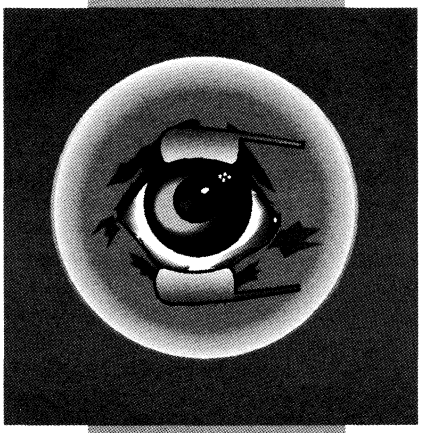

Analyzing Ocular-Motor Control Dysfunction

Insights into disease processes and normal function

L.F. Dell'Osso
Ocular Motor Nuerophysiology Laboratory, Veterans Affairs Medical Center; Departments of Neurology and Biomedical Engineering, Case Western Reserve University; University Hospitals of Cleveland

In the past three decades, research into the control of eye movements has been driven by both the application of control system theory and the use of the basic tools of engineering analysis. Inferences and conclusions derived from such analyses have prompted neurophysiologists to conduct animal experiments in attempts to discover both the pathways and the functions encoded in the neural signals involved in each type of eye movement. The ocular motor system consists of several interconnected and synergistic subsystems, each subserving a particular class of eye movements. When the eyes move conjugately in the same direction, the movements are termed "version" movements, and when they move disconjugately in opposite directions, "vergence" movements. At the most peripheral level, eye movements may be divided into three types: fast and slow versional eye movements, and vergence eye movements. The fast movements are usually called saccades. Each of these types may be further described by the stimulus eliciting it or the function accomplished by it. For example, a slow eye movement made in response to a moving target while the head is fixed is called a "pursuit" movement, while eye movement made in response to head motion while looking at (fixating) a stationary target is called a "vestibuloocular" movement. Both are slow, conjugate movements that are indistinguishable on a strip-chart recording if the conditions that elicited them are not known. In addition, eye movements may be classified into categories determined by whether they are designed to change eye position (*gaze-shifting*) or maintain eye position (*gaze-holding*). Thus, for the two cases described, a pursuit movement is gaze-shifting and a vestibuloocular movement is gaze-holding.

This article will examine several ways in which the application of engineering analysis tools have contributed to our understanding of both dysfunction (e.g., eye movements provoked by disease) and normal function of the ocular motor system. In the past 30 years, beginning with the work of Gerald Westheimer, Larry Stark, Laurence Young and David Robinson, there have been scores of studies of ocular motility that were firmly based on engineering principles and carried out using a classic control-systems approach. I will limit the discussion to concentrating on only a few such studies that have eminated from our laboratory. Specifically, I will focus on studies into two eye-movement disorders: gaze-evoked nystagmus (GEN) and congenital nystagmus (CN). These studies were conducted on humans with one or the other of these conditions and had the dual aims of 1) understanding the mechanisms underlying the pathological eye movements and 2) using the findings to infer how the normal ocular motor system is organized.

Ocular Motor Control

Before describing studies of ocular-motor dysfunction, I will review some basic properties of the normal ocular motor system and its architecture. Only version eye movements will be considered in this article. Specifically, saccades, pursuit, and the vestibuloocular reflex (VOR) will be included. The ocular motor system may be conceptualized as consisting of three subsystems acting synergistically to move the eyes in response to inputs eliciting each of these types of movement. Where possible, the outputs of the subsystems will be combined into a final common pathway.

The Common Neural Integrator

The purpose of the VOR is to move the eyes in a direction opposite to head motion, thereby preserving eye position in space. In 1968, Robinson observed that the proper functioning of the VOR required a neural network performing mathematical integration of the afferent signal on the eighth cranial nerve [1]. It was already known that the signal coming from the semicircular canals on the eighth nerve was coded in head *velocity*, and that

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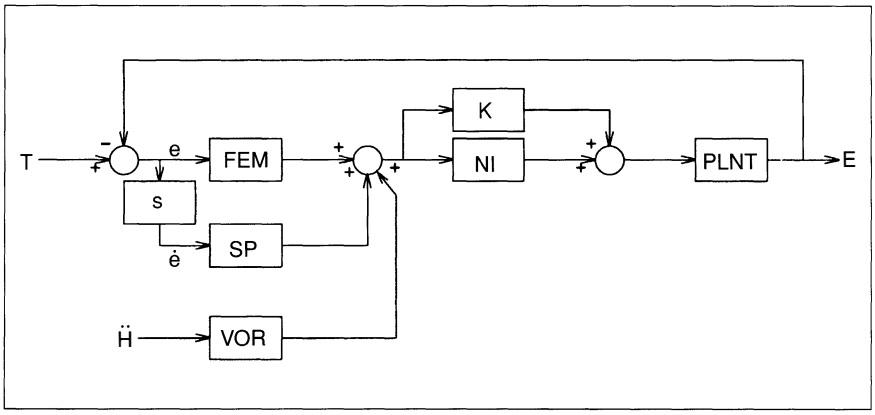
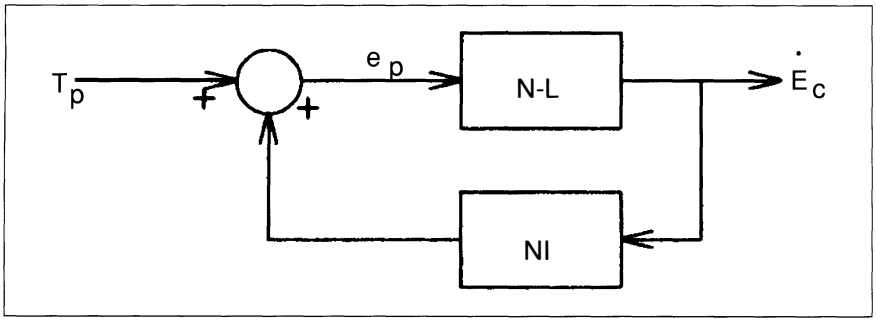


