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THE OCULAR MOTOR CONDITIONS FOR OSCILLOPSIA

INTRODUCTION

Oscillopsia (OSOP) is the subjective, illusory movement of the stationary world (Brickner, 1936). Normal ocular motor control systems can prevent slip of images on the retina from exceeding about 4°/sec. When retinal image velocity (RIV), commonly called «retinal slip», exceeds that value, visual acuity begins to decline and OSOP may result. OSOP is a common consequence of acquired nystagmus but not of saccadic intrusions or oscillations. Also, most patients with congenital nustagmus (CN) do not experience OSOP despite slow phase RIV that may exceed 100°/sec. This subjective feeling of motion of the environment has been associated with acquired nystagmus caused by an Arnold-Chiari malformation (Bell et al., 1987) or even carbamazepine therapy (Chrousos et al., 1987). Reduction of the nystagmus has caused an equal diminution of the OSOP (Pedersen et al., 1980). Since any excessive RIV may be related to OSOP, nystagmus need not be present. A change in the vestibulo-ocular reflex (VOR) gain sufficient to cause high RIV may also result in OSOP. Verhagen et al. (1987) reported three cases in a family with congenital vestibular areflexia. There was no nystagmus in these patients and the OSOP occurred only during head or body movements. OSOP during movement and without nystagmus was also caused by aminoglycoside ototoxicity (Marra et al., 1988).

Although there is a direct relationship between RIV and visual acuity, there isn't one with OSOP. The magnitude of OSOP cannot be directly related to RIV; in acquired downbeat nystagmus the magnitude of OSOP is approximately 0.37 of the nystagmus magnitude. At the far end of the spectrum are those patients with CN in whom there are both high RIV and no OSOP. Brandt and Dieterich (1988) proposed that the dissociation between RIV and

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OSOP can be explained by a combination of two separate mechanisms that involve motion perception. These are: a physiological elevation of thresholds to detect object-motion with moving eyes; and a pathological elevation of thresholds to detect object-motion with either infranuclear ocular motor palsy or supranuclear ocular oscillations. They found that thresholds for egocentric detection of object motion were higher in patients with downbeat nystagmus than in normals and the thresholds increased with increasing nystagmus amplitude. There was a partial suppression of visual motion perception for both the RIV caused by the nystagmus and for objects moving within the visual scene. During smooth pursuit, the motion detection threshold of the visual background increases proportionally with eye velocity. Thus, there appears to be a visual motion perception suppression during eye movements that may reflect a basic sensory-motor mechanism. It has been suggested that the nervous system contains mechanisms whose tuning characteristics in both space and time can be modified and make it possible to do what a camera cannot; resolve form and motion simultaneously and independently (Burr e Ross, 1986). This independence is used clinically by the oscillatory movement displacement threshold (the smallest amplitude of oscillation that causes the perception of movement) to access ocular neural dysfunction in the presence of media opacities (Whitaker e Buckingham, 1987).

In our initial attempt to further understand the relationship between RIV and OSOP, we studied retinal image stabilization in subjects with CN (Leigh et al., 1988). All subjects reported OSOP during retinal image stabilization but the condition of stabilization varied from one individual to another. We concluded that several mechanisms operate to maintain spatial constancy in CN; some individuals appear to use one more than another. The mechanisms used included extra-retinal signals, elevated threshold for motion detection and «suppression» of visual input except during foveation periods. When only part of the visual field was stabilized, one subject reported OSOP of that stabilized central field while the peripheral surround was perceived as not moving. He could reverse the perceptions, causing OSOP of the surround instead of the central field.

Since RIV is related to OSOP in acquired nystagmus, the effects of retinal image stabilization were studied in nystagmus caused by neurologic disease (Leigh et al., 1988). The stabilization was progressively increased in eight patients with acquired nystagmus until OSOP was abolished; this was achieved at RIV of 5°/sec or less. In five patients, further increases in stabilization caused the OSOP to reappear in the opposite direction. In addition to reducing OSOP, stabilization improved visual acuity in 4 of 5 patients tested. Both electronic stabilization and an optical device were used to stabilize images of the real world on the retina (Rushton e Rushton, 1984; Rushton e Cox, 1987). Although the optical device provides stabilization over a small central field, it is useful in helping patients to read or watch television by reducing or eliminating their OSOP.

