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The mechanism of oscillopsia and its suppression

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We studied the mechanisms of oscillopsia *suppression* in subjects with infantile nystagmus syndrome, fusion maldevelopment nystagmus syndrome, and acquired nystagmus (AN). Hypothetical possibilities for perceptual stability were the following: (1) epochs of clear and stable vision during foveation periods of nystagmus waveforms; (2) cancellation by efference copy of motor output; (3) a combination of the effects of both foveation-period stability and efference-copy cancellation; or (4) elevated motion-detection threshold and vision suppression. Observations, studies, and models of oscillopsia suppression allowed comparison of these possibilities. Data from individual subjects supported some of the putative hypotheses. However, only one hypothesis remained viable that could explain how *all* subjects maintained perceptual stability despite their different nystagmus types, waveforms, and variability. Robust suppression of oscillopsia was only possible using efference-copy feedback of the motor output containing these specific nystagmus signals to cancel that motion from the retinal error signals. In cases of AN, where oscillopsia could *not* be suppressed, the deficit was postulated to interfere with or lie outside of this efference-copy feedback loop.

Keywords: oscillopsia; nystagmus; model; efference copy; foveation

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Introduction

Eye movement data-based studies and computer modeling of ocular motor *dysfunction*, especially of infantile nystagmus syndrome (INS), have elucidated many of the mechanisms and functional interconnections governing *normal* ocular motor function. Eye movement recording analysis of ocular motor system (OMS) responses to standard visual stimuli confounded by the impediments caused by internal oscillations has both allowed insights into OMS function not possible from the study of normals alone and uncovered functional mechanisms that better-defined normal OMS control.[1–16] Applying that same approach to studying the mechanisms underlying the *perception* of motion of stationary objects, "oscillopsia" (OSOP), we instead studied the mechanisms of OSOP *suppression* in subjects with INS and fusion maldevelopment nystagmus syndrome (FMNS, another type of nystagmus of infancy), acquired nystagmus (AN), or combinations of these distinct oscillations. As will become evident, perceptual stability or its absence (OSOP) involves sites throughout the brain, including sensory, motor, computational, and higher perceptual areas.

Under normal viewing conditions, subjects with either INS or FMNS do not experience OSOP, while those with AN do. Thus, it is not symptomatic in the former group but may be debilitating in the latter. However, under certain viewing conditions, and in the laboratory, subjects with INS may also experience OSOP.

The hypothetical possibilities were that perceptual stability was mediated by (1) epochs of clear and stable vision during the relatively stable foveation periods of the nystagmus waveforms; (2) cancellation by efference copy of the motor output driving the nystagmus; (3) a combination of the effects of both foveation-period stability and efference-copy cancellation; or (4) elevated motion-detection threshold and vision suppression outside of foveation periods. Although included for

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completeness, the fourth hypothesis fails upon even cursory examination. Elevated motion-detection thresholds are much smaller than the speeds of nystagmus slow phases, and vision is not truly suppressed during slow phases.[17] The saccadic latencies of INS subjects to target jumps during slow phases are normal, indicating their detection within the same time intervals as in normals. Thus, the fourth hypothesis will not be discussed further in this review.

There is a large body of research on the topic of OSOP, and this paper is not meant to be a review of that literature.[18–31] Rather, it is aimed at uncovering and modeling the mechanism of OSOP suppression and is based on four of the author's observations and six studies of OSOP made by the author and his colleagues over a period of approximately 57 years. The bibliographies of these studies contain many relevant studies of OSOP. The approach taken in studying OSOP has its foundation in prior studies of ocular motor dysfunction that elucidated mechanisms of normal function (i.e., the gaze-evoked nystagmus model demonstrated the need for different neural integrators for saccadic generation and eye-position maintenance and that the former was resettable;[3] and studies and models of INS and FMNS demonstrated that "retinal-error" OMS models were too simplistic,[1,9,10,12,13] efference copy and target reconstruction were necessary,[1] proprioception was important to OMS function and INS therapy,[32,33] and inappropriately timed saccades induce smooth-pursuit errors.)[15] Thus, rather than ask, "Why do subjects with AN have OSOP (i.e., what is the mechanism of OSOP)?" we asked, "Why don't subjects with INS or FMNS have OSOP (i.e., what is the mechanism of OSOP suppression)?"