1. Block diagram of the version system with vestibular input, illustrating the difference between the closed-loop saccadic (FEM) and smooth pursuit (SP) mechanisms and the open-loop vestibuloocular (VOR) apparatus. The velocity commands of each subsystem are shown summing and utilizing the final common neural integrator (NI). The NI output sums with the velocity commands and drives the extraocular muscles and globe (PLNT) to produce the required eye position (E). T is target position, e = T-E is retinal error position, ė is retinal error velocity, $\ddot{H}$ is head acceleration, and s indicates differentiation in Laplace notation.



2. Block diagram of the saccadic pulse generator, with the output of a neural integrator (NI) used to set the width of the pulse of activity shaped by the non-linearity (N-L). T_p is the perceived target position, e_p the perceived error position, and $\dot{E}_c$ the eye velocity command.

the discharge rate of ocular motoneurons was predominantly coded in eye *position.*

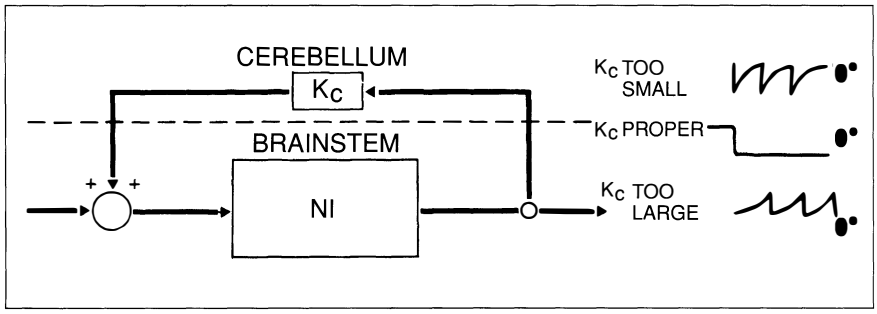
If the VOR was to move the eyes at a velocity equal and opposite to the head velocity, the motoneurons required a signal that was the integral with respect to time of the afferent head-velocity signal. Therefore, an integrator plays a major role in the forward path of the VOR. After the existence of the integrator was predicted, based on this simple mathematical analysis of the signals involved, neurophysiological supporting evidence began to appear. A step of electrical stimulation applied to the reticular formation resulted in constant-velocity eye movements in monkeys [2] (i.e., the ramp of eye position vs. time was the integral of the step of excitation). Researchers also found a 90 degree phase lag between vestibular and ocular motoneurons during sinusoidal rotation of monkeys [3], in line with the operation of an integrator, which exhibits a 90 degree phase lag between its input and output.

In addition to the VOR, another important slow eye movement (SEM) is smooth pursuit. The purpose of the smooth pursuit subsystem is to move the eyes with a velocity equal to that of a moving target. The input to the smooth pursuit subsystem is retinal error (slip) velocity and its output to the motoneurons must also be an eye-position signal. As is the case for the VOR, a velocity signal must be transformed into a position signal; neural integration is also needed by the smooth pursuit subsystem. The optokinetic subsystem, which moves the eyes in response to global retinal image motion, similarly requires integration of retinal slip velocity signals.

The ocular motor plant (the globe, muscles and supporting tissue) is a highly overdamped system [4]. That is, it is slow and sluggish to respond to changes in innervation. Despite this response, the eyes are moved extremely rapidly when fixation is changed from one target to another. These fast eye movements (FEM) are called saccades. Burst cells in the pontine paramedian reticular formation create a pulse of high frequency, which is relayed through the motoneurons to the extraocular muscles, creating saccades [5]. To complete the saccade and hold the eye in its new position, the burst of activity must be followed by a constant step change in activity of the motoneurons. Since the burst cell activity (the pulse) can be considered an eye-velocity command [6], the required eye-position command (the step) can be achieved by integration of the pulse. The saccadic subsystem requires neural integration for the same reasons as that for the SEM subsystems.

Thus, each of these three ocular motor subsystems requires neural integration. Neurophysiological evidence suggests that a common neural integrator (NI) performs this function for all three subsystems [7]. All ocular motoneurons carry an eye-position signal that is independent of the type of movement involved. The NI integrates all the velocity commands to produce a single, composite eye-position command at its output. Figure 1 is a simple block diagram illustrating the common NI responding to saccadic (FEM), pursuit (SP), and vestibuloocular (VOR) velocity commands. The direct pathway (K) around the NI provides the velocity information that is also carried by ocular motoneurons. Both the FEM and SP subsystems are enclosed by the negative feedback of vision at the retina, but the VOR is open loop (i.e., there is no feedback).