Since the identification in the early 1970's of the foveation periods in CN waveforms, their importance in visual acuity was obvious. All therapies for CN have stressed reduction of the nystagmus waveform intensity with the hoped-for result that the foveation-period durations would be increased. What was less appreciated was the importance of stable, on a beat-to-beat basis, foveation periods in the suppression of OSOP in subjects with CN. This was partially due to the early hypothesis that OSOP was suppressed in CN by efferent copy of the CN waveform (Dell'Osso, 1968). Based on the premise that the best way to study OSOP in normals is to understand its suppression in those with CN, we have begun to study this phenomenon. Inherent in this premise is the hypothesis that the mechanisms employed by subjects with CN to suppress OSOP do *not* represent specially developed abilities but rather result from the application of the normal capabilities present in the ocular motor system. If this hypothesis is true, the subject with CN becomes an excellent *model* for the study of normals who acquire OSOP due to neurological deficit in later life and, once the mechanism employed by subjects with CN is understood, we should be able to therapeutically apply this knowledge to subjects with acquired OSOP.

Case 1

We studied the waveform changes in a subject with hereditary CN who experienced intermittent OSOP after an episode of loss of consciousness (Dell'Osso e Leigh, 1990, 1991a). Using the same type of phase-plane analysis employed to study foveation periods in CN, we found that his normal horizontal CN fell into a foveation window defined by $0 \pm 0.5°$ and $0 \pm 4°$/sec limits on eye position and velocity respectively. However, when he complained of OSOP he exhibited a different waveform whose horizontal motion *never* entered this foveation window; his vertical eye movements were so small as to never leave the vertical foveation window. Thus, when his foveation periods fell within this horizontal foveation window on a beat-to-beat basis he was able to suppress horizontal OSOP and when his waveform did not allow foveation periods to fall within the window he could not suppress that OSOP. We performed the same analysis during horizontal retinal image stabilization (RIS) and the results did not change. We concluded from this that RIS by itself was insufficient to suppress OSOP and further that the mechanism that prevents horizontal OSOP in subject with horizontal CN requires the ocular motor stability provided by the CN foveation periods; without it horizontal OSOP is not suppressed even during RIS. Thus, we infer that what is needed to suppress OSOP is a short period of time during which the subject can foveate a target of interest with a low retinal slip velocity on a repeatable basis. In subjects with acquired oscillations, ways must now be found to alter the oscillations in such a way to achieve these periods of stable target foveation.

The above patient had *uniplanar* CN that resulted in transient *uniplanar* OSOP when the stability of his foveation window was insufficient in the horizontal plane. Since both the nystagmus and the OSOP were in the same plane, we could not separate contributions of the nystagmus waveform and the horizontal jitter of the foveation periods to the perceived OSOP.

CASE 2

We next had the opportunity to study a patient with rare *biplanar* CN who began to experience uniplanar or biplanar OSOP depending on which eye she fixated with (Dell'Osso e Leigh, 1991b, 1992). This patient, who was her own control in each plane, provided the data necessary to identify the key role played by foveation-period stability in each plane towards suppression of OSOP in that plane.

She had strabismus surgery at 18 months of age and her CN was diagnosed at age 13. She noted intermittent OSOP during her teens. At age 23, she was given medications (including lithium) for post-partum depression and began to complain of OSOP. She experienced mainly horizontal OSOP when fixating with her right eye and mainly vertical OSOP when using her left eye; she was not binocular and did not complain of diplopia. She also reported that during right-eye fixation, the OSOP could sometimes be diagonal and during left-eye fixation, counterclockwise elliptical.

We found that, in addition to her horizontal CN (presumably present since birth) she had a see-saw vertical component that resulted in a diagonal nystagmus of both eyes. The fixating eye (right or left) beat downward and nasally while the other beat upward and temporally. In the horizontal plane, this was a *reversed* latent component. The nystagmus of the right eye was diagonal with equal horizontal and vertical components while that of the left was elliptical with a large horizontal and small vertical component.

We employed phase-planes (position vs. velocity plots in a given plane), scan-paths (horizontal vs. vertical position or velocity plots for a given eye) and conjugacy plots (left eye vs. right eye position for each plane) to identify the dynamics of the foveation periods in each plane during fixation with each eye. These biplanar analyses of her waveforms revealed that during right-eye fixation, there was a horizontal position instability of the foveation periods on a beat-to-beat basis. Occasionally, a vertical foveation-period instability accompanied the horizontal instability; it never appeared alone. During left-eye fixation, there was a vertical velocity instability of the foveation periods. Occasionally, a horizontal foveation-period instability was also present; it too, never appeared alone.

Conclusions

Thus, despite a *biplanar* CN in either fixating eye, a predominantly *uniplanar* OSOP resulted that was correlated with a foveation-period instability in the same plane and unrelated to the predominant plane of the CN oscillation. Only when there were foveation-period instabilities in both planes simultaneously, was the OSOP determined by the actual excursions of the CN waveforms. What emerged from our analysis was the conclusion that planar stability of the foveation period was the determining factor in whether OSOP was suppressed or not. Comparing the data from these two rare CN patients to that of a subject with idiopathic CN and no OSOP we concluded that the presence of well-developed foveation periods in each plane was the necessary and sufficient condition for perceptual stability in that plane and the absence of same would result in OSOP in either or both planes.

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