Results

The following is a summary of the observations and studies that form the basis for my conclusions. The methodological and especially analytical details and figures are too numerous to include in this paper; they may be found in the original studies.

Observation 1: a child with INS sees oscillating afterimages of magnesium flashbulbs

In roughly 1945, a young child with INS (the author) noticed that the retinal afterimage he received every time a flashbulb was used to take his picture not only moved with each saccade he made to different parts of his visual field (as it did for his friends with normal eye movements), but also continuously oscillated horizontally away from and back to his point of regard, not across the line of regard, as he would later discover was commonly stated in the medical literature. That latter observation was critical in discovering and documenting the key factor determining improved visual acuity in INS, the *foveation periods* contained in each cycle of the oscillation.[2]

Looking back on that initial observation of afterimage OSOP, it would support the hypothesis that efference copy was responsible for OSOP suppression of the moving images on the retina but could not account for the retinally stable afterimage; i.e., it did not support the foveation-period stability mechanism.

Study 1: an attempt to model OMS responses of a subject with INS

This study was completed in partial fulfillment of the author's obtaining a PhD degree in Electrical Engineering with a Biomedical Engineering option from the University of Wyoming.[1] The object was to simulate the behavioral responses to various common transient target inputs that were measured from a subject with INS. It was demonstrated that a subject with INS exhibited essentially normal responses to these inputs, albeit, with the INS waveform superimposed on them. Thus, saccades and smooth pursuit were normal and not "reversed," as some would later claim. Also, full-field optokinetic stimulation resulted in normal circular vection and optokinetic after-nystagmus; i.e., it too was normal and not "reversed." Subsequent studies of smooth pursuit and optokinetic nystagmus demonstrated that they had normal gains and that the misperception of "reversed" responses was due to a null shift causing the INS to reverse.[5–7,34] The simulation was accomplished using a computer model containing an internal oscillation (to simulate the INS), but it was otherwise a normal OMS model. The inputs applied included pulse, step, ramp, and step–ramp changes in target position.

All models of the OMS at the time (and most still currently in use) were retinal error reflex models; i.e., a retinal error signal was used to directly drive the motor subsystems (e.g., saccadic and pursuit).

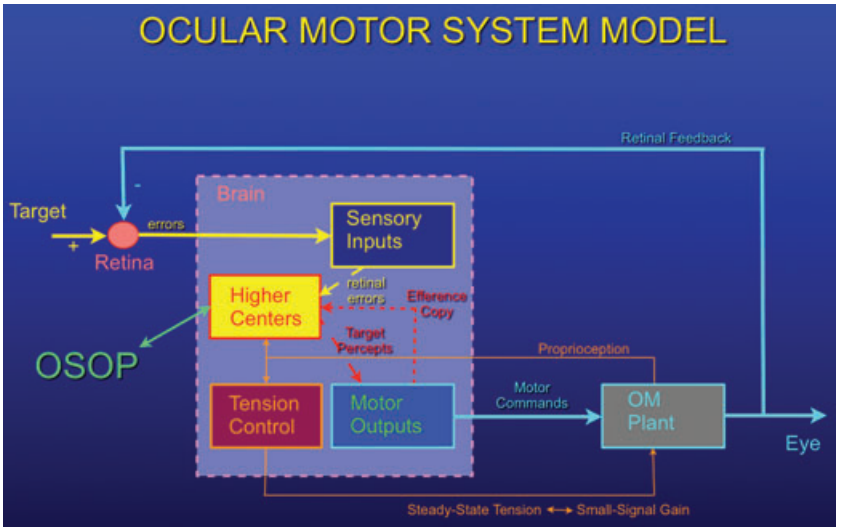

Figure 1. **A functional block diagram of the ocular motor system signals and pathways, including both efference copy and proprioception. As indicated, the perception or suppression of OSOP is generated in higher centers using both retinal and efference-copy signals. The "OM Plant" consists of the eyeball with its surrounding muscles and fascia.**