Robinson used an NI in his "bang-bang" model of saccadic generation to feed back a signal that turned off the burst neurons [8] In this pulse generator model (Fig. 2), a perceived position error causes a velocity pulse output, shaped by the non-linearity (N-L) shown, whose width is determined by the output of the NI. Since an NI was needed for the eye-position signal (Fig. 1), its output was used as the source for the signal needed by the pulse generator. Although efficient in terms of the number of elements required to do the job, the model is not especially flexible for the study of normal variability and plasticity, and also has limited use for studying dysfunction. It is for this latter reason that we expanded the model by separating the two integrator functions in



3. Block diagram of the cerebellar positive feedback path with a gain (K_C) around the leaky neural integrator (NI). (Reprinted with permission from [34]).

the models used in our laboratory. One, the *local* integrator, was resettable and used to determine saccadic pulse width; the other, the *common* integrator, provided the eye-position signal.

If the same NI determines both the pulse width and the final eye position, then altering its gain (deg/(spikes/sec) for eye position) could change both the initial trajectory and the final position of a saccade. However, studies of plasticity have shown both these variables to be under independent control [9-11]. Also, there are types of nystagmus (eye oscillation), caused by NI dysfunction, in which the slow-phase waveforms (but not those of the saccadic fast phases) are determined by the NI dysfunction; a single NI would affect both. Figure 3 illustrates the slow-phase waveforms produced by common NI time constants that are either too high or too low. The normal common NI has a "leaky" time constant of about 25 s in the dark [12]; in the light, the time constant is reduced by cerebellar feedback [13]. Too short a time constant produces decreasing-velocity slow phases, and too long a time constant produces increasing-velocity slow phases. Slow phase waveforms are diagnostic of certain types of nystagmus.

Gaze-Evoked Nystagmus

One of the early studies performed in our laboratory was of an acquired ocular-motor condition, gaze-evoked nystagmus (GEN). This is an acquired oscillation of the eyes that prevents maintained eccentric gaze. The afflicted individual is able to fixate objects at or near primary position (i.e., straight ahead), but when trying to look to either side the eyes fail to maintain gaze on the target. Instead, they drift with a decreasing-velocity towards primary position. The drift is interrupted with a saccade to reset gaze laterally on target, but the eyes continue to drift centripetally. The repetition of these alternate drifts and resetting saccades results in the nystagmus oscillation. The direction of the GEN is defined by the saccadic fast phases and is, therefore, lateral in the direction of attempted gaze. GEN and other types of acquired nystagmus result in the illusory movement of the environment, due to the constant retinal image motion produced by the nystagmus. This effect, called "oscillopsia," is an extremely debilitating condition. Oscillopsia can prevent reading, watching TV, driving, and even walking.

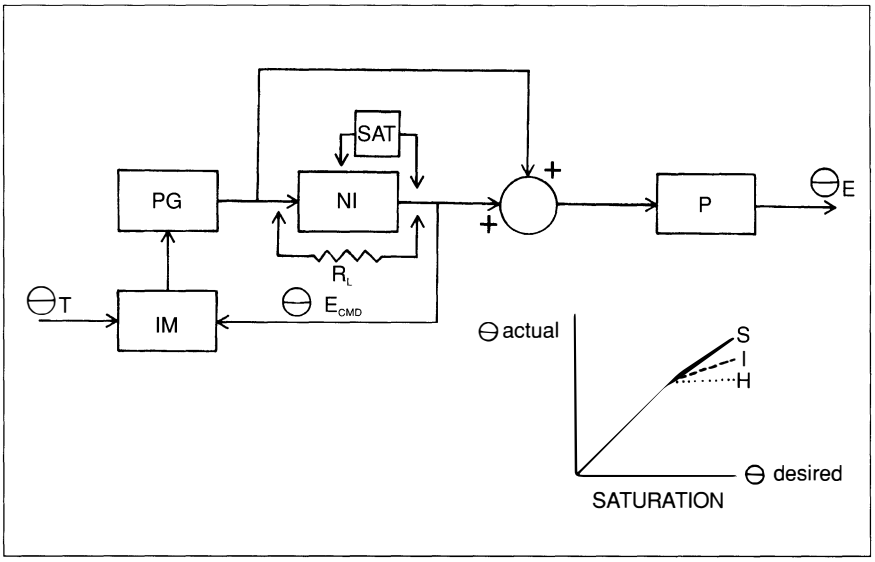
Independent Neural Populations

The study of GEN produced an early model of NI nystagmus, which we simulated on an analog computer [14]. Here, as is shown in the block diagram of Fig. 4, the NI was "lesioned" in two ways to produce GEN. The integrator was either made leaky (meaning its time constant was reduced) or it was made to saturate to varying degrees. Recognizing the inherently distributed nature of a physiological NI, we divided the model's NI into two parts, each representing a portion of those neurons responsible for integrating velocity commands into position commands. In this way, a portion of the neuronal pool could be lesioned and the resulting GEN studied. With such a partial deficit, the model produced a GEN that decayed to a non-primary position gaze angle, mimicking that seen in some patients.

