departments of Neurology, Case Western Reserve University and University Hospitals of Cleveland, Cleveland, Ohio.

|| Present address: The Department of Neurology, Geisinger Medical Center, Danville, Pennsylvania.

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Reprint requests: L.F. Dell'Osso, PhD, Ocular Motor Neurophysiology Laboratory, Veterans Administration Medical Center (127A), 10701 East Boulevard, Cleveland, OH 44106.

Table 1. Conditions causing oscillopsia in congenital nystagmus

Condition	*Oscillopsia present*	*Oscillopsia absent*
Real world	S3*, S4*	S1, S2
After-image		
—in dark	S1†, S2	S3, S4
—viewing LED	S1, S2, S3‡, S4*	
Optical stabilization		
—viewing LED	S1†, S2	S4
—viewing laboratory	S1†, S3	S2, S4
Electronic stabilization	S1†, S3*	

* Intermittent oscillopsia (see text).
† Could suppress oscillopsia.
‡ In opposite directions.

We used three methods to stabilize images upon the retina and under each condition subjects were asked whether the visual stimulus or stimuli were stationary or moving. The first method was a parafoveal photo-flash after-image applied monocularly; it subtended a visual angle of approximately 1°. The after-image was first viewed in a completely dark room and then with a solitary light emitting diode (LED) in an otherwise dark room. Two subjects reported on the effects of an after-image in an illuminated room. Second, we used an optical method to stabilize retinal images as described by Rushton and Rushton.[8] Briefly, the system consists of a high positive spectacle lens and high negative contact lens. The spectacle lens focuses rays of light from an object close to the center of rotation of the eyeball. Thus, if the eyeball rotates, light rays from the object are focused at the same point in the eye. To extend back the focus from the center of the eyeball to the retina requires optics that move with the eye and this is accomplished using a high negative contact lens. This device is proven to be an effective treatment for oscillopsia in certain patients with acquired nystagmus.[9] Our subjects wore a combination of +32D spectacles and −58D contact lens to achieve over 90% stabilization of retinal images. The contact lens device was placed on one eye and the other eye was occluded. With the device the field of view is about 30° and this is surrounded by a ring scotoma. Unstabilized vision is present in the far periphery. While wearing the device our subjects first viewed a solitary LED in a dark room and then looked out of the laboratory window. The third method that we used to stabilize images upon the retina was electronic feedback. We recorded eye movements using the magnetic search coil technique (rotating phaser system) and then fed the horizontal eye position signal through amplifiers to drive our galvonometers and so control the location of an Amsler grid subtending 20° × 20° on a tangent screen in front of the subject. This stabilization system had a linear range of ±10° and a bandwidth of 0–40 Hz. This bandwidth is more than adequate for the slow phases of nystagmus (frequencies of 2–5 Hz) that cause the perception of oscillopsia. When this system has been used during visual acuity testing on subjects with nystagmus, acuity has either increased or remained unchanged when images were stabilized. The scleral contact lens coils were precalibrated before insertion into the subjects' eyes. Our system provides accurate and stable calibration independent of the subject or any ocular oscillations present. In all tasks, fixation of either a target or an after-image was required of the subject.

Results

The results for all subjects (S1–S4) are summarized in Table 1. On direct questioning, two of our subjects (S3 and S4) reported occasional oscillopsia during *normal viewing;* in one (S3) this was related to the gaze angle in which nystagmus was maximal. With an *after-image* in the dark, two subjects (S1 and S2) reported oscillopsia, two did not. S1 was able to suppress oscillopsia of the after-image with voluntary effort. With a background LED, all reported oscillopsia, two (S1 and S2) of the after-image, one (S4) of the LED and one (S3) of both after-image and LED with motion in opposite directions. When the room was illuminated, S1 and S2 reported oscillopsia of the after-image; for S2 the movement was greater in the lighted background.

With the *optical device* two subjects (S1 and S2) reported oscillopsia with the LED, one (S4) did not. The fourth individual (S3) was not tested in this way. As with the after-image, S1 could suppress oscillopsia of the LED. When looking through the laboratory window with the optical device, two individuals (S1 and S3) reported oscillopsia of the central field and two did not. Although S1 reported oscillopsia of the stabilized central field, the peripheral surround was perceived as not moving. S1 could reverse the perceptions, causing oscillopsia of the surround rather than the central field.

With *electronic stabilization* in two subjects (S1 and S3), both reported oscillopsia of the stabilized field of vision; however, S1 could easily suppress this with voluntary effort (Figure 1) and in S2, oscillopsia was gaze-dependent, being present in the direction in which the nystagmus was maximal. Neither subject knew what degree of electronic stabilization was being used at any given instant but, by the perception of oscillopsia, could appreciate when stabilization

was turned on. S1 noted that the unstabilized visual periphery was perceived as stable when there was oscillopsia of the stabilized central field and vice-versa. During the periods when the S1 reported oscillopsia, the frequency and amplitude of the nystagmus changed compared with those periods when oscillopsia was suppressed. When visual acuity was measured in S1 during electronic stabilization and S2 during optical stabilization, no significant change was noted.