When such a model was used in an attempt to simulate the responses of an individual with INS, it failed because the retinal error signal contained the eye oscillation in addition to the target information. Since the subject's responses demonstrated the ability to extract and respond to only the target information from the combined retinal error and eye-oscillation signal, it was obvious that any model of the OMS containing an internal oscillation would require the same ability. This was accomplished in the simulation by feeding back the motor command of the oscillation and, after the proper delay, combining it with the retinal error signal, thereby eliminating the oscillation. Although initially done in that nonphysiological way at or just after the retina, this was the first demonstration that such an efference copy of motor commands was a necessary part of normal ocular motor control. Simplistic, retinal error reflex models were incapable of simulating the rich variety of normal and abnormal ocular motor behavior that had been recorded in many laboratories and documented in the literature. As a consequence of this conclusion, all subsequent models of OMS function and dysfunction generated from my research were built on the foundation of efference copy and target-signal reconstruction.[3,4,8–14,16] It should be noted that this same requirement for efference copy applies to normal OMS due to "noise" created by a variety of sources. Additionally, it has been demonstrated that during smooth pursuit that subsystem has no access to retinal slip information; i.e., it is responding to a different signal.[35] I contend that this signal is reconstructed target velocity.

The necessity of employing efference copy of the nystagmus signal in the model before it could simulate the many responses of an INS subject, as well as normals, supports efference copy as the mechanism for OSOP suppression since it allows construction of a stable signal of target position and velocity uncontaminated by the INS oscillation. Figure 1 shows the components of an OMS model and their interconnections. As noted above, retinal error signals do not directly drive the motor output subsystems but rather are used in higher centers along with efference-copy signals to generate stable target signals, which then drive the motor output subsystems. In this more realistic type of model, OSOP and its suppression may be studied. The roles of the proprioceptive signals that go to higher centers have not yet been elucidated; however, in addition to their role in maintaining extraocular muscle steady-state tension, they may also contribute to maintaining appropriate gains in internal models of the ocular motor plant used to generate the reconstructed target signals.

Study 2, observation 2: the effects of retinal image stabilization on INS

In our first formal study of OSOP suppression in INS, we measured the effects of retinal image stabilization (RIS) on four subjects with INS.[20] None of these subjects experienced OSOP under normal conditions. However, RIS produced OSOP of the stabilized image on the retina in all subjects, with some idiosyncratic characteristics. An important observation was that since our methods allowed only partial-field RIS, if the stabilized image appeared to oscillate, the remaining, moving images appeared stationary; however, when the stabilized image was perceived as stable (a transformation that some subjects could accomplish), then the moving images on the remaining portion of the retina would be perceived as moving. Thus, if perceptual stability was imposed on a retinally stabilized image, then the moving images on the rest of the retina, which were normally perceived as stable, would necessarily be perceived as moving.

This study supported both the efference-copy mechanism and its combination with the foveation-period stability mechanism, since the INS subjects had relatively stable foveation periods.

Study 3: intermittent OSOP in INS and loss of consciousness

In this study, we analyzed the eye movements of a subject with INS who had lost consciousness and thereafter suffered intermittent OSOP.[36] He alternated between a jerk-left with extended foveation (JLef) waveform and a biased jerk-right (JR) waveform. During the former, his phase plane showed well-developed foveation and he had no OSOP. During the latter, the phase plane of his waveform did not enter the foveation window and he had OSOP. RIS during both types of nystagmus did not alter the occurrence of OSOP during JR or its absence during JLef. He could suppress the RIS-induced OSOP during JLef but not during JR. Despite a small vertical component to his nystagmus, no vertical OSOP was perceived at any time.

This study supported both the efference-copy mechanism and its combination with the foveation-period stability mechanism, since the INS subject had relatively stable foveation periods. The inability to suppress OSOP during JR was attributed to the addition of an AN to his INS, which was due to a deficit occurring outside of the efference-copy feedback loop or, possibly, interfering with the generation of the stable, reconstructed target signals that drive the motor subsystems.

Study 4: OSOP suppression in INS plus AN following lithium

The subject of this study had diagonal INS with adult-onset OSOP after lithium administration.[37] The plane of his OSOP was dependent on the fixating eye. We employed phase-plane, conjugacy-plot, and both position and velocity scan-path analyses of this subject's waveforms under differing conditions. Under all conditions, the plane with suppressed OSOP also had well-developed foveation periods, whereas the plane with OSOP did not.