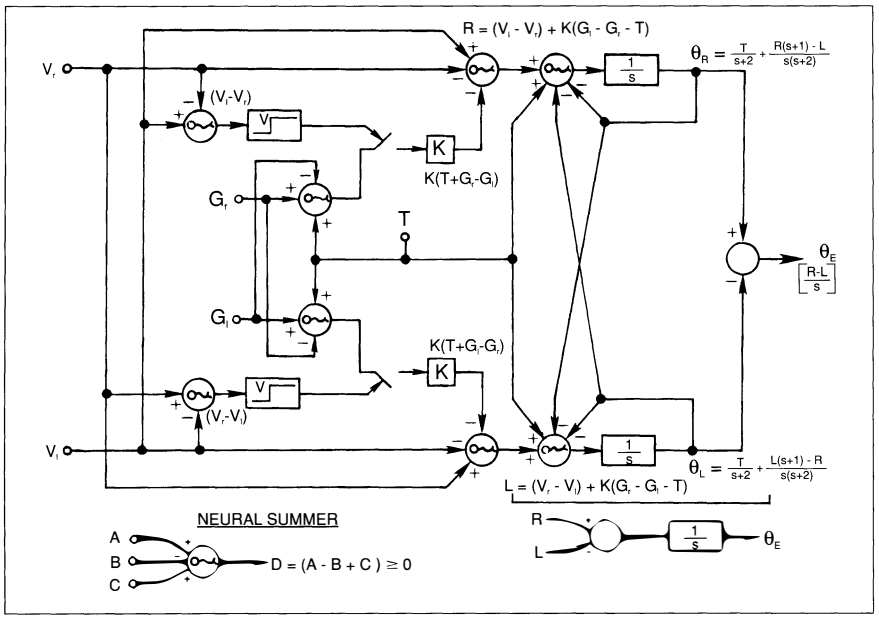
Local Neural Integrator

In Fig. 4, the NI shown is the common NI, shared by the saccadic, pursuit, and VOR subsystems. We hypothesized that a local NI, necessary to shape saccades, was contained within the pulse generator (PG). The internal monitor (IM) used the eye-position command to determine if a corrective saccade was needed. The local NI within the PG set the pulse width of the saccade.

In addition to GEN, the model predicted different effects on saccadic trajec-



4. Block diagram of the model used to simulate gaze-evoked nystagmus. The input target position (Θ_T) is compared by the internal monitor (IM) to the eye position command ($\Theta_{E_{CMD}}$) and the necessary saccades initiated in the pulse generator (PG). The IM contains an error threshold below which no correction is called for. A refractory circuit also prevents calling for a correction during a saccade. The PG contains nonlinearities that reproduce the physiological variations in pulse height and a local neural integrator that determines pulse duration. The output of the PG is integrated in the common neural integrator (NI). The pulse and step are summed, with the resulting innervation used to drive the plant (P) to produce the desired eye position (Θ_E). Both a leaky NI (R_L) and saturating NI (SAT) are indicated as ways to produce gaze-evoked nystagmus. The inset shows typical saturation characteristics used (soft, S; intermediate, I; hard, H).



5. Signal flow diagram of a model for the Alexander's law variation of vestibular nystagmus containing the necessary quantitative interaction among signals. Mathematical relationships utilize Laplace notation where applicable. The push-pull interconnected neural integrators (1/s) integrate differential inputs and pass common tonic (T) signals. Neural summers can have only positive outputs. (Reprinted with permission from [17]).

tories, depending on the particular NI defect used. Leaky NI's produced overshooting saccades whereas saturating NI's produced normal saccades in the region before the saturation took effect.

One of the major results of this study of GEN was the hypothesis that two NI's are necessary in the ocular motor system: the common NI that determines eye position; and the local, resettable NI that determines saccadic duration. Later studies in monkeys would arrive at the same conclusion regarding the ocular motor systems of normals [15, 16].

Bilateral "Push-Pull" Neural Integrator

An imbalance in the firing rates of the vestibular neurons on each side of the brain stem causes vestibular nystagmus. This condition increases in amplitude as gaze is directed towards the fast phases (Alexander's law). Although we were able to use a unilateral model of the ocular motor system for the GEN study, a bilateral model was necessary for studying the Alexander's law variation of vestibular nystagmus [17]. This model is an extension of an earlier one (1974) developed in our laboratory, and features bilateral NI's connected in a push-pull fashion.

Figure 5 is the signal flow diagram for this model; we will concentrate only on the NI interconnections. As shown, all neural summers are restricted to outputs greater than or equal to zero (simulating neurons). Since there is a tonic firing rate on each vestibular input and a tonic output to each motoneuron and muscle (even during fixation at primary position), the NI's are interconnected in a manner that results in both the integration of the differential input (left vs. right) and the simple relaying of the common tonic inputs to the motoneurons. The lower right-hand equivalent diagram for the two interconnected NI's shows that eye position is a function of only the differential input, while the equations for each NI output show the existing tonic signal. A bilateral model with connections across the midline is necessary for illustrating both the above phenomena and the importance of the crossing fibers; these features would be lost if a unilateral model were used. This use of reciprocal inhibition of NI's was borrowed directly from the commonly used push-pull architecture of electronic circuits. This technique solved the problem of integrating only differential signals and not common-mode signals, and was adopted in later models of the NI itself [18, 19].

Congenital Nystagmus

Another area of ocular-motor research where engineering methods have proved fruitful is in studying several types of infantile nystagmus. One of these, congenital nystagmus (CN), is an oscillation of the eyes that usually appears at birth or during early infancy but may appear later in rare instances. The 12 CN waveforms identified are variations of two basic types—pendular and jerk—with periods of extended target foveation present during each cycle [20].