Discussion

It is well known that individuals with congenital nystagmus (CN) do not usually complain of the illusory movement of their visual world (oscillopsia). Less than ten of the more than 450 CN subjects tested in our Laboratory have spontaneously reported oscillopsia. Despite substantial retinal motion caused by their own ocular oscillations, they perceive a stable world and properly interpret real motion of elements within that world, despite the large range of CN amplitudes, frequencies and waveforms present at different times in a given individual and foveation periods when retinal motion is reduced to zero by temporary cessation of ocular motion. The two subjects of this study who did report occasional oscillopsia under specific conditions are not representative of the general population of CN subjects. It is probable, however, that more direct questioning about specific conditions of fixation would elicit more positive responses about the presence of oscillopsia.

We have found that artificial stabilization of images of stationary objects on the retina in individuals with CN may cause oscillopsia. This paradoxical effect was variable both from individual to individual and in terms of the method of stabilization that produced oscillopsia, suggesting that a variety of factors contribute to visual stability in CN.

Of the various possible mechanisms that might account for visual stability in CN, only one would seem likely to cause oscillopsia during artificial stabilization of retinal images: the use of an extra-retinal signal to cancel out the effects of the CN on vision. It has been hypothesized that oscillopsia is automatically eliminated by using efference copy of the CN waveform to cancel out the nystagmus-induced retinal motion, leaving target motion to be perceived as such.[10] (Whether this extra-retinal signal is an efference copy or corollary discharge of the nystagmus, or rather, extraocular proprioception, will not be discussed here; recent reports favor the former).[11]

When an after-image is flashed onto the fovea of a normal subject the perception is one of a stable after-image superimposed on the stable fixation target. If

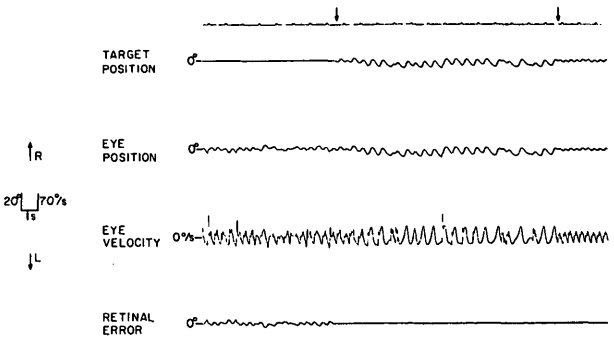

Fig. 1. Eye movements of subject S1 during electronic stabilization (beginning at first arrow). At second arrow S1 reported that the retinally stabilized field was perceived as stable. R—right, L—left and timing marks are at 1-second intervals.

the subject moves his eyes, the perception is that of an after-image "moving with the eyes" over the stable surround.[12] The perception of after-image oscillation over stationary targets, made by three of the four subjects (S1, S2 and S3), is equivalent to that of normals since our subjects' eyes were constantly moving. They correctly perceived the targets as stationary and the after-images as moving despite the retinal motion of the former and retinal stability of the latter.

Could our subjects' reports of oscillopsia during retinal image stabilization be due to relative motion between a stabilized central retinal image and an unstabilized peripheral retinal image? With the optical device, the far periphery of vision is unstabilized and during electronic stabilization the edge of the tangent screen was visible, though at low luminance. The threshold for detecting motion in the retinal periphery is elevated compared with the central retina and this elevation is greater in CN.[6] Thus, relative motion between central and peripheral retinal images seems unlikely to be the main mechanism for oscillopsia during optical or electronic stabilization. However, S1, an experienced observer, was able to transiently reverse the percept so that the central image was stable and the visual periphery was oscillating. During this "reversal" his nystagmus frequency changed, a finding that deserves further study in other subjects; S3 did not show a change in nystagmus frequency.

In two subjects, oscillopsia was reported with an after-image while in darkness, a situation in which there is no conflict between central and peripheral cues. Recently, Kommerell has reported on the effects of parafoveal after-images in seven subjects with CN.[13] Five of the seven reported oscillopsia though the magnitude of the oscillopsia was only 50% of the nystagmus. This finding implies that if the brain does use an extra-retinal signal to cancel out the effects of nystagmus on vision, this cancellation is only partial. Such an assumption is consistent with models of eye

movements that embody an efference copy mechanism the gain values of which are less than one, typically 0.6.[14] If this is so, then—as suggested by our results—more than one mechanism may be operating to maintain visual constancy in CN. What other methods are possible?

Impairment of vision cannot be held responsible for visual constancy in CN since many individuals have normal, or near-normal, visual acuity. However, as mentioned above, individuals with CN have increased thresholds for motion detection. The mechanism for this is unclear, though it is possible that quick-phases of nystagmus, when present, may contribute in the same way that voluntary saccades increase the threshold for detection of stimulus displacement.[7]

Finally, our results do not exclude the possibility that visual information is "suppressed" at all times except during foveation periods when retinal image slip is minimal. Matin et al have shown the visual smear due to voluntary saccades is probably omitted from vision by a process of backward masking.[15] Perhaps a similar mechanism might occur during the high-velocity slow phases of CN.

Our findings suggest new experiments that might be used to settle the relative roles of the various proposed mechanisms for suppressing oscillopsia in CN. By presenting stabilized visual stimuli at specific points in the CN waveform under stabilized or unstabilized conditions, it should be possible to determine how much visual suppressive mechanisms are contributing. It should be noted, however, that it has been shown that small and large field visual stimuli may be perceived quite differently during conditions of retinal image stabilization.[16] Our results do indicate that more than one mechanism may be involved in suppressing oscillopsia in CN and that individuals vary as to which mechanism is being predominantly used.

Key words: oscillopsia, congenital nystagmus, retinal stabilization

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