This study strongly supported the foveation-period stability mechanism and led to the hypothesis that well-developed foveation periods were both necessary and sufficient for perceptual stability and that they must be simultaneously present in both planes to preclude OSOP in either plane. That is, the brain uses those epochs of clear and stable vision to infer stability during the time between foveation periods, when subjects with INS do not perceive motion smear. Although apparently true for this subject, that hypothesis was soon disproved as a general rule.

Study 5: OSOP suppression in INS, AN, and FMNS plus AN

Given the conflicting findings of the prior studies, we undertook a more inclusive study of three subjects with INS, two with AN and one with FMNS plus AN; we also retrospectively included the subjects of studies 3 and 4, mentioned above.[38] None of the three INS subjects experienced OSOP whether or not they exhibited well-developed foveation periods during specific time intervals or tests. Both of the AN subjects experienced OSOP despite having well-developed foveation periods. The subject with horizontal FMNS and vertical AN (downbeat nystagmus) had well-developed foveation periods in both planes but experienced OSOP only in the vertical plane. The two subjects from studies 3 and 4, who had INS and AN, experienced OSOP transiently with poor foveation quality.

This study supported the efference-copy mechanism and disproved the foveation-period stability mechanism. When present, OSOP was postulated to

arise from a deficit outside the efference-copy loop or interfering with it.

Observations 3 and 4, study 6: OSOP of the scintillating scotoma (migraine aura) in INS

One final study was prompted by observations made possible by the serendipitous occurrence of a migraine aura appearing to the author.[39] The perceived scintillating scotoma stemming from a relatively stationary position in the cortex (albeit slowly growing and drifting away from the mapped foveal area) was also perceived as constantly oscillating in synchrony with the INS despite the periods of extended foveation contained in the waveform. That is, there was OSOP of the scintillating scotoma.

Additionally, a diplopic image secondary to a decompensated phoria during the migraine was perceived as oscillating vertically (presumably due to the subclinical see-saw nystagmus that was part of the "horizontal" INS). When this diplopic condition was duplicated in the lab using a vertical prism in front of one eye, the vertical OSOP could not be suppressed. Similar to the case of partial RIS, where only one portion of the retinal signals could be perceived as stable, with OSOP of the other, only the images of one eye could be perceived as stable when there was diplopia.

These observations and studies further supported the efference-copy mechanism and disproved the foveation-period stability mechanism.

Discussion

The results of the four observations made by a person with INS plus several studies into the mechanism of OSOP suppression made using subjects with INS, AN, FMNS, and various combinations of these distinct types of nystagmus are summarized in Table 1. From those data, efference copy of motor output signals emerges as the mechanism by which the OMS allows stable percepts of the world under normal and abnormal conditions but cannot do so for deficits occurring outside of its sites of action or under conflicting input information (i.e., partial RIS or different image information from each eye in a binocular subject). However, there may be specific patients in whom well-developed foveation periods are also necessary to suppress OSOP.[23] It has been shown that vector subtraction may be the mechanism by which retinal and extraretinal motion are discriminated.[40] Additionally, the important role of proprioception in maintaining visual clarity has been demonstrated in a study of motion smear due to passive eye rotation.[41]

Behavioral OMS model simulation of OSOP suppression

The above hypothesis has been expressed in a behavioral model of the OMS[16] where, despite complex and changing nystagmus waveforms, stable reconstructed target position and velocity signals are generated; they form the basis for accurate perception of those factors in individuals with internally generated eye oscillations.

Figures 2 and 3 show afferent, computed, and output signals present in such a model (e.g., version 1.5 of a behavioral OMS model downloadable from www.omlab.org) during both step- and ramp-target inputs under normal and INS simulations. Figure 2 demonstrates that reconstructed (perceived) target position is stable (no OSOP) both when simulating normals (as expected) and despite the complex oscillations of the eye and retinal position error input in INS. Similarly, in Figure 3, reconstructed (perceived) target velocity is also stable (no OSOP) when simulating normals (as

Table 1. Hypotheses, observations, and studies

Hypothesis	O1 ~1945	S1 1968	S2/O2 1988	S3 1992	S4 1992	S5 1997	O3,4/S6 2002
H1	–	–	–	–	+	x	x
H2	+	+	+	+	–	+	+
H3	–	–	+	+	–	x	x

O#, observation #; S#, study #; H1, foveation-period stability hypothesis; H2, efference-copy cancellation hypothesis; H3, combination of hypotheses 1 and 2.