Target foveation occurs when the image of the target is on the fovea, the retinal region of highest visual acuity. The slow phases of jerk waveforms are of increasing velocity. Studies into this disorder have uncovered properties of the ocular motor system that are hidden in normals. In addition, the criteria by which individuals with CN suppress oscillopsia (the illusory perception of movement of the environment) caused by the continual movement of images on the retina has provided insight into the mechanisms used by normals, and may be applied towards suppressing the oscillopsia experienced by those with acquired nystagmus.

Fixation Subsystem

It has long been thought that there was a specific fixation subsystem that maintained the image of a stationary target on the fovea. Our early work demonstrated that CN waveforms described oscillations of the eyes away from and back to the target of interest, not across the line of sight, as was stated in the medical texts of the time. It was known that some individuals with CN could exhibit normal visual acuity if there were no accompanying deficits to their afferent visual systems. Based on that observation and the knowledge that the velocity of a sinusoidal oscillation goes through zero only at each peak and is maximum at its midpoint, the hypothesis was advanced that CN waveforms were biased such that target foveation occurred at one of the peaks of each cycle [21].

Previous descriptions of oscillations across the line of sight, which were incompatible with good visual acuity, were based solely on clinical observations. It was not until accurate methods of recording eye movements were developed by engineers, and the signals processed by high-bandwidth, dc-coupled electronics, that the CN waveforms could be properly studied. Two such methods are infrared reflection, and scleral search coils in rotating magnetic fields. Such quantitative recordings of pendular CN showed characteristic "flat spots" at one peak of each sinusoidal cycle. Furthermore, these flat spots lined up from cycle to cycle, suggesting that their position was under

Eye Movement Measurement Techniques

There are two basic methods of recording eye movements that have been used extensively in research for the past three decades. One makes use of infrared (IR) light reflected from the eyeball, and the other uses a voltage induced in a coil affixed to the eye in a magnetic field. Both methods have advantages and drawbacks, and their introduction in the 1960s revolutized the field which had previously been limited by the use of electrooculography (EOG). EOG used skin electrodes to measure the dipole characteristic of the eyeball (the retina is electronegative with respect to the cornea). This method was not sensitive enough to record small movements, suffered from drift, powerline, and muscle artifact, and could not be used for vertical movements due to eyelid artifact.

The IR method uses two phototransistors aimed at the border between the iris (colored portion) and sclera (white portion) of each eye. Each eye is illuminated by an IR source. The voltages from each pair of phototransistors are differentially amplified and nulled when the eye is in the primary (0 degree) position. As each eye rotates, one of the phototransistors "sees" more reflected IR light and the other less. The resulting voltage is proportional to eye position. Two of these setups are mounted in normal eyeglass frames so that they can be moved independently in each direction (up-down, left-right and in-out) by means of adjusting screws. For horizontal eye movements, they are adjusted to give symmetrical outputs for equal eye movements in the left and right directions. For vertical eye movements, the phototransistor outputs are summed and they are pointed at the lower border of the iris and sclera. The IR method is non-invasive, stable, precise, accurate, and not subject to the artifacts of EOG. It is linear within ± 20 degrees horizontally and $\pm$ 10 degrees vertically, and has high bandwidth.

The scleral search coil method uses coils of fine wire embedded in annular contact lenses or, for animal work, surgically attached to the eye. The subject is placed in large coils of wire that induce magnetic fields in each direction. The fields are excited at non-harmonically related frequencies so that the composite signal can be split into horizontal and vertical components, reflecting eye movements in these planes. If, in addition, a second coil (wound as a figure "8") is embedded in the contact lens, torsional eye movements can be measured. Also, a coil on the head will measure head movements. Either the coil-signal amplitudes or phases can be used to obtain the eye-position signals. This method allows simultaneous measurement of eye movement in three planes of the eyes and head. It has a usable range of about ± 40 degrees, is highly precise and accurate, and has high bandwidth. However, it is invasive and can only be used for 20-30 minutes at a time.

active control. The opposite peaks showed no such position control. These flat spots in CN records were named "foveation periods," reflecting their presumed functional relationship to target imaging on the fovea. Retinal cinematography, with a laser target imaged on the retina [21], showed this finding to be an accurate description of the CN waveforms.

The existence of foveation periods in CN waveforms was the first direct evidence of a fixation subsystem in ocular-motor control. That is, despite an oscillation of unknown origin in CN, target foveation could still be achieved for some brief intervals of time (30 - 400 ms) during each cycle. Despite this early evidence for the existence of a fixation subsystem, most models of "normal" ocular motor systems failed to include it. Only recently has the fixation subsystem undergone a resurgence of interest [22-24].

Braking Saccade

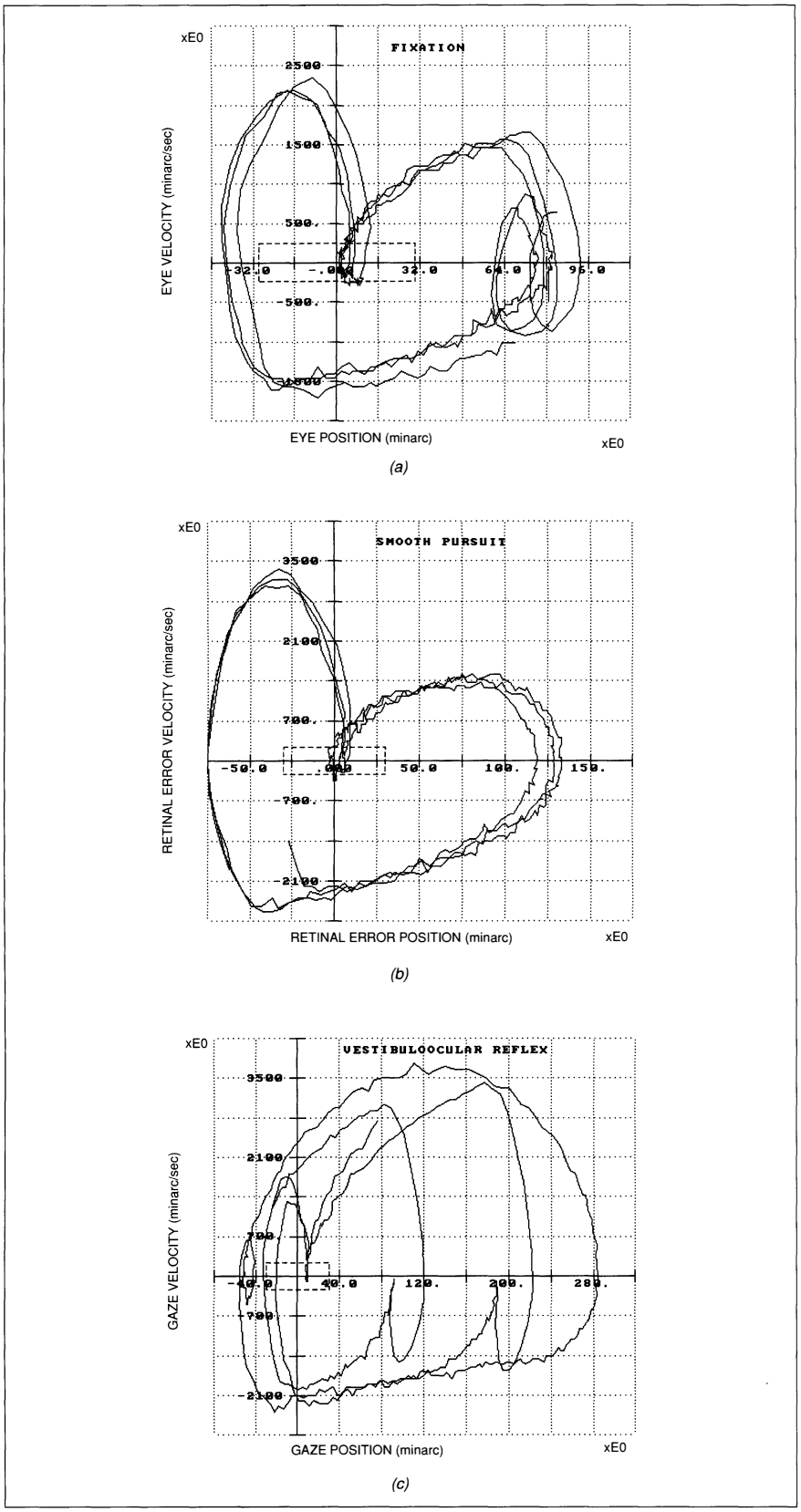
Another capability of the ocular motor system demonstrated by accurate recordings of CN is its use of the saccadic system in a way that differs from its primary mission. In foveate animals, saccades are used to bring images of targets of interest onto the fovea (i.e., to foveate a target). CN waveforms were shown to have small "braking" saccades that stopped the accelerating motion of the eyes away from the target, but did not bring the eyes back to the target [25]. This is a novel use of the saccadic system by individuals with an ongoing oscillation. In normals, repetitive resetting saccades (fast phases), unrelated to any specific target, occur in several types of nystagmus. The braking saccades of CN may use this same built-in mechanism to halt a runaway CN slow phase.

Phase-Plane Analysis

The use of phase planes for analysis is common in systems engineering. The phase plane allows simultaneous representations of a variable and its derivative (e.g., position and velocity) on a single plot, which is especially useful in analysis of cyclic events. The earliest use of phase planes for ocular motility studies was in periodic alternating nystagmus [26]. Phase planes allowed the authors to interrupt this periodically reversing nystagmus at a point in the limit cycle that caused the nystagmus to cease for a short time. A second use of phase planes was in the analysis of saccades and smooth pursuit in normals [27]. At about the same time as this later study, we were applying phase planes to the analysis of foveation dynamics in CN during fixation [28], smooth pursuit [29], and the VOR [30]. Here, we observed the cyclic nature of the CN waveforms themselves, rather than of a control-system limit cycle. The ability to simultaneously observe position and velocity of the eye relative to a target under these various conditions provided unique insight into the underlying ocular motor mechanisms at work. Previous attempts to quantify fixation, the "gain" of pursuit, or the VOR were contaminated by the CN oscillation itself.

The trajectories seen on phase plane plots are always in a clockwise direction if the conventions of rightward direction and velocity being positive are followed. Saccadic movements appear as high-velocity clockwise loops; rightward saccades would show positive velocities and directions, while leftward saccades would be negative. The trajectories of respective slow movements would also appear clockwise, with lower velocities. During fixation, phase planes enable immediate identification of those periods when the target image is both stable and on the fovea. In the analysis of smooth pursuit or of the VOR, phase planes of retinal image motion or gaze identify those periods of stability indicative of good pursuit, or VOR, respectively.