Note: +, supported/consistent; –, not supported/inconsistent; x, disproved.

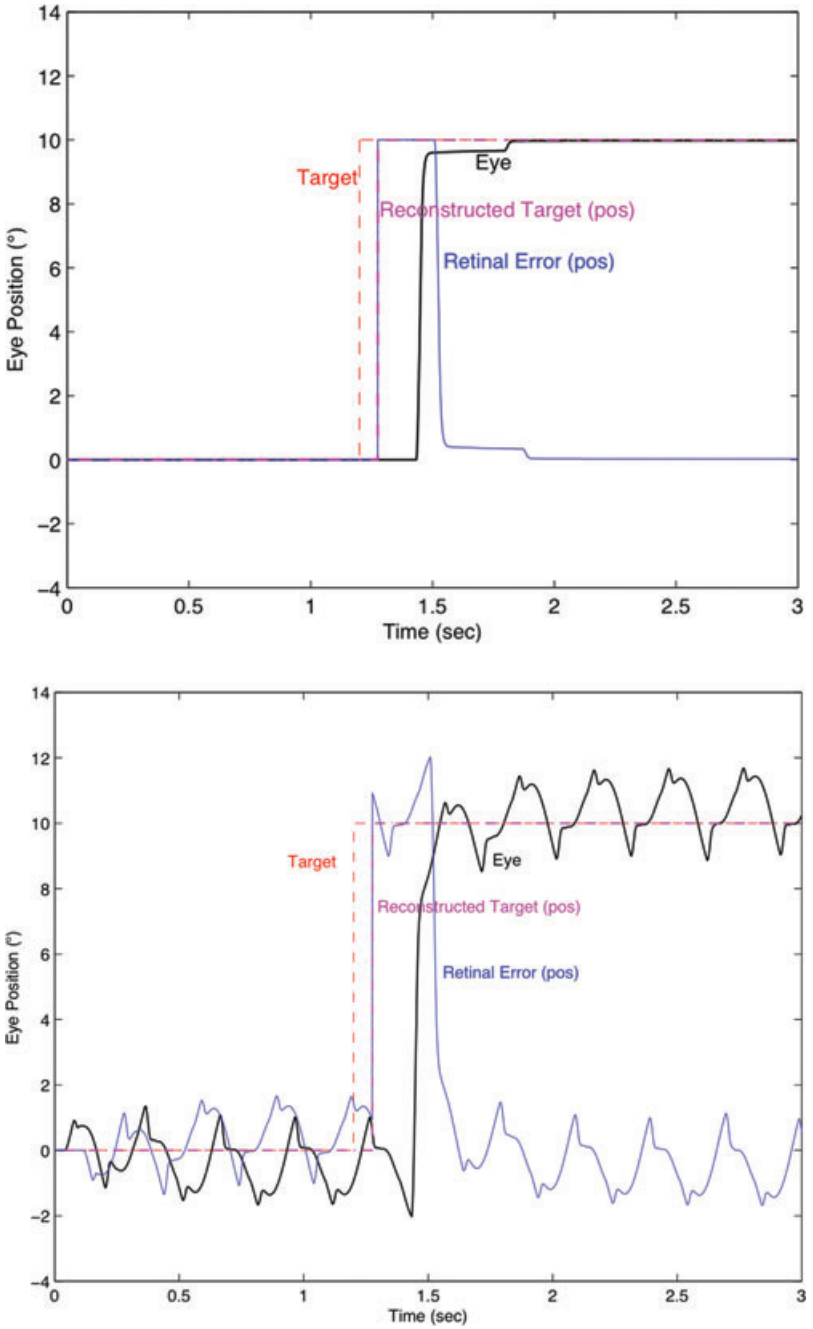

Figure 2. **Model simulation of ocular motor system responses to a step change in target position. Normal system response (top) and response with INS (bottom). Target (dashed red), retinal error position (solid blue), eye (solid heavy black), and reconstructed target position (dash-dot magenta) signals are shown.**

expected) and despite the complex oscillations of the eye and retinal velocity error input in INS. Because of the use of efference copy of motor output signals, the model does not have OSOP and accurately simulates individuals with INS who also do not. Therefore, only deficits that either interfered with the operation of the efference-copy feedback loop and its ability to reconstruct stable target signals or