In the study of fixation, we found that the foveation periods of successive CN cycles were superimposed on the phase plane as foveation cusps (Fig. 6a) [28]. The cusps follow the rightward foveating saccades (narrow, high-positive-velocity rightward loops to the left of centerline) and are centered at (0,0). Thus, despite the high velocity oscillations, the ocular motor systems of individuals with CN were



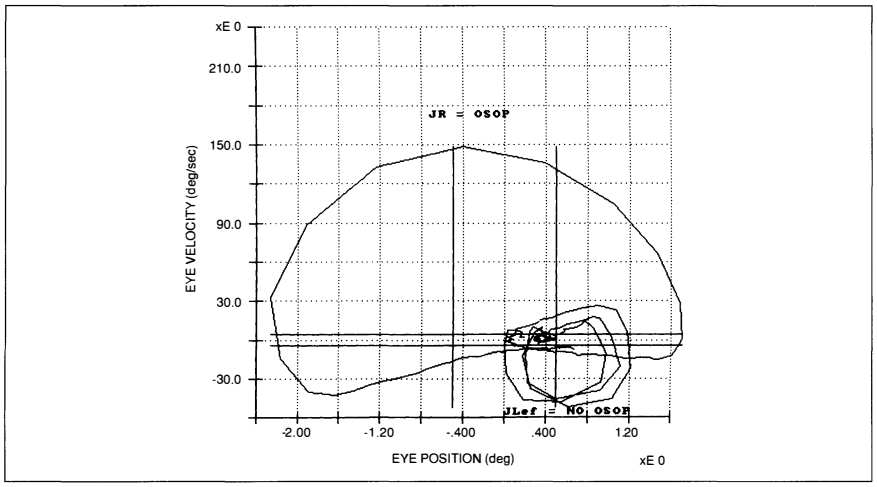
6. Phase planes of the horizontal motion of an individual with congenital nystagmus during (a) fixation of a stationary target, (b) pursuit of a moving target and (c) head rotation while fixating a stationary target. In all three situations, the foveation periods form overlapping cusps that lie within the foveation window (dashed lines) described in the text.

able to place images of targets of interest precisely on the fovea, with minimal retinal slip velocity on a cycle-to-cycle basis. This activity enabled the high visual acuities possible despite the CN.

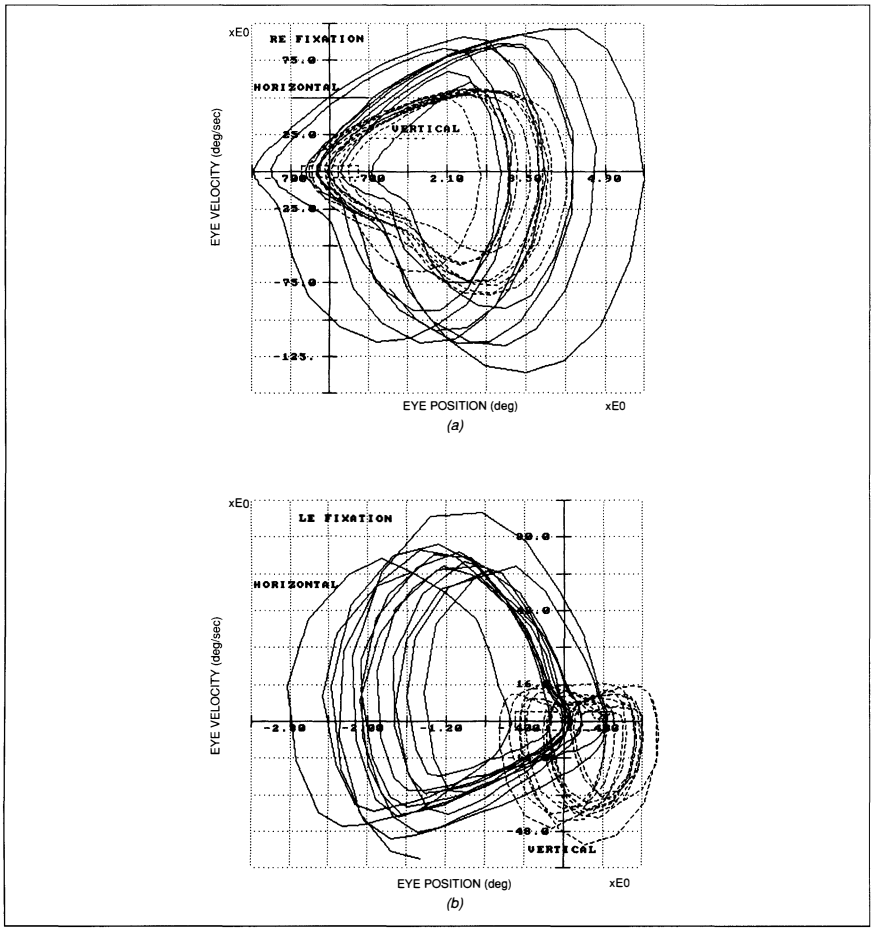
By subtracting target motion from eye motion and plotting the phase planes of retinal slip, we showed that during smooth pursuit the same superimposition of foveation cusps occurred (Fig. 6b) [29]. This demonstrated that smooth pursuit was normal in this individual with CN, since he could attain the same repetitive positioning of the moving target on the fovea with minimal slip as he did for stationary targets. Indeed, one could calculate the "gain" of smooth pursuit during these foveation periods, when the eyes would otherwise be relatively motionless. At all other times during the CN cycle, gain is undefined because eye motion is not causally related to target motion (i.e., eye motion is caused by the CN oscillation, not the target motion). We found these foveation-period gains to be normal. Finally, during the VOR, a phase plane of gaze (the difference between eye-in-head and head-in-space) showed this same superimposition of foveation cusps (Fig. 6c) [30]. Foveation-period VOR gains were also normal. Thus, the VOR was also operating normally in individuals with CN.

One of the most important uses of phase planes thus far has been in the study of oscillopsia and its suppression in those with CN. As mentioned above, oscillopsia is a devastating symptom of acquired nystagmus, in which the visual scene appears to move. The alleviation of this affliction is of prime importance. However, individuals with CN do not complain of oscillopsia. Therefore, we studied the suppression of oscillopsia in CN in an attempt to understand the underlying mechanisms involved and develop a theory for acquired nystagmus. The mechanism by which individuals with CN suppressed oscillopsia was unknown. By studying two unique cases of individuals with CN who acquired oscillopsia later in life, we were able to establish position and velocity criteria that, when satisfied, resulted in the suppression of oscillopsia. On the phase plane, these criteria appear as a "foveation window," within whose boundaries (± 0.5 by ± 4 degrees/sec) foveation cusps are required to fall if oscillopsia is not to be perceived. These boundaries were deduced from data on acuity in normal subjects, along with its variation with both retinal image position and velocity.

Figure 7 shows the phase plane of one individual who developed intermittent os-



7. Phase plane of the horizontal motion of an individual with congenital nystagmus during a transition from one cycle of a jerk right (JR) waveform (causing oscillopsia (OSOP)) to three cycles of a jerk left (JL_{ef}) waveform (no oscillopsia). Only during the latter waveform did the foveation periods lie within the foveation window (rectangle described by the intersection of pairs of horizontal and vertical lines). The numbers are points along one of the foveation periods. (Reprinted with permission from [31]).



8. Horizontal (solid) and vertical (dashed) phase planes of an individual with congenital nystagmus during 2 sec of fixation with (a) the right and (b) the left eye. The horizontal position scatter shown in (a) and vertical velocity scatter shown in (b) correspond to the directions of perceived oscillopsia reported for these respective fixation conditions. The foveation windows are shown by the dashed lines in each phase plane. (Reprinted with permission from [32]).

cillopsia in adulthood, which corresponded to intervals during which his foveation cusps fell outside the foveation window on the phase plane [31]. One such oscillopsia producing CN cycle of rightward beating CN, followed by three cycles of leftward beating CN (no oscillopsia), is shown. In another individual, who also developed oscillopsia in adulthood, we were able to document that the plane of the oscillopsia corresponded to the plane in which the foveation window criteria was violated [32]. This correspondence was independent of the eye oscillation which, in this case, was diagonal. Figure 8a shows the horizontal and vertical phase planes of the right eye during fixation with that eye, which produced horizontal oscillopsia. Note that the foveation window boundaries are violated by the horizontal positions of successive foveation cusps. With left-eye fixation and vertical oscillopsia, the horizontal and vertical phase planes of that eye (Fig. 8b) show that the vertical velocity boundary of the foveation window is exceeded.

We have recently discovered another capability of the saccadic system from our studies of latent/manifest latent nystagmus (LMLN), another type of infantile nystagmus [33]. Here, saccadic fast phases are generated to *produce* retinal errors, rather than to correct them. The phase planes of LMLN show such fast phases, whose endpoints lie outside the foveation window required to suppress oscillopsia, followed by slow phases that enter this window.

Conclusions

As these few examples demonstrate, the application of simple techniques of engineering analysis to eye-movement data obtained by well-engineered methods has contributed to our understanding of ocular motor dysfunction. Also, studies of these "experiments of nature" have uncovered built-in capabilities of the *normal* ocular motor system in an attempt to attain and prolong repeatable (cycle to cycle) target foveation, thereby allowing high acuity and the suppression of oscillopsia.

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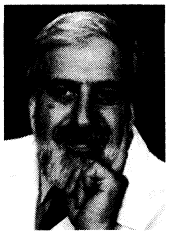
Louis F. Dell'Osso is a Professor in the Departments of Neurology and Biomedical Engineering at Case Western Reserve University, a biomedical engineer at the Cleveland VAMC, and Director of the



Ocular Motor Neurophysiology Laboratory. He received his B.E.E. from Brooklyn Polytechnic Institute in 1961 and Ph.D. in E.E. with a biomedical major from the University of Wyoming in 1966. He is a senior member of the IEEE and IEEE EMB Society, a fellow of the North American Neuro-Ophthalmology Society, and a member of the AAS, and the Association for Research in Vision and Ophthalmology.

He may be reached at the Ocular Motor Neurophysiology Laboratory, Veterans Affairs Medical Center (127A), 10701 East Boulevard, Cleveland, OH 44106. Tel: (216) 421-3224; Fax: (216) 844-3160; E-mail: lfd@po.cwru.edu

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