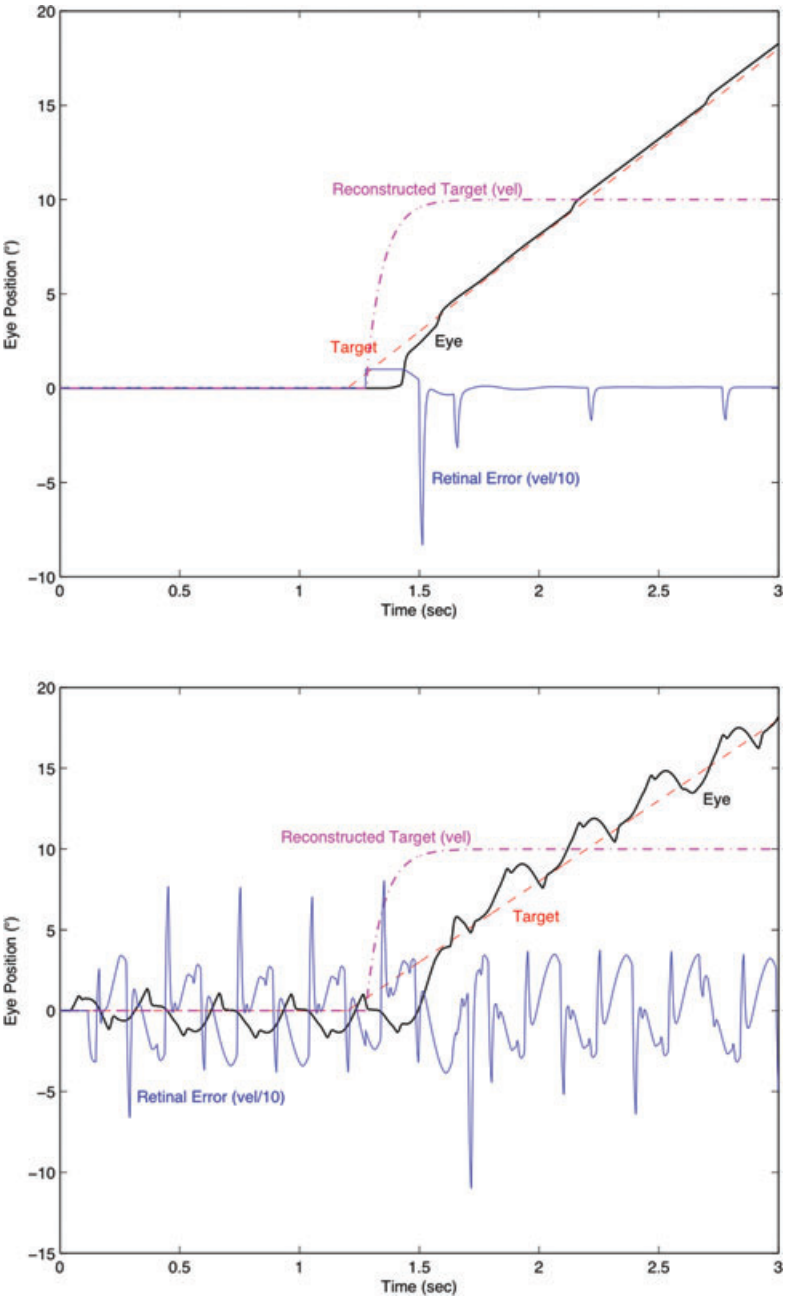

Figure 3. Model simulation of ocular motor system responses to a ramp change in target position. Normal system response (top) and response with INS (bottom). Target (dashed red), retinal error velocity/10 (solid blue), eye (solid heavy black), and reconstructed target position (dash-dot magenta) signals are shown.

lie outside of that loop would be expected to cause OSOP.

Conflicts of interest

The authors declare no conflicts of interest.

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